ANNEXURE-"F"

Information of Mentor of Training Centre

It shall be verified by the Head of the concerned Training Center

Sr. No.	Particular	-	Information to be filled
1.	Name of Mentor	:	Dr. Jigna Pathak
2.	Date of Birth	:	05/04/1975
3.	Address	:	501, Plesant View Society, Plot: 56/57, Sector-14, Vashi, Navi Mumbai-400703.
4.	Tel. No./ Mob. No.	:	9819175805
5.	E-mail id	:	drjignapathak@gmail.com
6.	Nationality	:	Indian
7.	Qualification in details: (attach documentary proof)	:	BDS, MDS
8.	Teaching experience Health Science: Profession experience / Consultant /Mentor (attached document proof with signature of Head of the Institute. Also it is mandatory to attach self-attested photocopy of the Experience Certificate of each Mentor in the subject of concern fellowship/ Certificate course)	:	19yrs. 6months 27dys.
9.	Present Appointment at Institute /Hospital/College	:	Professor
10.	Publications (List & Proof)	:	1. Shegaonkar A, Patel S, Swain N, **Pathak J**, Hosalkar R, Waghmare A. Evaluation and Correlation of Clinicopathological Parameters of Oral Squamous Cell Carcinoma of Gingivobuccal Sulcus with Lymph Node Status - A Retrospective Institutional Analysis in Navi Mumbai. J Evolution Med Dent Sci . 2021;July 10(30): 2294-9. 2. **Pathak J**, Swain N, Pathak D, Shrikanth G, Hosalkar R. Role Of Various Stakeholders In Application OfArtificial Intelligence To Forensic Odontology-APotential Perspective. Annals of Dental Specialty .2021; Jan-March 9Vol. 9(1): 47-52. 3. Shreesh Mhatre V, **Pathak J**, Patel S, Poonja LS, Swain N, Dekate K, Bhandarwar A. Morphological analysis of palatal rugae patterns in a population of Maharashtrian ancestry: a cross-sectional study. J Forensic Odontostomatol. 2020 Sep 30;2(38):12-21. 4. Swain N, Thakur M, **Pathak J**, Swain B. SOX2, OCT4 and NANOG: The core embryonic stem cell pluripotency regulators in oral carcinogenesis. J Oral Maxillofac Pathol 2020;24: 368-73. 5. Punjabi V, Patel S, **Pathak J**, Swain N. Comparative evaluation of staining efficacy of calcofluor white and acridine orange for detection of *Candida* species using fluorescence microscopy - A prospective microbiological study. J Oral Maxillofac Pathol. 2020;24(1):81-86. doi:10.4103/jomfp.JOMFP_315_18 6. Dr. Ketki Shirke, **Dr Jigna Pathak**. Lichen Planus- An Update From etiopathogenesis to management (May 2020) Lambert publication 978-6202565417

10.	Publications (List & Proof)	:	7.	Dr. Amit Shah, Dr.Shilpa Patel, **Dr.Jigna Pathak,** Dr. Niharika Swain and Dr. Shwetha Kumar. Conceptualizing Cancer Stem Cells in Head and Neck Squamous Cell Carcinoma. Emerging Issues in Science and Technology Vol. 3 978-93-89816-20-4
			8.	**Pathak J**, Hosalkar RM, Sidana S, Swain N, Patel S. Benign cementoblastoma involving left deciduous first molar: A case report and review of literature. J Oral MaxillofacPathol. 2019;23(3):422–428. doi:10.4103/jomfp.JOMFP_193_19
			9.	Bhandarwar AU, Patel S**, Pathak J**, Swain N, Mhatre VS, Tekade S.Immunoexpression And Correlation Of Tumour Infiltrating B Lymphocytes, In Oral Squamous Cell Carcinoma With Lymph Node Status-A Retrospective Study.IJSR.2019; 8(4):27-29
			10.	Jain MN, Patel S, Dekate K, **PathakJ**, Shirke KJ, Patel T. Oral Verrucous Hyperplasia: A Case Series. J Contemp Dent 2018;8(3):163-167.
			11.	Shirke KJ, **Pathak J**, Swain N, Patel S, Patel T, Jain M. Oral Lichen Planus–A Brief Review on Treatment Modalities. J Contemp Dent 2018;8(3):137-143.
			12.	Patel T, Patel S, **Pathak J**, Swain N, Jain M, Shirke KJ. Dermoid and Epidermoid Cysts: A Case Series. J Contemp Dent 2018;8(3):153-156.
			13.	Gurav R, **Pathak J.** Myoepithelial cells in salivary gland: Physiology to pathology.. Lambert academic publishing.ISBN 978-613-9-82522-6.
			14.	Gurav R, **Pathak J**,Patel S, Swain N Evaluation of micronuclei count in exfoliated buccal mucosal cells amongst different age groups of normal healthy individuals: a quantitative study. IJCR 2018; 10(09): 73845-9.
			15.	Azmi K, Patel S ,**Pathak J** , Swain N, Gurav R,. Immunoexpression and correlation of cytotoxic t lymphocytes in oral squamous cell carcinoma with lymph node status: a retrospective study. IJSR 2018;7(4):42-44.
			16.	Gurav R, **Pathak J**,Patel S, Swain N, Azmi K. Evaluation and correlation of density of tumour associated macrophages in oral squamous cell carcinoma with or without lymph node involvement: a retrospective immunohistochemical analysis. IJSR. 2018;7(4):45-47.
			17.	Mhatre V and **Pathak J**. Verruco papillary lesions of oral cavity. ISBN: 978-613-8-18498-0. Lambert Publication Germany 2018.
			18.	Rathod S, Patel S, **Pathak J**, Swain N. Immunojistochemical expression of MCM-2 for evaluation pf proloferative activity in the epithelial lining of radicular cyst, dentigerous cyst and odontogenic keratiocyst -A retrospective study. J ContempDent 2018;1: 20-26.
			19.	Iyer J, **Pathak J**, Patel S.Micronuclei in oral and urothelial cells associated with tobacco habits.ISBN: 978-3-330-04205-6. Lambert Publication Germany2017
			20.	Hosalkar R, Patel S, **Pathak J**. Vascular Anomalies: Tumours& Malformation. ISBN: 978-3-330-05778-4. Lambert Publication Germany 2017.
			21.	Niharika Swain, **Jigna Pathak**, Shilpa Patel & Rashmi Hosalkar.MMP-9.Encyclopedia in signaling molecules (2nd edition) ISBN: 978-1-4419-0460-7. 2017
			22.	Niharika Swain,**Jigna Pathak**& Rashmi Hosalkar.Defensin.Encyclopedia in signaling molecules (2nd edition) ISBN: 978-1-4419-0460-7. 2017

10.	Publications (List & Proof)	:	23.	Bhandarwar A , Patel S, **Pathak J**, Swain N, Gandevivala A. Postsurgical Epidermal Inclusion Cyst in the Cheek. J Contemp Dent 2017; (3); 178- 80
			24.	Vishal Punjabi, Shilpa Patel, **Jigna Pathak**, Niharika Swain - Fibrolipoma of Lip in a Young Individual; A Rare Presentation. J Contemp Dent 2017 ; (3); 181-184
			25.	Vibhuti Mhatre, **Jigna Pathak**, Shilpa Patel, Niharika Swain, Adil Gandevivala- Diffuse Lipomatosis of Face. J Contemp Dent 2017; 7(3) ; 185 - 87
			26.	Nikitha Narayanan, **Jigna Pathak**, Shilpa Patel, Niharika Swain – Evaluation of Nuclear Morphometry in Oral Squamous Cell Carcinoma: - A Retrospective Study. J Contemp Dent. 2017;7(2): 107-13
			27.	Adenosine Deaminase- a Novel Diagnostic and Prognostic Biomarker for Oral Squamous Cell Carcinoma. Kelgandre DC, **Pathak J**, Patel S, Ingale P, Swain N.Asian Pac J Cancer Prev. 2016;17(4):1865-8.
			28.	Kehkashan E Azmi, Shilpa Patel, **Jigna Pathak**, Niharika Swain – Glandular Odontogenic Cyst Mimicker: A Novel Entity. J Contemp Dent .2016: 6(2) 145-148
			29.	Rajshri U Gurav, **Jigna Pathak**, Shilpa Patel, Niharika Swain - Giant Aneurysmal Bone Cyst of the Mandible. J Contemp Dent . 2016; 6(2) 149-153
			30.	Shikha Satish Bhatt, Shilpa Patel, **Jigna Pathak**, Niharika Swain – Osteosarcoma of Mandible. J Contemp Dent. 2016; 6(1) – 70 – 74
			31.	Nikitha Narayanan, **Jigna Pathak**, Shilpa Patel, Niharika Swain – Adenomatoid Odontogenic Cyst. J Contemp Dent. 2016: 6(1) – 96-99
			32.	Janaki Iyer, **Jigna Pathak,** Shilpa Patel, Niharika Swain. Asymptomatic, blue, dome- shaped lesion on buccal mucosa. OOO- 2016;6(6) 578-82
			33.	**Jigna Pathak**, Shilpa Patel, Janaki Subramanian Iyer, Neeta Mohanty – Basaloid squamous cell carcinoma of the maxilla – BMJ Case Report – 2015
			34.	Rathod S, Patel S, **Pathak J**, Swain N. Sub-periosteal Osteid osteoma. J Contemp Dent 2015 ; 5(2): 118-21.
			35.	Hosalkar RM, **Pathak J**, Swain N, Mohanty N. Pagetoidpolycystic fibrous dysplasia.BMJ Case report. 2015doi:10.1136/bcr-2014-209149.
			36.	Patel S,**Pathak J,**Dekate K, Mohanty N .Malignant peripheral nerver sheath tumor (MPNST) of mandible: solving the perplexity.BMJ Case report. 2015.
			37.	Rashmi Hosalkar, Shilpa Patel, **Jigna Pathak**, Niharika Swain, Leela Poonja – Serum Albumin, Globulin and Albumin/ Globulin Ratio in Oral Squamous Cell Carcinoma: A Prospective Study. J Contemp Dent. 2015 ;5(3): 149-52
			38.	Iyer JS, **Pathak J**, Patel S, Poonja L, Swain N – Comparative Evaluation of Micronuclei in Exfoliated Urothelial Cells in Patients with Smoking and Smokeless Tobacco- associated Lesions. J Contemp Dent. 2015; 5(2):93-7
			39.	Rashmi Hosalkar, Shilpa Patel , **Jigna Pathak**, Niharika Swain – Odontogenic Myxoma of Maxilla - J Contemp Dent. 2015;5(1):27-30.
			40.	Janaki S. Iyer, Kartik Poonja ,**Jigna Pathak** , Shilpa Patel , Leela Poonja – Low Grade Central Mucoepidermoid Carcinoma.J Contemp Dent. 2015;5(1): 31-4.
			41.	Niharika Swain, Shilpa Patel, **Jigna Pathak** – Comment on "Tumour thickness as a predictor of nodal metastases in oral cancer: Comparison between tongue and floor of mouth subsites '' by Balasubramanian Detal - Oral Oncol. March -2015
			42.	Pawar V, Patel S, Pathak J, Swain N, Hosalkar R, **Iyer J**. Immunohistochemical evaluation of Calretinin and Cytokeratin 19 in Odontogenic cyst and

	Ameloblastoma: A retrospective study.Journal of Contemporary Dentistry. 2015;5(2): 98 -103.
43.	Vipul Pawar, Shilpa Patel, **Jigna Pathak**, Niharika Swain –Verruciform Xanthoma- Histopathologically: A Distinct Entity - J Contemp Dent. 2014;4(3):181-84.
44.	Niharika Swain, Shwetha V.Kumar,SamapikaRoutray, **Jigna Pathak**, Shilpa Patel – Podoplanin - A novel marker in oral carcinogenesis – Tumor Biology. September 2014; 35 (9): 8407-13
45.	**Pathak J**, Swain N, Shilpa Patel, Poonja L.S – Histopathological variants of oral squamous cell carcinoma-institutionalase reports - J Oral MaxillofacPathol. 2014:18(1): 143-5
46.	Rahul Kadam,ShilpaPatel,**Jigna Pathak**, Niharika Swain,Shwetha Kumar-Trabecular Juvenile Ossifying Fibroma of the Craniofacial Skeleton : Etiopathogenesis and a Case Report of the Rare Entity . J Contemp Dent. 2014;4(1):51-55.
47.	Shah A, Shilpa Patel,**PathakJigna** ,Swain N,Shah S,- An insight into diagnostically challenging salivary gland malignancy with case report : Polymorphous low grade adenocarcinoma – JDRSD 2014 : (1) 20-30
48.	Amit Shah,ShilpaPatel,**JignaPathak**,Swain N ,Kumar Shwetha–The evolving concepts of cancer stem cells in Head and Neek Squamous Cell Carcinoma – The Scientific World Journal. http://dx.doi.org/10.1155/2014/842491
49.	Kelgandre D.C, **Pathak Jigna** ,Patel Shilpa ,Poonja L.S, Swain N, Dekate K – Aneurysmal Bone Cyst with Central Giant Cell iGranuloma in Head and Neck Granuloma of the Mandible : ACaseReort with A Brief Review on Pathogenesis – Journal Of Dental Press ; 1(2) 118-127
50.	Niharika Swain, Shwetha Kmar,**JignaPathak**,Shilpa Patel –Soft tissue myoepithelial carcinoma of neck; A Rare case report with review of literature- Journal of Oral and Maxillofacial Surgery Medicine,and Pathology ;September-2013
51.	Rahul Kadam,ShilpaPatel,**JignaPathak**,Nikharika ,Shwetha Kumar- Focal Cemento-osseous Dysplasia - J Contemp Dent. 2012;3(2):112-115.
52.	Rahul Kadam,ShilpaPatel,**JignaPathak**,Nikharika ,Shwetha Kumar- Focal Cemento-osseous Dysplasia - J Contemp Dent. 2012;3(2):112-115.\
53.	Niharika Swain,**Jigna P.,** Leela S Poonja, Yogita P. – Etiological Factors of Recurrent Aphthous Stomatitis: A Common Perplexity J Contemp Dent. 2012;2(3):96-100.
54.	Niharika Swain,Shilpa Patel, L.S. Poonja, **Jigna Pathak,** Kamlesh Dekate- Orthokeratinized Odontogenic Cyst – J Contemp Dent.2012;2(2):31-33.
55.	Niharika S., Shwetha K, Richa, **and Pathak J**. DEFENSINS: Potent biomarkers in Oral Squamous cell Carcinoma. Oral Oncol. 2012; 48: e29-e30.
56.	Dekate K, Niharika S, **Pathak J**, Poonja L Demoblastic Ameloblastoma. J Contemp Dent. 2012: 2(1); 47-49.
57.	**Pathak J,** Poonja L, Shwetha K, Niharika S, Kamlesh D Concomittant Occurrence of Ameloblastic Fibro-Odontome and Compound Odontoma. JCD. 2011; 5(11):69-73
58.	**Pathak J,** Niharika Swain, Shwetha Kumar. Infiltrative Type of Bone Invasion in Oral Squamous Cell Carcinoma – A Case Report. J Contemp Dent. 2011; 1(2):49-52.
59.	Dekate k, Kini V, Kumar SV, **Pathak Jigna,**Poonja L. – Plasmablastic Lymphoma of Gingiva as Primary Oral Manifestation in Previously undiagnosed HIV Patient - A Case Report. IJOMP. 2011: 2 (2) : 31-34

			60.	**Pathak J**, Poonja L. Diagnostic aids in screening of Oral Cancer. JIDA 2010 March; 4(3):86-88.
			61.	**Pathak J**, Poonja L. Differential Diagnosis of Oral Ulcers Dental Horizon 2009 Dec;3(4):16-19.
			62.	**Pathak J**, Poonja L. Oral Submucous Fibrosis – Revisited. Dental Horizon 2010 August; 5(2):30-31.
11.	Post Graduate Teaching experience (Attach documentary evidence)	:	10 years 03months 15days.	
12.	Any other relevant information	:	-	

Pathak

Dr. Jigna Pathak
Name & Sign. of Mentor

Date: 20 / 05 /2022

For the use of affiliated Training Center:

I have verified the eligibility of the above Director as per the criteria of eligibility prescribed by the University vide clause no.7 of the University Direction No. 05/2017 (Amended).

Pathak

Sign & Stamp
Head of the Department
Date: 20/05/2022

Microbiology
Hospital
Navi Mumbai - 410 209.
Sec. 16, Kamothe

S. Somvally

Sign & Stamp
Dean/ Principal/ Director of Training Centre
Date: 20/05/2022.

Dean
M.G.M. Dental College and Hospital,
Kamothe, Navi Mumbai 410 209.

M.G.M. Dental College & Hospital
Kamothe
410209.

Training Centre Round Seal

मुंबई विद्यापीठ

UNIVERSITY OF MUMBAI

आम्ही मुंबई विद्यापीठाचे कुलपती, कुलगुरू आणि व्यवस्थापन परिषदेचे सदस्य असे प्रमाणित करतो की **पद्मश्री डॉ. डी. वाय. पाटील डेंटल कॉलेज अॅन्ड हॉस्पिटलच्या जीगना प्रियवर्दन देसाई,** त्या एप्रिल १९९७ मध्ये घेण्यात आलेली **दंतशल्य चिकित्सा स्नातक** परीक्षा **द्वितीय श्रेणीत** उत्तीर्ण झाल्या असून दिनांक २ डिसेंबर १९९८ रोजी मुंबई येथे झालेल्या दीक्षांत समारंभात त्यांना **दंतशल्य चिकित्सा स्नातक** ही पदवी प्रदान करण्यात आली आहे.

विद्यापीठाची मुद्रा व कुलपतींची स्वाक्षरी यांसह साक्षीने अंकित.

We, the Chancellor, Vice-Chancellor and Members of the Management Council of the University of Mumbai certify that ***Jigna Priyavadan Desai*** *of* ***Padmashree Dr. D. Y. Patil Dental College and Hospital*** *having passed the* ***Bachelor of Dental Surgery*** *degree examination held in April 1997 in the* ***Second Class,*** *the degree of* ***Bachelor of Dental Surgery*** *has been conferred on her at the Convocation held in Mumbai on 2nd December, 1998.*

In testimony whereof are set the Seal of the said University and the signature of the said Chancellor.

Dean
M. G. M. Dental College & Hospital
Kamothe, Navi Mumbai - 410 209.

मुंबई विद्यापीठ

UNIVERSITY OF MUMBAI

आम्ही मुंबई विद्यापीठाचे कुलपती, कुलगुरू आणि व्यवस्थापन परिषदेचे सदस्य असे प्रमाणित करतो की **नायर हॉस्पिटल डेंटल कॉलेजचे** देसाई जिग्ना प्रियवदन हंसा, ह्या ऑक्टोबर २००१ मध्ये घेण्यात आलेली **दंतशल्य चिकित्सा अधिस्नातक** परीक्षा **शाखा ६: मुखविकृतिशास्त्र आणि सूक्ष्मजीवशास्त्र** या विषयात **उत्तीर्ण** झाल्या असून दिनांक २० डिसेंबर २००२ रोजी मुंबई येथे झालेल्या दीक्षांत समारंभात त्यांना **दंतशल्य चिकित्सा अधिस्नातक** ही पदवी प्रदान करण्यात आली आहे.

विद्यापीठाची मुद्रा व कुलगुरूंची स्वाक्षरी यांसह साक्षीने अंकित.

We, the Chancellor, Vice-Chancellor and Members of the Management Council of the University of Mumbai certify that **Desai Jigna Priyavadan Hansa** of **Nair Hospital Dental College** having passed the **Master of Dental Surgery** degree examination held in October 2001 in the **Passes** with the subject of **Branch VI : Oral Pathology & Microbiology**, the degree of **Master of Dental Surgery** has been conferred on her at the Convocation held in Mumbai on 20th December, 2002.

In testimony whereof are set the Seal of the said University and the signature of the said Vice-Chancellor.

MUMBAI

for Controller of Examinations
22/3/2016 University of Mumbai

Dean
M. G. M. Dental College & Hospital
Kamothe, Navi Mumbai - 410 209.

Sd/-
Bhalchandra L. Mungekar
कुलगुरू VICE CHANCELLOR

DUPLICATE

2002-MDSM-07

YERALA MEDICAL TRUST & RESEARCH CENTRE'S

DENTAL COLLEGE AND HOSPITAL

Institutional Area , Sector - 4 , Kharghar , Navi Mumbai - 410 210

Telefax : Dean - 758 0879 E-mail : ymtden0@netscape.com

Ref No. : YMTD/221/04

Date : 15/04/04

TO WHOMSOEVER IT MAY CONCEREN

This is to certify that Dr. Jigna Pathak ,(M.D.S.) was working in our Institution as a Lecturer in the Department of Oral Pathology & Microbilogy from 01.10.2002 to 31.03.2004

Dr.A.P.Chitre,
Dean Dean
Yerala Medical Trust
Dental College & Hospital
Sector No. 4, Kharghar,
Navi Mumbai-410 210.

Dean
M. G. M. Dental College & Hospital
Kamothe, Navi Mumbai - 410 209.

MAHATMA GANDHI MISSION DENTAL COLLEGE & HOSPITAL
Accredited by NAAC with "A" Grade
Plot No. 1 & 2 Sector-01 (Old 18 & 19),
Kamothe, Navi Mumbai- 410209
E-Mail ID: mgmdch@mgmmumbai.ac.in

Dr. Srivalli Natarajan
Dean

Tel: 022- 27436604
022-27433185

No.MGM/DCH/ 790 /2022

Date: 20/05/2022

Experience Certificate

This is to certify that Dr. Jigna Pathak is working in the Department of Oral Pathology in MGM Dental College & Hospital, Kamothe, Navi Mumbai and her experience is as under :

- Lecturer – 01/04/2004 to 30/09/2005
- Reader – 01/10/2005 to 30/09/2010

1.

- Professor – 01/10/2010 to Till Date.

M.G.M. Dental College & Hospital Kamothe Navi Mumbai 410209.

S. Srivalli.

Dean

महाराष्ट्र आरोग्य विज्ञान विद्यापीठ
MAHARASHTRA UNIVERSITY OF HEALTH SCIENCES
वणी रोड, म्हसरुळ, नाशिक - ४२२ ००४
Vani Road, Mhasrul, Nashik – 422 004
Vidya Thakare
Dy. Registrar
Phone: 0253-2539199/239 / EPABX: 0253-2539100 - 300 / Fax: 0253-2539200
E-mail: pgacademic@muhsnashik.com / Web: www.muhsnashik.com

No. MUHS/ PG/E-2/ PGTRC/ 291 /2012 Date: 30/01/2012

To
The Dean / Principal,
MGM's Dental College & Hospital
Sector–18, Kamothe,
Navi Mumbai – 410 20

Sub:- Recognition as Post-Graduate Teacher's

1) Your college letter No. MGM/DCH/892/2011 dt. 05/11/2011
2) Your college letter No. MGM/DCH/910/2011 dt. 15/11/2011
3) University letter no. MUHS/PG/E-2/PGTRC/2879/2011 dt. 08/12/11
4) PGTRC meeting dated 16/12/2011
5) Your college letter No. MGM/DCH/1000/2011 dt. 13/12/2011
6) University letter No. MUHS/MET,Pune/150/2012 dt. 27/01/2012

Sir/Madam,

With reference to the above cited subject, I am directed to inform you that in view of the norms prescribed as per provision under the section 29 (2) (l) of the MUHS Act, 1998 Hon'ble Vice-Chancellor is pleased to grant recognition as Post-Graduate Teacher to the following teacher(s) of your College **subject to the terms and conditions of appointment order for imparting instructions** to the Post Graduate Degree in the subject mentioned against their name.

Sr. No.	Name of the Teacher	Subject	Status of PG Recognition
01	Dr. Sabita M. Ram	Prosthodontics & Crown & Bridge	w.e.f. 16/12/2011
02	Dr Nadgere Jyoti B	Prosthodontics & Crown & Bridge	w.e.f. 16/12/2011
03	Dr Sonawane Smita R.	Oral & Maxillofacial Surgery	w.e.f. 16/12/2011
04	Dr Pathak Jigna R	Oral Pathology & Microbiology	w.e.f. 16/12/2011

Kindly note that the recognition given by the University is valid till the above said teacher(s) are in services of the private College or attains the age of superannuation whichever is earlier as per the rules made by the University from time to time.

You are requested to handover the copy of letter to the concerned teacher(s).

Yours faithfully,

Dy. Registrar
I/C Academic Section (PG)

Copy to : 1) The Controller of Examinations, MUHS, Nashik
2) The Synopsis Section, MUHS, Nashik

[Note: In case, if it is found at later stage that information furnished in Post Graduate Recognition form by any teacher is incorrect, PG Recognition granted by the University will stand cancelled.]

Morphological analysis of palatal rugae patterns in a population of Maharashtrian ancestry: a cross-sectional study

Vibhuti Shreesh Mhatre[1],
Jigna Pathak [2],
Shilpa Patel [1],
Leela S. Poonja [1],
Niharika Swain [1],
Kamlesh Dekate [1],
Amit Bhandarwar [1]

[1] *Department of Oral & Maxillofacial Pathology, MGM Dental College & Hospital, Navi Mumbai - India*
[2] *Department of Oral & Maxillofacial Pathology, MGM Dental College & Hospital and MGMIHS, Navi Mumbai - India*

Corresponding author:
drjignapathak@gmail.com

The authors declare that they have no conflict of interest.

KEYWORDS

Palatal rugae,
Ancestry,
Identification tool.

J Forensic Odontostomatol
2020. Sep;(38): 2-12:21
ISSN :2219-6749

ABSTRACT

Aim: To analyze the morphological parameters of palatal rugae in a population of Maharashtrian ancestry.

Material and methods: This study was conducted on 1000 subjects of Maharashtrian ancestry with at least 3 generations on the mother's and father's side. Their palatal impressions were obtained with alginate and the casts were analyzed for length, shape and direction of palatal rugae.

Results: Our results showed that the most predominant rugae were primary followed by secondary and fragmentary with significant differences between them. The most prevalent rugae shapes found were straight followed by wavy followed by curved with significant differences between them. According to direction, forward rugae were significantly higher than perpendicular rugae and backward rugae.

Conclusion: The rugae are considered to have population specific configurations. This baseline data of patterns of palatal rugae in a sample of Maharashtrian ancestry may serve `as an accessory tool' for population identification in Forensic Dentistry.

INTRODUCTION

Palatoscopy or palatal rugoscopy is the name given to the study of palatal rugae in order to establish a person's identity. Palatal rugae (rugae palatinae or plicae palatinae transversae) are defined, according to the Glossary of Prosthodontics Terms, as anatomical folds or wrinkles; the irregular ridges of folds of fibrous connective tissue located on the anterior third of the palate behind the incisive papilla. Rugae are secured at an internal position in the oral cavity and are well protected by the lips, cheeks, buccal pad of fat, tongue, teeth and bone and hence are protected from trauma and high temperatures. Their uniqueness, stability and resistance to damage facilitate their use in forensic investigations.

When it is difficult to identify the individual by conventional methods such as fingerprints or DNA analysis, particularly in cases of fragmented bodies in mass disasters, palatal rugoscopy can serve as an alternative method in human identification. There seems to be a remarkable association between rugae forms and ethnicity of a person. Previous literature states that palatal rugae patterns may be specific to racial groups facilitating population identification.[1] The few studies which are done to find this association, show that specific patterns are predominant in specified populations.[1] Hence this study was undertaken to analyze the morphological parameters of palatal rugae and determine the predominant palatal rugae pattern in a population of Maharashtrian ancestry visiting our dental institution.

24. Dawasaz AA, Dinkar AD, "Rugoscopy: predominant pattern, uniqueness and stability assessment in the Indian goan population," J Forensic Sci 2013;58(6):1621–27.
25. Basnet BB, Parajuli PK and Shakya R. A Study of Palatal Rugae Patterns in the Populations of Mongoloid and Tharu Ethnic Groups of Eastern Nepal. Austin J Anat. 2017; 4(2): 1067
26. Rath R, Reginald BA. Palatal rugae: An effective marker in population differentiation. J Forensic Dent Sci 2014; 6:46-50.
27. Shetty DK, Machale PS, Savant SC, Taqi SA. Comparison of palatal rugae patterns in Kodava and Malayalee populations of South India. J Forensic Dent Sci 2013; 5:85-9.
28. Kommalapati RK, Katuri D, Kattappagari KK, Kantheti LPC, Murakonda RB, Poosarla CS, Chitturi RT, Gontu SR, Baddam VRR. Systematic Analysis of Palatal Rugae Pattern for Use in Human Identification between Two Different Populations, Iran J Public Health 2017;46(5):602-607.
29. Saadeh M, Ghafari JG, Haddad RV, Ayoub F, Association Among Geometric Configurations of Palatal Rugae. J Forensic Odonstomatol 2017; 35(1): 33–34.

Review Article

SOX2, OCT4 and NANOG: The core embryonic stem cell pluripotency regulators in oral carcinogenesis

Niharika Swain[1], Mansee Thakur[2], Jigna Pathak[1], Biswaranjan Swain[3]

[1]Department of Oral Pathology, MGM Dental College and Hospital, MGM Institute of Health Sciences, [2]Department of Medical Biotechnology, MGM School of Biomedical Sciences, MGM Institute of Health Sciences, Navi Mumbai, Maharashtra, [3]Department of Electronics and Communications Engineering, Institute of Technical Education and Research, S'O'A Deemed to be University, Bhubaneswar, Odisha, India

Abstract Embryonic stem cells provide their major contribution to embryogenesis through formation of germ layers as they have pluripotency potential and capacity for self-renewal. Retention of pluripotency of these stem cells depends on expression/level of transcription factors, i.e., SOX2, OCT4 and NANOG. During organogenesis, the altered expression of the molecules also influences these stem cells to lose their pluripotency and turn toward the lineage selection. As the differentiation progresses, the maintenance of the somatic cells including the oral squamous cells also depends on differential expression of the transcription factors to some extent. Recently, many experimental and observational studies documented the significant contribution in carcinogenesis of various human cancers. In this review, we have attempted to summarize the evidences indicating about the putative role of these master pluripotency regulators in various phases of oral carcinogenesis i.e. initiation , progression and prognosis of oral squamous cell carcinoma.

Keywords: NANOG, OCT4, oral carcinogenesis, SOX2

Address for correspondence: Dr. Niharika Swain, B-103, Ganesh Plaza, Plot No: A109, Sector-6, Karanjade, Panvel, Navi Mumbai - 410 206, Maharashtra, India.
E-mail: niharikadec30@gmail.com

Submitted: 18-Jan-2020, **Revised:** 08-Jun-2020, **Accepted:** 11-Jun-2020, **Published:** 09-Sep-2020

INTRODUCTION

Stem cells are defined as unspecialized cells which can differentiate into any cell type of an organism and also retain the capacity of self-renewal. Broadly, stem cells can be divided in five groups according to the degree of differentiation potential, i.e., (i) totipotent, (ii) pluripotent, (iii) multipotent, (iv) oligopotent and (v) unipotent stem cells.[1,2] Totipotent stem cells such as zygote and all cells in eight-cell stage morula possess the highest differentiation potential and capacity to form both embryo and extraembryonic structures including the placenta. Pluripotent stem cells retain the ability to differentiate into lineages of all three germ cell layers but cannot generate extraembryonic structures which include embryonic stem cells (ESCs) and induced pluripotent stem cells (iPSCs).[3] ESCs are derived from the inner cell mass (ICM) of blastocysts of embryo whereas iPSCs are genetically reprogrammed and derived from the epiblast layer of implanted embryos. Once stem cells get restricted in a particular tissue, the potency of self-renewable ability decreases further in multipotent stem cells (MSCs) as they lack a high level of telomerase including hematopoietic stem cells and dental pulp stem cells.[4] Furthermore, oligopotent and unipotent stem cells exhibit more restricted lineages

Access this article online

Quick Response Code:

Website: www.jomfp.in

DOI: 10.4103/jomfp.JOMFP_22_20

How to cite this article: Swain N, Thakur M, Pathak J, Swain B. SOX2, OCT4 and NANOG: The core embryonic stem cell pluripotency regulators in oral carcinogenesis. J Oral Maxillofac Pathol 2020;24:368-73.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Zakrzewski W, Dobrzy M, Szymonowicz M, Rybak Z. Stem cells: Past, present, and future. Stem Cell Res Ther 2019;5:1-22.
2. Gawlik-Rzemieniewska N, Bednarek I. The role of NANOG transcriptional factor in the development of malignant phenotype of cancer cells. Cancer Biol Ther 2016;17:1-10.
3. Díez Villanueva P, Sanz-Ruiz R, Núñez García A, Fernández Santos ME, Sánchez PL, Fernández-Avilés F. Functional multipotency of stem cells: What do we need from them in the heart? Stem Cells Int 2012;2012:817364.
4. Bissels U, Diener Y, Eckardt D, Bosio A. Characterization and classifi cation of stem cells. In: Regenerative Medicine – From Protocol to Patient. Cham, Switzerland: Springer International Publishing; 2016. p. 1-25.
5. Zhao W, Ji X, Zhang F, Li L, Ma L. Embryonic stem cell markers. Molecules 2012;17:6196-236.
6. Fritsch MK, Singer DB. Embryonic stem cell biology. Adv Pediatr 2008;55:43-77.
7. Ginis I, Luo Y, Miura T, Thies S, Brandenberger R, Gerecht-Nir S, *et al.* Differences between human and mouse embryonic stem cells. Dev Biol 2004;269:360-80.
8. Kimura H, Tada M, Nakatsuji N, Tada T. Histone code modifications on pluripotential nuclei of reprogrammed somatic cells. Mol Cell Biol 2004;24:5710-20.
9. Hepburn AC, Steele RE, Veeratterapillay R, Wilson L, Kounatidou EE, Barnard A, *et al.* The induction of core pluripotency master regulators in cancers defines poor clinical outcomes and treatment resistance. Oncogene 2019;38:4412-24.
10. Xu J, Zheng Z, Du X, Shi B, Wang J, Gao D, *et al.* A cytokine screen using CRISPR-Cas9 knock-in reporter pig iPS cells reveals that Activin A regulates NANOG. Stem Cell Res Ther 2020;11:67.
11. Kashyap V, Rezende NC, Scotland KB, Shaffer SM, Persson JL, Gudas LJ, *et al.* Regulation of stem cell pluripotency and differentiation involves a mutual regulatory circuit of the NANOG, OCT4, and SOX2 pluripotency transcription factors with polycomb repressive complexes and stem cell microRNAs. Stem Cells Dev 2009;18:1093-108.
12. Verneri P, Vazquez Echegaray C, Oses C, Stortz M, Guberman A, Levi V. Dynamical reorganization of the pluripotency transcription factors Oct4 and Sox2 during early differentiation of embryonic stem cells. Sci Rep 2020;10:5195.
13. Loebel DA, Watson CM, De Young RA, Tam PP. Lineage choice and differentiation in mouse embryos and embryonic stem cells. Dev Biol 2003;264:1-4.
14. Thomson M, Liu SJ, Zou L, Smith Z, Meissner A, Ramanathan S, *et al.* Pluripotency circuit members mediate germ layer fate choice of embryonic stem cells. Cell 2017;145:875-89.
15. Fu TY, Hsieh IC, Cheng JT, Tsai MH, Hou YY, Lee JH, *et al.* Association of OCT4, SOX2, and NANOG expression with oral squamous cell carcinoma progression. J Oral Pathol Med 2016;45:89-95.
16. Qiao B, He B, Cai J, Yang W. The expression profile of Oct4 and Sox2 in the carcinogenesis of oral mucosa. Int J Clin Exp Pathol 2014;7:28-37.
17. Michifuri Y, Hirohashi Y, Torigoe T, Miyazaki A, Kobayashi J, Sasaki T, *et al.* High expression of ALDH1 and SOX2 diffuse staining pattern of oral squamous cell carcinomas correlates to lymph node metastasis. Pathol Int 2012;62:684-9.
18. Moharil RB, Dive A, Khandekar S, Bodhade A. Cancer stem cells: An insight. J Oral Maxillofac Pathol 2017;21:463.
19. Baghai Naini F, Aminishakib P, Abdollahi A, Hodjat M, Mohammadpour H, Kardouni Khoozestani N. Relative expression of OCT4, SOX2 and NANOG in oral squamous cell carcinoma versus adjacent non- tumor tissue. Asian Pac J Cancer Prev 2019;20:1649-54.
20. de Vicente JC, Donate-Pérez Del Molino P, Rodrigo JP, Allonca E, Hermida-Prado F, Granda-Díaz R, *et al.* SOX2 expression is an independent predictor of oral cancer progression. J Clin Med 2019;8:1-14.
21. de Vicente JC, Rodríguez-Santamarta T, Rodrigo JP, Allonca E, Vallina A, Singhania A, *et al.* The emerging role of NANOG as an early cancer risk biomarker in patients with oral potentially malignant disorders. J Clin Med 2019;8:1-16.
22. Kopp JL, Ormsbee BD, Michelle Desler AR. Small increases in the level of Sox2 trigger the differentiation of. Stem Cells 2008;26:903-11.
23. Niwa H, Miyazaki J, Smith AG. Quantitative expression of Oct-3/4 defines differentiation, dedifferentiation or self-renewal of ES cells. Nat Genet 2000;24:372-6.
24. Cai J, He B, Li X, Sun M, Lam AK, Qiao B, *et al.* Regulation of tumorigenesis in oral epithelial cells by defined reprogramming factors Oct4 and Sox2 . Oncol Rep 2016;36:651-8.
25. Chiou SH, Yu CC, Huang CY, Lin SC, Liu CJ, Tsai TH, *et al.* Positive correlations of Oct-4 and Nanog in oral cancer stem-like cells and high-grade oral squamous cell carcinoma. Clin Cancer Res 2008;14:4085-95.
26. Du L, Yang Y, Xiao X, Wang C, Zhang X, Wang L, *et al.* Sox2 nuclear expression is closely associated with poor prognosis in patients with histologically node-negative oral tongue squamous cell carcinoma. Oral Oncol 2011;47:769-13.
27. Freier K, Knoepfle K, Flechtenmacher C, Pungs S, Devens F, Toedt G, *et al.* Recurrent copy number gain of transcription factor SOX2 and corresponding high protein expression in oral squamous cell carcinoma. Genes Chromosomes Cancer 2010;49:9-16.
28. Huang CF, Xu XR, Wu TF, Sun ZJ, Zhang WF. Correlation of ALDH1, CD44, OCT4 and SOX2 in tongue squamous cell carcinoma and their association with disease progression and prognosis. J Oral Pathol Med 2014;43:492-8.
29. Pradhan S, Guddattu V, Solomon MC. Association of the co-expression of SOX2 and Podoplanin in the progression of oral squamous cell carcinomas – An immunohistochemical study. J Appl Oral Sci 2019;27:e20180348.
30. Tsai LL, Hu FW, Lee SS, Yu CH, Yu CC, Chang YC. Oct4 mediates tumor initiating properties in oral squamous cell carcinomas through the regulation of epithelial-mesenchymal transition. PLoS One 2014;9:e87207.
31. Züllig L, Roessle M, Weber C, Graf N, Haerle SK, Jochum W, *et al.* High sex determining region Y-box 2 expression is a negative predictor of occult lymph node metastasis in early squamous cell carcinomas of the oral cavity. Eur J Cancer 2013;49:1915-22.
32. Elkashty OA, Ashry R, Tran SD. Head and neck cancer management and cancer stem cells implication. Saudi Dent J 2019;31:395-416.
33. Villodre ES, Kipper FC, Pereira MB, Lenz G. Roles of OCT4 in tumorigenesis, cancer therapy resistance and prognosis. Cancer Treat Rev 2016;51:1-9.
34. Tsai LL, Yu CC, Chang YC, Yu CH, Chou MY. Markedly increased Oct4 and Nanog expression correlates with cisplatin resistance in oral squamous cell carcinoma. J Oral Pathol Med 2011;40:621-8.

Original Article

Comparative evaluation of staining efficacy of calcofluor white and acridine orange for detection of *Candida* species using fluorescence microscopy – A prospective microbiological study

Vishal Punjabi, Shilpa Patel, Jigna Pathak, Niharika Swain

Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India

Abstract

Context: *Candida* is a yeast-like fungus, and it causes candidiasis. Since it is commonly encountered in many cases, the need of the hour is for rapid and reliable method for identification of these fungi in tissue sections.

Aim: The aim of this study was to evaluate and compare the staining efficacy of calcofluor white (CFW) and acridine orange (AO) for the detection of *Candida* species in formalin-fixed paraffin-embedded tissue samples of oral squamous cell carcinoma (OSCC) using fluorescence microscopy.

Settings and Design: Sample size comprised forty cases of OSCC.

Materials and Methods: Before tissue sampling, a swab of the area was taken, it was immediately inoculated on Sabouraud's dextrose agar media and germ tube test was performed for positive cultures for species identification. Tissue sections were obtained from cases of OSCC from the formalin-fixed paraffin-embedded tissue blocks of the same cases in which microbiological assessment was done at the time of tissue sampling, were stained with CFW and AO stain, respectively, and were examined using a fluorescent microscope.

Statistical Analysis Used: Descriptive statistics were expressed in numbers and percentage. Independent *t*-test (unpaired *t*-test) and Chi-square test were used. $P \leq 0.05$ was taken to be statistically significant.

Results: The mean number of microorganisms per high-power field stained by CFW and AO was 6.35 and 2.57, respectively, and a statistically significant difference ($P \leq 0.001$) was observed. CFW compared to swab culture gave $P = 0.018$, which showed a statistically significant association.

Conclusions: CFW is a better fluorescent stain when compared to AO to detect *Candida* species in tissue sections of OSCC cases.

Keywords: Acridine orange, calcofluor white, *Candida*, fluorescence

Address for correspondence: Dr. Vishal Punjabi, 101/4, Sutkartar CHS, Khar (W), SV Road, Mumbai - 400 052, Maharashtra, India.
E-mail: drvishalpunjabi@gmail.com

Submitted: 24-Dec-2018, **Revised:** 06-Sep-2019, **Accepted:** 06-Jan-2020, **Published:** 08-May-2020

INTRODUCTION

Candida is a yeast-like fungus. It exists in three forms, namely pseudohyphae, yeast and chlamydospore. It reproduces by asexual budding and forms pseudohyphae.[1] These species grow rapidly at 25°C–37°C.[1] These yeasts are relatively common commensal organisms found in the

Access this article online

Quick Response Code:

Website: www.jomfp.in

DOI: 10.4103/jomfp.JOMFP_315_18

How to cite this article: Punjabi V, Patel S, Pathak J, Swain N. Comparative evaluation of staining efficacy of calcofluor white and acridine orange for detection of *Candida* species using fluorescence microscopy – A prospective microbiological study. J Oral Maxillofac Pathol 2020;24:81-6

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

REFERENCES

1. Sivapathasundaram B, Gururaj N. Mycotic infections of the oral cavity. In: Rajendran R, Sivapathasundaram B, editors. Shafer's Textbook of Oral Pathology. 6th ed. India: Elsevier; 2009. p. 363-7.
2. Bancroft J, Floyd A. Light Microscopy. In: Suvarna S, Layton C, Bancroft J, editors. Bancroft's Theory and Practice of Histological Technique, 7th ed. United Kingdom. Churchill Livingstone Elsevier; 2013. p. 63-4.
3. Hageage GJ, Harrington BJ. Use of calcofluor white in clinical mycology. Lab Med 1984;15:109-12.
4. Naik KL, Shetty P, Shroff SE, Karnekar VK, Prasad KM, Madathil LP. Detection of *Candida* species by acridine orange fluorescent dye in exfoliative smears of oral candidiasis. S J Oral Sci 2014;1:41-6.6.
5. Chick EW. Acridine orange fluorescent stain for fungi. Arch Dermatol 1961;83:305-9.
6. Ananthanarayan R, Paniker CK. Textbook of Microbiology. 7th ed. India: Orient Longman Pvt. Ltd.; 2005. p. 617.
7. Marsh PD, Martin MV, Lewis MA, Williams DW. Oral Microbiology. 5th ed. United Kingdom. Elsevier; 2009. p. 173-75.
8. Kumar RS, Ganvir S, Hazarey V. Candida and calcofluor white: Study in precancer and cancer. J Oral Maxillofac Pathol 2009;13:2-8.
9. Jahanshahi G, Shirani S. Detection of *Candida albicans* in oral squamous cell carcinoma by fluorescence staining technique. Dent Res J (Isfahan) 2015;12:115-20.
10. Harrington BJ, Hageage GJ. Calcofluor white: A review of its uses and applications in clinical mycology and parasitology. Lab Med 2003;34:361-7.
11. Chick EW, Behar VS. A simple fluorescent method for the detection of superficial fungi in skin and hair. A combined stain with acridine orange and potassium hydroxide. J Invest Dermatol 1961;37:103-5.
12. Hornstein OP, Grässel R, Schirner E. Prevalence rates of candidosis in leukoplakias and carcinomas of the oral cavity. Arch Dermatol Res 1979;266:99-102.
13. Cawson RA, Binnie WH. Candidal leukoplakia and carcinoma: A possible relationship. In: Mackenzie IA, Dabelsteen E, Squier C, editors. Oral Pre-Malignancy. Iowa: University of Iowa Press; 1980. p. 59-66.
14. Cawson RA. Chronic oral candidiasis and leukoplakia. Oral Surg Oral Med Oral Pathol 1966;22:582-91.
15. Berkovits C, Tóth A, Szenzenstein J, Deák T, Urbán E, Gácser A, *et al.* Analysis of oral yeast microflora in patients with oral squamous cell carcinoma. Springerplus 2016;5:1257.
16. Krogh P, Holmstrup P, Thorn JJ, Vedtofte P, Pindborg JJ. Yeast species and biotypes associated with oral leukoplakia and lichen planus. Oral Surg Oral Med Oral Pathol 1987;63:48-54.
17. Lynch DP, Gibson DK. The use of Calcofluor white in the histopathologic diagnosis of oral candidiasis. Oral Surg Oral Med Oral Pathol 1987;63:698-703.
18. Dass SM, Vinayaraj EV, Pavavni K, Pallam A, Rao MS. Comparison of KOH, calcofluor white and fungal culture for diagnosing fungal onychomycosis in an urban teaching hospital. Indian J Microbiol Res 2015;2:148-53.
19. Pickett JP, Bishop CM, Chick EW, Baker RD. A simple fluorescent stain for fungi. Selective staining of fungi by means of a fluorescent method for mucin. Am J Clin Pathol 1960;34:197-202.
20. Monheit JG, Brown G, Kott MM, Schmidt WA, Moore DG. Calcofluor white detection of fungi in cytopathology. Am J Clin Pathol 1986;85:222-5.

(2)

Chapter 4
Print ISBN: 978-93-89816-20-4, eBook ISBN: 978-93-89816-21-1

Conceptualizing Cancer Stem Cells in Head and Neck Squamous Cell Carcinoma

Amit Shah[1*], Shilpa Patel[1], Jigna Pathak[1], Niharika Swain[1] and Shwetha Kumar[1]

DOI: 10.9734/bpi/eist/v3

ABSTRACT

There is increasing evidence that the growth and spread of cancers is driven by a small subpopulation of cancer stem cells (CSCs) - the only cells that are capable of long-term self-renewal, proliferation and generation of the phenotypically diverse tumor cell population. CSCs have been identified and isolated in a variety of human cancers including head and neck squamous cell carcinoma (HNSCC). Studies of many cancer types including HNSCC have identified CSCs using specific markers, but it is still unclear as to where in the stem cell hierarchy these markers fall. This is compounded further by the presence of multiple CSC subtypes within HNSCC, making investigation reliant on the use of multiple markers. The concept of cancer stem cells may have profound implications for our understanding of tumor biology and for the design of novel treatments targeted toward these cells. In this chapter we explore the current knowledge in CSC markers. We further attempt to conceptualize the role of CSCs in HNSCC – its implication in tumorigenesis and the possible additional approach in current treatment strategies.

Keywords: Cancer stem cells; head and neck squamous cell carcinoma; tumor biology; tumorigenesis.

1. INTRODUCTION

Head and neck squamous cell carcinoma (HNSCC) is the sixth most common type of cancer worldwide, with about 200000 newly diagnosed cases and approximately 128000 deaths per year [1,2] Arising from the epithelium lining, the oral cavity, tongue, pharynx, larynx, and sinonasal tract, HNSCC is more likely to metastasize than other cancers, with around 50% lymph node metastasis at diagnosis [2]. Global increase in incidence and mortality associated with HNSCC have intensified efforts in the field of research pertaining to tumor biology and therapeutics. The mortality due to HNSCC is mainly caused by local recurrence and cervical lymph node metastasis, and occasionally by distant organ metastasis. Research in cancer therapeutics has helped in targeting pathways that appear to contribute in tumourigenesis and metastasis with greater efficacy and fewer unwanted side effects. An important premise guiding this work is the cancer stem cell hypothesis. The cancer stem cell (CSC) theory of tumourigenesis was originally proposed in the late 1970s and were first described in hematologic malignancies in 1994 [3]. Since then, CSC have been identified in multiple other solid organ malignancies, including CNS, pancreatic, lung, colon and recently HNSCC [4-8].

The consensus definition of a cancer stem cell that arrived at an 'American Association of Cancer Research Workshop on cancer stem cell' is a cell within a tumor that possesses the capacity to self-renew and to cause the heterogeneous lineages of cancer cells that comprise the tumor [9]. Various alternative terms have been used in the literature, such as "tumor-initiating cell" and "tumorigenic cell" to describe putative cancer stem cells. The origin of these cells, their role in cancer progression and metastasis, CSC identification and progression and possible therapeutic approaches with special implications on HNSCC are highlighted here.

[1]*Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Kamothe, Navi Mumbai 410209, India.*
[*]*Corresponding author: E-mail: amitshah@dr.com;*

91. Argiris A, Harrington KJ, Tahara M, Jeltje S, Pauline C, Ana FC, et al. Evidence-based treatment options in recurrent and/or metastatic squamous cell carcinoma of the head and neck. Front Oncol. 2017;7:72.
92. Thiery JP. Epithelial–mesenchymal transitions in tumour progression. Nat Rev Cancer. 2002; 2(6):442–54.
93. Kalluri R, Weinberg R. The basics of epithelial–mesenchymal transition. J Clin Invest. 2009; 119(6):1420–8.
94. Major AG, Pitty LP, Farah CS. Cancer stem cell markers in head and neck squamous cell carcinoma. Stem Cells Int. 2013;319489.
95. Smith A, Teknos TN, Pan Q. Epithelial to mesenchymal transition in head and neck squamous cell carcinoma. Oral Oncology. 2013;49(4):287–292.
96. Pannuti A, Foreman K, Rizzo P, Osipo C, Golde T, Osborne B, et al. Targeting notch to target cancer stem cells. Clin Cancer Res. 2010;16:3141-3152.
97. Takahashi-Yanaga F, Kahn M. Targeting wnt signaling: Can we safely eradicate cancer stem cells? Clin Cancer Res. 2010;16:3153-3162.
98. Takezaki T, Hide T, Takanaga H, Nakamura H, Kuratsu J, Kondo T. Essential role of the Hedgehog signaling pathway in human glioma initiating cells. Cancer Sci. 2011;102:1306-1312.
99. Smith J, Ladi E, Mayer-Proschel M, Noble M. Redox state is a central modulator of the balance between self-renewal and differentiation in a dividing glial precursor cell. Proc Natl Acad Sci U S A. 2000;97(18):10032-7.
100. Thomson JA, Itskovitz-Eldor J, Shapiro SS, Waknitz MA, Swiergiel JJ, Marshall VS, et al. Embryonic stem cell lines derived from human blastocysts. Science. 1998;282:1145-7.
101. Zhao Y, Bao Q, Renner A, Camaj P, Eichhorn M, Ischenko I, et al. Cancer stem cells and angiogenesis. Int J Dev Biol. 2011;55(4-5):477-82.
102. Calabrese C, Poppleton H, Kocak M, Hogg TL, Fuller C, Hamner B, et al. A perivascular niche for brain tumor stem cells. Cancer Cell. 2007;11(1):69-82.
103. Krishnamurthy S, Dong Z, Vodopyanov D, Imai A, Helman JI, Prince ME, et al. Endothelial cell-initiated signaling promotes the survival and self-renewal of cancer stem cells. Cancer Res. 2010;70(23):9969-78.
104. Paez-Ribes M, Allen E, Hudock J, Takeda T, Okuyama H, Vinals F, et al. Anti-angiogenic therapy elicits malignant progression of tumors to increased local invasion and distant metastasis. Cancer Cell. 2009;15: 220-231.
105. Tang C, Ang BT, Pervaiz S. Cancer stem cell: Target for anti-cancer therapy. FASEB J. 2007; 21(14):3777-85.

Biography of author(s)

Dr. Amit Shah [MDS]
Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Kamothe, Navi Mumbai 410209, India.

He working as a practicing and consulting Oral and Maxillofacial Pathologist. He completed his post graduation in the subject in the year 2014 securing 2nd rank in Maharashtra University of Health Sciences, Nashik. His area of interest is head and neck oncology, stem cell biology and genetics, and molecular biology. He has made immense work related with these areas and has presented and published research papers in various scientific forums.

DISCLAIMER
This chapter is an extended version of the article published by the same author(s) in the following journal.
The Scientific World Journal, 2014: 8, 2014, Article ID 842491.

ORIGINAL RESEARCH PAPER

Volume-8 | Issue-4 | April-2019 | PRINT ISSN No 2277 - 8179

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

IMMUNOEXPRESSION AND CORRELATION OF TUMOUR INFILTRATING B LYMPHOCYTES, IN ORAL SQUAMOUS CELL CARCINOMA WITH LYMPH NODE STATUS-A RETROSPECTIVE STUDY

Oral Pathology

Dr. Amit U. Bhandarwar* Resident, MDS, Department of Oral Pathology and Microbiology, MGM Dental College & Hospital, Kamothe,Navi Mumbai *Corresponding Author

Dr. Shilpa Patel Professor & Head, MDS, Department of Oral Pathology and Microbiology, MGM Dental College & Hospital, Kamothe, Navi Mumbai.

Dr. Jigna Pathak Professor, MDS, Department of Oral Pathology and Microbiology, MGM Dental College & Hospital, Kamothe, Navi Mumbai

Dr. Niharika Swain Reader, MDS, Department of Oral Pathology and Microbiology, MGM DentalCollege & Hospital, Kamothe, Navi Mumbai

Dr. Vibhuti S. Mhatre Resident, MDS, Department of Oral Pathology and Microbiology, MGM Dental College & Hospital, Kamothe, Navi Mumbai

Dr. Satyajit Tekade Professor & Head, MDS, Department of Oral Pathology and Microbiology, Modern Dental College & Research Center, Indore

ABSTRACT

Objectives: To evaluate and correlate the immunoexpression of (TIBCs) Tumour Infiltrating B cells (CD20+) in Oral Squamous Cell Carcinoma with lymph node status.

Material And Methods: The 30 paraffin blocks of histopathologically diagnosed cases of OSCC treated with neck dissection which was retrieved from the archives of the Department of Oral Pathology and Microbiology. The sample size of (n=15) cases showing lymph node metastasis pN(+) & (n=15) without lymph node involvement pN(-). We evaluated the expression of TIBCs using antibodies specific for B cell, in OSCC and correlated the same with lymph node status.

Results: On statistically evaluating and correlating the mean immunoexpression of TIBCs (CD20+) with pathologic lymph node status in tumour front area of primary tumour, we found that the mean count of TIBCs (CD20+) in pN(+) cases was 21.92 whereas in pN(-) cases it was 22.51 (p value : 0.902) by **Mann Whitney Test.**

Conclusion: It is important to understand the diverse role of B cells & its immune response in cancer, yielding a novel role as a predictive & prognostic marker which impacts the therapeutic approaches.

KEYWORDS

B lymphocytes, TIBCs, OSCC

INTRODUCTION:

Oral cancer is a serious and growing problem in many parts of the World.[1] Carcinogenesis is a multistep process which mainly comprises of cellular or molecular changes in host cells as well as alteration in interaction between transformed cells and host defence system.[2] In addition to this, host defence system play a vital role in either promotion or regression during the process of carcinogenesis.[2] Immune cells that infiltrate tumors engage in an extensive and dynamic crosstalk with cancer cells. [3]. B cells play decisive roles in immunosuppression and in regulating antitumour response and in carcinogenesis.[4][5] The potential contribution of B cells in modulating the immune response to tumour development is less investigated. Thus the aim of this study was to evaluate morphological distribution & prognostic impact of B cell lymphocytes in correlation with lymph node metastasis, as in OSCC loco-regional lymph node metastasis is the most significant adverse prognostic marker and major determinant of poor survival rate.

MATERIALS AND METHODS

This study was conducted on 30 paraffin blocks of histopathologically diagnosed cases of OSCC treated with neck dissection which were retrieved from the archives of the Department of Oral Pathology and Microbiology. The study was carried out for a duration of 2 years from 2015-2017. Out of 30 samples, (n=15) cases showing without lymph node metastasis (pN-) **[figure** 1] & (n=15) with lymph node involvement (pN+) **[figure** 2]. The recurrent cases of OSCC were excluded from the study. Sections from the tumor proper were subjected to IHC staining technique with the CD20 marker for B lymphocytes. Evaluation of the Tumour infiltrating B lymphocytes (TIBCs) in tumor front area, correlated with lymph node status, of OSCC cases was done. **[Figure** 3, **figure** 4].

Method of analysis: Five randomly selected high power fields (HPF) 400X magnification, in the tumour front area of primary tumor were chosen and mean number of these B cells was calculated. **Mann-Whitney Test** was used to compare the mean immunoexpression of TIBCs (Cd20+), with the lymph node status. A significance level of 0 .05 was applied to decide the statistical significance of the hypothesis being tested.

Fig. 1 Section of pN(-) lymphnode. [H & E, 40x]

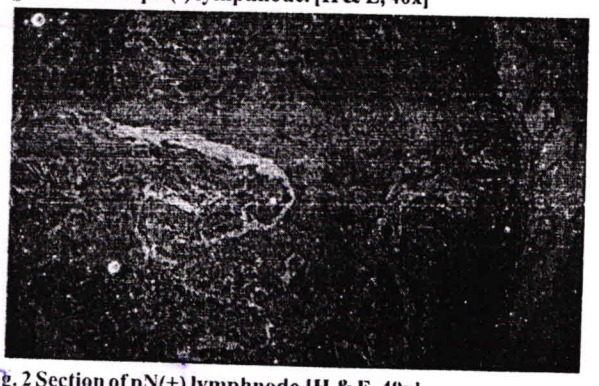


Fig. 2 Section of pN(+) lymphnode.[H & E, 40x]

yielding a novel role as a predictive & prognostic marker which impacts the therapeutic approaches. Till date, few studies have evaluated dynamics of B cells in pre and post specific anti-cancer therapy. Therefore, there is an urge for further studies authenticating specifically the role of B lymphocytes in OSCC which needs to be undertaken for establishing it as a novel immunotherapeutic biomarker.

CONFLICT OF INTEREST: Nil

REFERENCES

1. Warnakulasuriya Saman. Global epidemiology of oral and oropharyngeal cancer oral oncology.2009;45:309-316.
2. Williams HK. Molecular pathogenesis of oral squamous carcinoma. Mol Pathol. 2000 Aug;53(4):165-72.
3. Sergei I. Grivennikov1, Florian R. Greten2. and Michael Karin1 Immunity, Inflammation, and Cancer Cell. 2010 March 19; 140(6): 883–899.
4. Spaner D. and Angela Bahlo B Lymphocytes in Cancer Immunology J. Medin and D. Fowler (eds.), Experimental and Applied Immunotherapy, 2011, 37978-1-60761-980-2_2.
5. Tsou P, Katayama H, Ostrin EJ, Hanash SM. The Emerging Role of B Cells in Tumor Immunity. Cancer Res. 2016 Oct 1;76(19):5597-5601.
6. Mahmoud SM, Lee AH, Paish EC, Macmillan RD, Ellis IO, Green AR. The prognostic significance of B lymphocytes in invasive carcinoma of the breast. Breast Cancer Res Treat. 2012 Apr;132(2):545-53.
7. Pretscher D, Distel LV, Grabenbauer GG, Wittlinger M, Buettner M, Niedobitek G. Distribution of immune cells in head and neck cancer: CD8+ T-cells and CD20+ B- cells in metastatic lymph nodes are associated with favourable outcome in patients with oro- and hypopharyngeal carcinoma. BMC Cancer. 2009 Aug 22;9:292.
8. Ou Z, Wang Y, Liu L, Li L, Yeh S, Qi L, Chang C. Tumor microenvironment B cells increase bladder cancer metastasis via modulation of the IL-8/androgen receptor (AR)/ MMPssignals,impactjournals.com/oncotarget/ Vol. 6, No. 28, July 17, 2015
9. Podolsky MA, Bailey JT, Gunderson AJ, Oakes CJ, Breech K, Glick AB. Differentiated State of Initiating Tumor Cells Is Key to Distinctive Immune Responses Seen in H-Ras(G12V)-Induced Squamous Tumors. Cancer Immunol Res. 2017 Mar; 5(3):198-210.
10. Woo JR, Liss MA, Muldong MT, Palazzi K, Strasner A, Ammirante M, Varki N, Shabaik A, Howell S, Kane CJ, Karin M, Jamieson CA. Tumorinfiltrating B-cells are increased in prostate cancer tissue. J Transl Med. 2014 Jan 30;12:30.
11. Quan N, Zhang Z, Demetrikopoulos MK, Kitson RP, Chambers WH, Goldfarb RH,Weiss JM.Evidence for involvement of B lymphocytes in the surveillance of lung metastasis in the rat. Cancer Res. 1999 Mar 1;59(5):1080-9.

Case Report

Benign cementoblastoma involving left deciduous first molar: A case report and review of literature

Jigna Pathak[1], Rashmi Maruti Hosalkar[1], Sunil Sidana[2], Niharika Swain[1], Shilpa Patel[1]

Deparments of [1]Oral Pathology and Microbiology and [2]Oral and Maxillofacial Surgery, MGM's Dental College and Hospital, Navi-Mumbai, Maharashtra, India

Abstract Cementoblastoma, a benign mesenchymal odontogenic neoplasm is derived from ectomesenchymal cells of the periodontium. Cementoblastomas associated with primary teeth are extremely rare as permanent mandibular first molars are mostly affected. Only 17 cases of those associated with deciduous dentition have been reported so far. The present case report describes a true cementoblastoma of an 8-year-old male child in relation to the left first primary mandibular molar along with emphasis on differential diagnosis.

Keywords: Cementoblastoma, deciduous dentition, differential diagnosis, odontogenic neoplasm

Address for correspondence: Dr. Jigna Pathak, Deparment of Oral Pathology and Microbiology, MGM's Dental College and Hospital, Navi-Mumbai, Maharashtra, India.
E-mail: drjignapathak@gmail.com
Received: 18.06.2019, **Accepted:** 19.07.2019

INTRODUCTION

Cementoblastoma is a slow-growing, benign odontogenic neoplasm of mesenchymal origin, with unlimited growth potential and is derived from ectomesenchymal cells of the periodontium including cementoblasts.[1] Cementoblastoma was first described by Dewey in 1927[2] and was recognized first by Noeberg[1] in 1930. They are commonly seen in children and young adults; males are more frequently affected than females, with more occurrences in mandible than maxilla. Radiographically, benign cementoblastoma appears as a well-defined radio-opacity with a radiolucent peripheral zone. The growth rate for cementoblastoma is estimated to be 0.5 cm/year.[3] The histological features of cementoblastoma include cementum-like tissue with numerous reversal lines, and between these mineralized and trabecular hard tissues, fibrovascular tissue with cementoblast-like cells is present along with multinucleated giant cells.[4] The treatment of choice is complete removal of the lesion with extraction of associated tooth, followed by thorough curettage and peripheral ostectomy. The recurrence rate is 21.7%–37.1%.[3] It is a rare tumor with <300 cases ever reported in literature.[5] Cementoblastoma is more commonly associated with permanent mandibular first molars with deciduous teeth being rarely involved.[6] So far, only 17 cases[6-22] involving deciduous dentition have been reported [Table 1]. The present case report describes a true cementoblastoma in relation to the left first primary mandibular molar in an 8-year-old child along with emphasis on differential diagnosis.

CASE REPORT

An 8-year-old healthy male child reported to the Department of Oral and Maxillofacial Pathology of our

Access this article online

Quick Response Code:

Website: www.jomfp.in

DOI: 10.4103/jomfp.JOMFP_193_19

How to cite this article: Pathak J, Hosalkar RM, Sidana S, Swain N, Patel S. Benign cementoblastoma involving left deciduous first molar: A case report and review of literature. J Oral Maxillofac Pathol 2019;23:422-8.

Oral and Maxillofacial Pathology. Ch. 5. St Louis: Mosby; 2004. p. 153-4.
28. Kalburge JV, Kulkarni MV, Kini Y. Cementoblastoma affecting the mandibular first molar-a case report. Pravara Med Rev 2010;5:33-7.
29. Marx R, Stern D. Odontogenic Tumours, In: Oral and Maxillofacial Pathology: A Rationale for Diagnosis and Treatment. 2nd ed Ch. 15. Vol II. Illinois: Quintessence Publishing House; 2012. p. 704-6.
30. Monks FT, Bradley JC, Turner EP. Central osteoblastoma or cementoblastoma? A case report and 12 year review. Br J Oral Surg 1981;19:29-37.
31. Curé JK, Vattoth S, Shah R. Radiopaque jaw lesions: An approach to the differential diagnosis. Radiographics 2012;32:1909-25.
32. Napier Souza L, Monteiro Lima Júnior S, Garcia Santos Pimenta FJ, Rodrigues Antunes Souza AC, Santiago Gomez R. Atypical hypercementosis versus cementoblastoma. Dentomaxillofac Radiol 2004;33:267-70.
33. Cundiff EJ 2nd. Developing cementoblastoma: Case report and update of differential diagnosis. Quintessence Int 2000;31:191-5.
34. Sharma N. Benign cementoblastoma: A rare case report with review of literature. Contemp Clin Dent 2014;5:92-4.
35. Mudhiraj PV, Vanje MM, Reddy BN, Ahmed SA, Suri C, Taveer S, *et al.* Nature of hard tissues in oral pathological lesions – Using modified Gallego's stain. J Clin Diagn Res 2017;11:ZC13-5.

JCD

10.5005/jp-journals-10031-1243

Oral Verrucous Hyperplasia: A Case Series

[1]Mitesh N Jain, [2]Shilpa Patel, [3]Kamlesh Dekate, [4]Jigna Pathak, [5]Ketki J Shirke, [6]Tanvi Patel

ABSTRACT

Oral verrucous hyperplasia (OVH) is a slow growing, soft tissue premalignant lesion which can transform into oral cancer. Areca-nut and quid use do seem to have a significant influence on the appearance of oral verrucous hyperplasia. Most frequently observed sites are buccal mucosa and lateral border of the tongue. OVH begins as a white plaque of hyperkeratosis known as plaque type variant which can get further transformed into mass type with less keratinization, exophytic growth and proliferative features thus leading to malignant transformation and poorer prognosis. This article describes series of cases which have been diagnosed histopathologically as verrucous hyperplasia with its clinical presentation and histopathological variants along with the criteria elaborated by different authors in establishing a diagnosis and a brief overview of the treatment modalities.

Keywords: Oral verrucous hyperplasia, Verrucopapillary lesions, Verrucous carcinoma, Verrucous hyperplasia.

How to cite this article: Jain MN, Patel S, Dekate K, Pathak J, Shirke KJ, Patel T. Oral Verrucous Hyperplasia: A Case Series. J Contemp Dent 2018;8(3):163-167.

Source of support: Nil

Conflict of interest: None

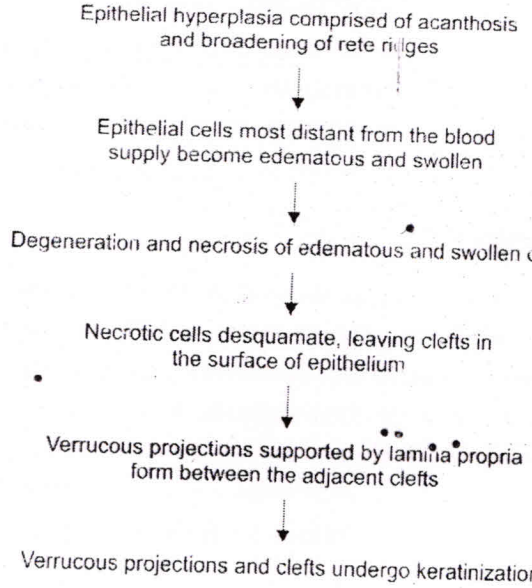
INTRODUCTION

Verrucous papillary lesions (VPLs) of the oral cavity are diagnostically challenging as they include a spectrum of benign, potentially malignant, and frankly malignant lesions.[1] VPL clinically presents themselves as a grey-white, exophytic growth in gingiva, buccal mucosa or any other part of the oral cavity and histopathologically they may range from a simple hyperkeratotic lesion to verrucous hyperplasia, verrucous carcinoma or even frank squamous cell carcinoma.[2]

Oral verrucous hyperplasia (OVH) is reclassified as "plaque" and "mass" variants while further stating that clinically both the variants can be called OVH, but histopathologically only "mass" variant fits the bill to be called the same and "plaque" type lesions can be called an oral verruciform leukoplakia.[3] Over some time, it has been observed that clinicians have found it difficult to distinguish verrucous hyperplasia from verrucous carcinoma due to its marked similarity in clinical appearance. Verrucous hyperplasia with the malignant potential of 3.1% over an average of 54.6 months is a high-risk lesion,[4] along with the fact that it has coexisted with verrucous carcinoma in 29% of cases, and therefore makes it a lesion to be reckoned with. Various treatment modalities include surgery, chemotherapy, radiation or combinations of these and photodynamic therapy which has been recently reported.

Flowchart 1 depicting histogenesis of verrucous hyperplasia:[5]

Flow Chart 1: Histogenesis of verrucous hyperplasia



CASE SERIES

Five cases of histopathologically diagnosed verrucous hyperplasia (Figs 1 to 4) from the year 2015 to 2016 have been retrieved from the archives of the Department of oral pathology and microbiology. Clinical and histopathological observations are summarized in Table 1.

DISCUSSION

Verrucous papillary lesions (VPLs) are a heterogeneous group of lesions, among which OVH has evoked considerable attention, as they have been found to be potentially malignant. Shear and Pindborg also observed difficulty in distinguishing verrucous hyperplasia (VH) and verrucous carcinoma (VC) clinically, and they first reported

[1]Postgraduate Student, [2]Professor and Head, [3]Associate Professor, [4]Professor

Department of Oral Pathology and Microbiology, Mahatma Gandhi Mission's Dental College and Hospital, Kamothe, Navi Mumbai, India

Corresponding Author: Mitesh N Jain, Postgraduate Student, Department of Oral Pathology and Microbiology, Mahatma Gandhi Mission's Dental College and Hospital, Kamothe, Navi Mumbai, India, email id: miteshjn749@gmail.com

were treated with wide excisions and the rest three were incisional biopsies. In terms of treatment modality, wide surgical excision of the lesion with adequate adjacent soft tissue margins to avoid recurrence with strict follow-up is the choice of treatment. However, the use of photodynamic therapy (PDT) is another effective treatment option for human premalignant and malignant lesions, because it is noninvasive, is well tolerated by patients, can be used repeatedly without cumulative side effects, and results in little scar formation. Two studies have shown that topical 5-aminolevulinic acid-mediated photodynamic therapy (ALA-PDT) can be used successfully for the treatment of OVH.[12] Though it is not difficult to diagnose verrucous lesion on the basis of their verrucous surface clinically, it is crucial that biopsies of verrucous lesions include a sufficient margin with adequate depth to get to a confirmatory diagnosis as verrucous hyperplasia has been seen coexisting with other lesions of the spectrum and more so with the recent study where it was observed that delay in treatment for the 'mass' type eventually leads to malignancy with a malignant transformation rate of 7% as compared to the plaque type which showed a malignant transformation rate of 3%[7] (Table 2).

CONCLUSION

It is evident from our case series that clinical diagnostic confusion still exists when it comes to VPL. To distinguish verrucous carcinoma from verrucous hyperplasia (plaque and mass type) histologically, it is necessary to include normal epithelium to ensure the right diagnosis. As it has malignant transformation potential, patients have to be treated in a manner similar to verrucous carcinoma.

REFERENCES

1. Kallarakkal TG, Ramanathan A, Zain RB. Verrucous papillary lesions: Dilemmas in diagnosis and terminology. International journal of dentistry. 2013;2013.
2. Zain R, Kallarakkal T, Ramanathan A, Kim J, Tilakaratne WM, Takata T et al. Exophytic Verrucous Hyperplasia of the Oral Cavity – Application of Standardized Criteria for Diagnosis from a Consensus Report. Asian Pacific Journal of Cancer Prevention 2016;17(9):4491-4501
3. Patil S, Warnakulasuriya S, Raj T, Sanketh DS, Rao RS. Exophytic oral verrucous hyperplasia: a new entity. Journal of investigative and clinical dentistry. 2016 Nov;7(4):417-423.
4. Grover S, Jha M, Sharma B, Kapoor S, Mittal K, Parakkat NK, Shivappa AB, Kaur R. Verrucous Hyperplasia: Case report and differential diagnosis. Sultan Qaboos University Medical Journal. 2017 Feb;17(1):e98.
5. Shear M, Pindborg JJ Verrucous hyperplasia of the oral mucosa. Cancer. 1980;46:1855-1862.
6. Murrah VA, Batsakis JG. Proliferative verrucous leukoplakia and verrucous hyperplasia. Ann Otol Rhinol Laryngol. 1994;103:660-663.
7. Wang YP, Chen HM, Kuo RC, Yu CH, Sun A, Liu BY, Kuo YS, Chiang CP. Oral verrucous hyperplasia: histologic classification, prognosis, and clinical implications. Journal of Oral Pathology & Medicine. 2009 Sep;38(8):651-656.
8. Hazarey V, Ganvir S, Bodnade A. Verrucous hyperplasia: A clinico-pathological study. J Oral Maxillofac Pathol. 2011 May-Aug; 15(2): 187–191.
9. Slootweg PJ, Muller H. Verrucous hyperplasia or verrucous carcinoma: An analysis of 27 patients. J Maxillofac Surg. 1983;11(1):13-19.
10. Klieb H, Raphael SJ. Comparative study of the expression of P53, Ki67, E-cadherin and MMP-1 in verrucous hyperplasia and verrucous carcinoma of the oral cavity. Head Neck Pathol. 2007 Dec; 1(2):118-122.
11. Paral KM, Taxy JB, Lingen MW. CD34 and αsmooth muscle actin distinguish verrucous hyperplasia from verrucous carcinoma. Oral surgery, oral medicine, oral pathology and oral radiology. 2014;117(4):477-482.
12. Chang Y, Yu C. Successful treatment of a large oral verrucous hyperplasia with photodynamic therapy combined with cryotherapy. Journal of Dental Sciences. 2013(8):87e90.

JCD

REVIEW ARTICLE

10.5005/jp-journals-10031-1238

Oral Lichen Planus–A Brief Review on Treatment Modalities

[1]Ketki J Shirke, [2]Jigna Pathak, [3]Niharika Swain, [4]Shilpa Patel, [5]Tanvi Patel, [6]Mitesh N Jain

ABSTRACT

Lichen planus is an autoimmune-mediated chronic inflammatory disease of unknown etiology, but studies have reported the role of cytotoxic T cells responsible for the disruption of basal keratinocytes and also causing the clinical symptoms. It is commonly seen in adults, with rare occurrence in children. It clinically manifests on the skin and oral mucosa, with skin lesions healing faster than the oral lesions. To obtain a diagnosis, a complete history and characteristic clinical features are usually sufficient for diagnosis, but there are certain other lesions like lichenoid reaction, contact sensitivity, white sponge nevus, pemphigoid and lupus erythematosus that show similar clinical characteristics, hence the need for histopathological evaluation using standard criteria given by Krutchkoff or World Health Organization (WHO). The treatment administered is always for eliminating symptoms and discomfort of the patients. A variety of pharmacological and natural alternatives have been used along with frequent follow up visits in case of a tropic and erosive lichen planus. The purpose of this paper is to review the current trends in the management of oral lichen planus.

Keywords: Lichen planus, Management, Update.

How to cite this article: Shirke KJ, Pathak J, Swain N, Patel S, Patel T, Jain M. Oral Lichen Planus–A Brief Review on Treatment Modalities. J Contemp Dent 2018;8(3):137-143.

Source of support: Nil

Conflict of interest: None

INTRODUCTION

Oral lichen planus (OLP) is a chronic mucocutaneous disease of multifactorial etiologies with Wilson first describing it in 1869. It was suggested by authors to an autoimmune disease triggered by antigens in the form of extrinsic or intrinsic factors that activate the lymphocytes and releases cytokines that are directed against the basilar keratinocytes leading to their apoptosis. It was seen to affect 0.5–1% of the worldwide population and 0.1–1.5% prevalence in India.[1]The condition affects the cutaneous or mucosal areas or both, where 50% of individuals with cutaneous lichen planus had oral mucosal lesions, and about 25% presented with, oral mucosal lesions alone.[2] It was seen most commonly in adults and had a female prediliction.[3]

An OLP commonly affects the buccal mucosa, gingiva, and the tongue, with an uncommon presentation on the palate.[4] It is usually seen as multiple lesions, bilaterally present in the mouth persisting for a longer duration of up to 25 years in comparison to the cutaneous counterpart.[5,6] It shows a chronic course having periods of dormancy and flare-ups with spontaneous remissions rarely seen.[7]

Andreasen[2] divided oral lichen planus into six clinical types: reticular, plaque-like, papular, erosive, bullous, and atrophic types where erosive and atrophic types caused discomfort and painful symptoms. On clinical examination, in the absence of the reticular type which is easily identifiable, the other types of oral lichen planus requires histopathological evaluation for a definite diagnosis. This is done using WHO criteria (2003) for diagnosis of OLP, that includes clinical criteria's, histopathological criteria's and final differentiation of lichen planus from lichenoid lesions.[8]

An OLP is a disease with a potential for malignant transformation. It has been noted that the rate of malignant transformation has decreased from 5.9% in 1924 to 0.5–1.1% in 2017.[9] However, this possibility may be reduced by patient counseling, consumption of a healthy diet and avoiding carcinogens.[10] The standard protocol for the management of oral lichen planus includes symptomatic relief with no complete cure. Various alternatives have been applied in the management which suggests the inadequacy of any single drug to provide relief. The present article hereby provides an overview of different treatment modalities in the management and the advancements made in obtaining control over the symptoms of OLP.

The OLP could be symptomatic (erosive, atrophic and bullous types) or asymptomatic (reticular or plaque types). The symptomatic oral lichen planus can cause a burning sensation, severe pain, inability to speak and swallow, which is seen to be the chief complaint of the patient requiring symptomatic relief[11] while the asymptomatic forms do not require pharmacological interven-

[1]Postgraduate Student, [2]Professor, [3]Lecturer, [4]Professor and HOD

[5]Department of Oral Pathology and Microbiology, Mahatma Gandhi Mission Dental College and Hospital, Navi Mumbai, Maharashtra, India

Corresponding Author: Ketki J Shirke, Postgraduate Student, Department of Oral Pathology and Microbiology, Mahatma Gandhi Mission Dental College and Hospital, Navi Mumbai, Maharashtra, India, Mobile: +918149235625, e-mail: shirkeketki09@gmail.com

34. Diana Mostafa, Bassel Tarakji. Photodynamic Therapy in Treatment of Oral Lichen Planus. J Clin Med Res. 2015 Jun;7(6):393-399.

35. Borgia F, Giuffrida R, Caradonna E, Vaccaro M, Guarneri F, Cannavò S. Early and late onset side effects of photodynamic therapy. Biomedicines. 2018 Mar;6(1):12.

36. Jajarm HH, Falaki F, Mahdavi O. A comparative pilot study of low intensity laser versus topical corticosteroids in the treatment of erosive-atrophic oral lichen planus. Photomedicine and laser surgery. 2011 Jun 1;29(6):421-425.

37. Clinton SK. Lycopene: chemistry, biology, and implications for human health and disease. Nutrition reviews. 1998 Feb 1;56(2):35-51.

38. Saawarn N, Shashikanth MC, Saawarn S, Jirge V, Chaitanya NC, Pinakapani R. Lycopene in the management of oral lichen planus: a placebo-controlled study. Indian Journal of Dental Research. 2011 Sep 1;22(5):639-643.

39. Keshari D, Patil K, Mahima VG. Efficacy of topical curcumin in the management of oral lichen planus: A randomized controlled-trial. J Adv Clin Res Insights. 2015;2:197-203.

40. Amirchaghmaghi M, Pakfetrat A, Delavarian Z, Ghalavani H, Ghazi A. Evaluation of the Efficacy of Curcumin in the Treatment of Oral Lichen Planus: A Randomized Controlled Trial. Journal of clinical and diagnostic research: JCDR. 2016 May;10(5):ZC134-ZC137.

41. Prasad S, Solanki S, Chinmaya BR, Tandon S, Ashwini B. The magic of herbal curcumin therapy in recurrent oral lichen planus. Am J Ethnomed. 2014;1:96-101.

42. Reddy RL, Reddy RS, Ramesh T, Singh TR, Swapna LA, Laxmi NV. Randomized trial of aloe vera gel vs triamcinolone acetonide ointment in the treatment of oral lichen planus. Quintessence international. 2012 Oct 1;43(9).

43. Zhang J, Zhou G. Green tea consumption: an alternative approach to managing oral lichen planus. Inflammation Research. 2012 Jun 1;61(6):535-539.

44. Preda EG, Pasetti P, Caggiula S, Nidoli G, Boggio E, Azzi R. Oral pathology of pscyhosomatic origin. Review of the literature. Dental Cadmos. 1990 Jan;58(1):66-72.

45. Akay A, Pekcanlar A, Bozdag KE, Altintas L, Karaman A. Assessment of depression in subjects with psoriasis vulgaris and lichen planus. Journal of the European Academy of Derma-tology and Venereology. 2002 Jul;16(4):347-352.

46. Rojo-Moreno J, Bagán J, Rojo-Moreno J, Donat JS, Milián MA, Jiménez Y. Psychologic factors and oral lichen planus: a psychometric evaluation of 100 cases. Oral surgery, oral medicine, oral pathology, oral radiology, and endodontology. 1998 Dec 1;86(6):687-691.

47. Radwan-Oczko M, Zwyrtek E, Owczarek JE, Szcze-niak D. Psychopathological profile and quality of life of patients with oral lichen planus. Journal of Applied Oral Science. 2018; 26.

JCD

10.5005/jp-journals-10031-1241

Dermoid and Epidermoid Cysts: A Case Series

[1]Tanvi Patel, [2]Shilpa Patel, [3]Jigna Pathak, [4]Niharika Swain, [5]Mitesh Jain, [6]Ketki J Shirke

ABSTRACT

Epidermoid and dermoid cysts of the skin are commonly encountered in surgical practice. Epidermoid cysts and dermoid cysts are uncommon developmental and acquired cystic malformations. Dermoid and epidermoid cysts occur less frequently in the head and neck region. These cysts constitute for 1.6 to 6.9% of all cysts in the head and neck area. However, the intraoral epidermoid and dermoid cysts are very rare and account for less than 0.01% of all cysts in the oral cavity. Here we present a series of five cases of dermoid and epidermoid cysts. The study was carried out on histopathologically diagnosed cases of epidermoid cysts and dermoid cysts were retrieved from the archives of Department of Oral and Maxillofacial Pathology from a period of 2015 to 2018. Four cases of epidermoid cysts and one case of dermoid cyst were reported. This article mentions the distinguishing histopathological features of these two cysts which help the surgeon to carry out a definitive treatment protocol

Keywords: Dermoid cyst, Epidermoid cyst, Malignant transformation Oral, Rare.

How to cite this article: Patel T, Patel S, Pathak J, Swain N, Jain M, Shirke KJ. Dermoid and Epidermoid Cysts: A Case Series. J Contemp Dent 2018;8(3):153-156.

Source of support: Nil

Conflict of interest: None

BACKGROUND

Epidermoid and dermoid cysts of the skin are commonly encountered in surgical practice. However, the epidermoid and dermoid cysts are rarely encountered within the oral cavity. In 1955, the concept of the dermoid cyst was updated by Meyer. Three histological variants were given which were: a true dermoid cyst, the epidermoid cyst, and the teratoid variant.[1] Epidermoid cysts and dermoid cysts are uncommon developmental and acquired cystic formations. The dermoid cysts are more often congenital whereas the epidermoid cysts are most often acquired ones.[2] Epidermoid cysts are rare, slowly growing, benign cysts. Around 7% of the cysts are located in the head and neck region.[3] They are derived from abnormally growing ectodermal tissue. These cysts grow anywhere in the body. Histopathologically, they are lined by stratified squamous epithelium and lumen without skin appendages.[4] Dermoid cysts of the head and neck are believed to represent sequestration of the epidermis along the lines of embryonic closure. They are the developmental cysts usually occurring during the third to fifth week of embryological development.[5] Lesions occur on the scalp, glabella and the bridge or dorsum of the nose and they usually present as solitary lesions. Histologically the dermoid cyst shows keratinized stratified squamous epithelium along with sebaceous glands, sweat glands, and hair follicles.

Dermoid and epidermoid cysts occur less frequently in the head and neck region. These cysts constitute for 1.6 to 6.9 % of all cysts in the head and neck area.[6] However the intraoral epidermoid and dermoid cysts are very rare and account for less than 0.01% of all cysts in the oral cavity.[7]

In this article, we present a case series emphasizing on the differentiating histological features of the epidermoid cyst and dermoid cyst.

SERIES OF CASE REPORTS

A total of five histopathologically diagnosed cases of epidermoid cysts and dermoid cysts, retrieved from the archives of Dept. of Oral and Maxillofacial Pathology from a period of 2015 to 2018 were evaluated. Four cases of epidermoid cysts and one case of dermoid cyst were studied. All the cases were re-evaluated clinicopathologically. Both macroscopic and microscopic histopathological features were noted (Table 1). The microscopic features were evaluated according to the type of keratinization present, features present in the lining and the connective tissue wall, presence of epidermis and skin appendages (Figs 1 to 5).

DISCUSSION

The developmental cutaneous cysts occurring in the head and neck region are the dermoid and epidermoid cysts. However, these cysts rarely occur intraorally. A clear distinction has been made from the common inclusion cysts of the skin which are a sebaceous cyst, milia, epidermal cyst or trichilemmal cyst. The terms sebaceous cyst and milia are used clinically and no longer have any pathological connotation. Epidermal cysts and trichilemmal

[1]Postgraduate Student, [2]Professor and Head, [3]Professor, [4]Reader

[5]Department of Oral Pathology and Microbiology, Mahatma Gandhi Mission's Dental College and Hospital, Kamothe, Navi Mumbai, India

Corresponding Author: Tanvi Patel, Postgraduate Student, Department of Oral Pathology and Microbiology, Mahatma Gandhi Mission's Dental College and Hospital, Kamothe, Navi Mumbai, India email: drtanvipatel92@gmail.com

and discarding a number of concepts, Seward in 1965 suggested that between the contributions from the mandibular arches to the tongue anteriorly, is the most likely site for their origin. However, this implies an endodermal derivation which was unlikely for a structure that contains skin adnexa. Ettinger and Manderson in 1973 suggested that implantation keratinizing epidermoid cysts may occur in other parts of the mouth as a result of trauma. In 1965, Seward believed that dermoid and epidermoid cysts of the midline of the floor of the mouth always originate above the mylohyoid muscle. Allard in 1982, differentiated between a sublingual or genioglossal type which is located between the geniohyoid muscle and the oral mucosa, and a geniohyoid (submental) type which is positioned between the geniohyoid and mylohyoid muscle. In the review of King et al. in 1994, 52% of cases were recorded as sublingual, 26% of cases as submental and 6% of cases as submandibular. The remaining 16% of cases occupied more than one space.[8]

Histologically, in young epidermoid cysts several layers of squamous and granular epithelium can be usually recognized whereas, in the older epidermal cysts, the wall appears to be markedly atrophic and may consist of only one or two rows of flattened cells. The cyst contains horny material arranged in laminated layers. Presence of melanin pigmentation, melanocytes and melanophages can also be observed in some cases.[11] A distinct granular layer and lamellated keratin without calcification are a characteristic feature of this cyst.[13] In our cases, the sites of occurrence were the cheeks, sublingual and right parotid (epidermoid cyst). The histological features observed in our cases were in accordance with the above histological features with all cases showing orthokeratinization. The cystic lumen showed the presence of few foci of keratin flakes. Two to three cell layer epithelium with the presence of a prominent granular cell layer was observed in all our cases. Two cases also showed melanin pigmentation. Chronic inflammatory cell infiltration was also observed in two cases of the epidermoid cyst. Thus all the cysts were seen to have individual distinct characteristics. After the follow up of the patient, no recurrence of the lesions has been observed.

The treatment for these cysts is surgical excision. Once excised surgically, the recurrence of these cysts is very less frequent. Intraorally occurring dermoid and epidermoid cysts are usually asymptomatic. They can show acute symptoms when secondarily infected. There have been some case reports showing the malignant transformation of these cysts. Malignant transformation is seen in 2% of the cases of the dermoid cysts[14] and 0.011% to 0.013% cases of the epidermoid cysts.[15] Ikeda et al. in 1990, reported a case of basal cell carcinoma arising from the epidermoid cyst. Lopez-Rios et al. in 1999, reported a case of squamous cell carcinoma arising from the connective tissue wall of the epidermoid cyst. Devine et al. in 2000, reported a case of a dermoid cyst in the sublingual region showing the carcinomatous transformation. Thus the diagnosis of these cysts should be made after ruling out other developmental, infectious and malignant lesions.

CONCLUSION

Thus to conclude it is seen that the intraoral occurrence of the dermoid and an epidermoid cyst is a very rare entity. Both the cysts can undergo malignant transformation. However, the rate of malignant transformation in a dermoid cyst is higher than that if an epidermoid cyst. Thus both these cysts should be distinguished based on their histopathological features to rule out the malignant transformation. This will, therefore, help the surgeon to carry out a definitive treatment protocol.

REFERENCES

1. Shenoy N, Poojary D, Mohan R, Naik R, Baliga M. "Epidermoid cyst of the floor of the mouth," National Journal of Maxillofacial Surgery. 2014;5(1):79-83.
2. Cho.Y, Lee.D. "Clinical Characteristics of Idiopathic Epidermoid and Dermoid Cysts of the Ear," J Audiol Otol, 2017 Jul; 21(2):77-80.
3. Dutta M, Saha J, Biswas G, Chattopadhyay S, Sen I, Sinha R. "Epidermoid Cysts in Head and Neck: Our Experiences, with Review of Literature," Indian J Otolayngol Head Neck Surg, 2013 Jul;65(1):14-21.
4. Janarthanam J, Mahadevan S., " Epidermoid Cyst of Submandibular Region," J Oral Maxillofac Pathol, 2012 Sep-Dec; 16(3): 435-437.
5. Makhija D, Sisodiya N, Shah H, Waghmare M. " Cystic Congenital Scalp Inclusion Dermoid: A Case Report," Dev Period Med, 2016;4:287-288.
6. Jham BC, Duraes GV, Jham AC, Santos CR. "Epidermoid Cyst of the Floor of the Mouth: A Case Report," JCDA, 2007; 73: 525-528.
7. Kini YK, Kharkar VR, Rudagi BM, Kalburge JV. "An Unusual Occurrence of Epidermoid Cyst in the Buccal Mucosa: A Case Report with Review of Literature," J. Maxillofac Oral Surg. 2013;12(1):90-93.
8. Shear M , Speight P. Cysts of the Oral and Maxillofacial Regions, 4th Edition: 181-183
9. Sabhalok SS, Shetty LS, Sarve PH, Setiya SV, Bharadwaj SR. "Epidermoid and Dermoid Cysts of the Head and Neck Region," PlastAesthet Res, 2016;3:347-350.
10. Dillon JR, Avillo AJ, Nelson BL. " Dermoid Cyst of the Floor of the Mouth", Head Neck Pathol, 2015 Sep;9(3):376-378.
11. Lever's Histopathology of the Skin, Tenth Edition, 800-804
12. Erich JB. "Sebaceous, mucous, dermoid and epidermoid cysts," Am J Surg. 1940;50:672-677.
13. Rosai J., Rosai and Ackerman's Surgical Pathology, Ninth Edition. 1:151-152.
14. Sanghera P, El Modir A, Simon J. Malignant transformation within a dermoid cyst: a case report and literature review. Archives of gynecology and obstetrics. 2006 Jun 1;274(3):178.
15. Rathna S. "Epidermal cyst with malignant transformation: A case report," Journal of Diagnostic Pathology and Oncology. 2017;2(1):13-14.

ISSN: 0975-833X

Available online at http://www.journalcra.com

International Journal of Current Research
Vol. 10, Issue, 09, pp.73845-73849, September, 2018

DOI: https://doi.org/10.24941/ijcr.32449.09.2018

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

RESEARCH ARTICLE

EVALUATION OF MICRONUCLEI COUNT IN EXFOLIATED BUCCAL MUCOSAL CELLS AMONGST DIFFERENT AGE GROUPS OF NORMAL HEALTHY INDIVIDUALS: A QUANTITATIVE STUDY

[1,*]Dr. Rajshri Uttam Gurav, [2]Dr. Jigna Pathak, [3]Dr. Shilpa Patel and [4]Dr. Niharika Swain

[1]Resident, MDS, Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Kamothe, Navi Mumbai, India

[2]Professor, MDS, Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Kamothe, Navi Mumbai, India

[3]Professor and Head, MDS, Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Kamothe, Navi Mumbai, India

[4]Reader, MDS, Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Kamothe, Navi Mumbai, India

ARTICLE INFO

Article History:
Received 16th June, 2018
Received in revised form
27th July, 2018
Accepted 10th August, 2018
Published online 30th September, 2018

Key Words:
Exfoliative cytology;
Healthy individuals; Micronuclei;
Total number of micronuclei;
Number of cells with micronuclei;
Papanicolaou stain.

ABSTRACT

Background: Micronuclei (MN) are a small additional nucleus and are readily identifiable by light microscopy. Biologically, MN is the chromosome fragments or whole chromosomes that lag behind at anaphase during nuclear division. MN occurs due to genetic damage of the cell and the MN scoring is the indicator of the genetic damage. But, it has been shown by various studies that MN formation is not always related with genetic damages and may be developed from the physiological damage of double stranded DNA break when a cell enters from G0 to G1 phase of the cell cycle. Therefore simple presence of MN may not indicate any disease as this may be seen even in normal healthy cell. Only high MN count may be suggestive of a genetic damage. Thus MN1 counting in normal healthy individuals can be used to supervise genotoxicity, biomonitoring of diseases, screening of preneoplastic diseases and identification of high risk patients.

Objectives:

- To compare total number of micronuclei and number of cells with micronuclei in exfoliated buccal mucosal cells amongst different age groups of normal healthy individuals.
- To determine a normal range of total number of micronuclei and number of cells with micronuclei in exfoliated buccal mucosal cells amongst different age groups of normal healthy individuals in the studied population.

Methods: This study was conducted on normal healthy individuals (n=500) age ranged from 18-70 years. Based on age normal healthy individuals were categorized into 5 age groups: Group A: 18-30 years; Group B: 31-40 years; Group C: 41-50 years; Group D: 51-60 years and Group E: 61-70 years. Each age group comprised n=100 normal healthy individual. The exfoliated cytosmears prepared from oral buccal mucosa of normal healthy individuals and stained with Papanicolaou (PAP) technique. We calculated the total number of MN (TMN) and number of cells with MN (CMN) per normal healthy individual since some cells had multiple MN.

Results: The mean of TMN found were increased with increase in age and this difference was statistically significant. ($p = 0.007$) The mean of CMN also found were increased with increase in age. However, statistical test did not show any significant difference amongst them. ($p = 0.071$) In the normal healthy individuals, the normal range for TMN and CMN was 1 to 12 and 1 to 10 respectively.

Conclusions: There is an increase in total number of MN (TMN) and number of cells with MN (CMN) with increasing age.

Citation: *Dr. Rajshri Uttam Gurav, Dr. Jigna Pathak, Dr. Shilpa Patel and Dr. Niharika Swain*, **2018.** "Evaluation of micronuclei count in exfoliated buccal mucosal cells amongst different age groups of normal healthy individuals: a quantitative study", *International Journal of current research*, 10, (09), 73845-73849.

Dean
M. G. M. Dental College & Hospital
Kamothe, Navi Mumbai - 410 209.

Ford JH, Schultz CJ, Correll AT. 1988. Chromosome elimination in micronuclei: A common cause of hypoploidy. *Am J Hum Genet.*, 43:733–40.

Hando JC, Nath J, Tucker JD. 1994. Sex chromosomes, micronuclei and aging in women. *Chromosoma.* 103:186–92.

Holland N, Bolognesi C, Kirsch-Volders M, Bonassi S, Zeiger E, Knasmueller S, et al. 2008. The micronucleus assay in human buccal cells as a tool for biomonitoring DNA damage.The HUMN project perspective on current status and knowledge gaps. *Mutat Research.*, 659:93–108.

Jadhav K, Gupta N, Ahmed MB. 2011. Micronuclei: An essential biomarker in oral exfoliated cells for grading of oral squamous cell carcinoma. *J Cytol.*, 28(1):7-12.

Karahalil B, Karakaya AE, Burgaz S. 1999. The micronucleus assay in exfoliated buccal cells: application to occupational exposure to polycyclic aromatic hydrocarbons. *Mutat Res.* 442(1):29-35.

Konopacka M. 2003. Effect of smoking and aging on micronucleus frequencies in human exfoliated buccal cells. Neoplasma. 50(5):380-2.

Lucero L, Pastor S, Suárez S, Durbán R, Gómez C, Parrón T, Creus A, Marcos R. 2000. Cytogenetic biomonitoring of Spanish greenhouse workers exposed to pesticides: micronuclei analysis in peripheral blood lymphocytes and buccal epithelial cells. *Mutat Res.* 464(2):255-62.

Orta T, Gunebakan S. 2012. The effect of aging on micronuclei frequency and proliferation in human peripheral blood lymphocytes. *Indian J Hum Genet.*, 18(1):95-100.

Palaskar, S., Jindal, C. 2011. Evaluation of micronuclei using Papanicolaou and may Grunwald–Giemsa stain in individuals with different tobacco habits: a comparative study. *J Clin Diagn Res.*, 4:3607–13

Pickering MT, Kowalik TF. 2006. Rb inactivation leads to E2F1-mediated DNA double-strand break accumulation. Oncogene, 25:746–55.

Samanta , S., Dey P. 2012. Micronucleus and its applications. Diagn Cytopathol. 40(1):84-90.

Thierens H, Vral A, Morthier R, Aousalah B, De Ridder L. 2000. Cytogenetic monitoring of hospital workers occupationally exposed to ionizing radiation using the micronucleus centromere assay. Mutagenesis. 15:245–49.

****

ORIGINAL RESEARCH PAPER

Volume-7 | Issue-4 | April-2018 | ISSN No 2277 - 8179 | IF : 4.758 | IC Value : 93.98

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

IMMUNOEXPRESSION AND CORRELATION OF CYTOTOXIC T LYMPHOCYTES IN ORAL SQUAMOUS CELL CARCINOMA WITH LYMPH NODE STATUS: A RETROSPECTIVE STUDY

Oncology

Dr. Kehkashan Azmi* Resident, MDS, Department of Oral Pathology and Microbiology, MGM Dental College & Hospital, Kamothe, Navi Mumbai *Corresponding Author

Dr. Shilpa Patel Professor & Head, MDS, Department of Oral Pathology and Microbiology, MGM Dental College & Hospital, Kamothe, Navi Mumbai.

Dr. Jigna Pathak Professor, MDS, Department of Oral Pathology and Microbiology, MGM Dental College & Hospital, Kamothe, Navi Mumbai

Dr. Niharika Swain Reader, MDS, Department of Oral Pathology and Microbiology, MGM Dental College & Hospital, Kamothe, Navi Mumbai

Dr. Rajshri Gurav Resident, MDS, Department of Oral Pathology and Microbiology, MGM Dental College & Hospital, Kamothe, Navi Mumbai

ABSTRACT

Background & Objectives: In OSCC, the presence of regional lymph node metastasis at presentation is the most significant adverse prognostic factor and a major determinant of poor survival. Tumor-infiltrating lymphocytes (TILs) often infiltrate solid malignant tumours and extensive lymphocyte infiltration has been related with a more favourable prognosis in patients with various cancers. OSCC often contain large mononuclear cell infiltrates, comprised mainly of T cells, which could reflect an in situ immune reaction against the malignant OSCC cells. The aim of this retrospective study was to evaluate the expression of Cytotoxic T lymphocyte in OSCC using immunohistochemical marker CD8+(CTLs) and correlate these findings with the status of lymphnode.

Methods: The study was conducted on tissue sections obtained from histopathologically diagnosed cases of OSCC (n=30) retrieved from the archives of Department of Oral and Maxillofacial Pathology. The sample consisted of cases showing lymph node metastasis (n=15) and those without pathologic lymph node involvement (n=15). The sections were evaluated by using immunohistochemical staining technique with marker CD8 for Cytotoxic T lymphocytes. The mean immunoexpression of Cytotoxic T lymphocyte was evaluated and correlated with lymphnode status.

Results: A statistically significant increase in the count of CTLs (CD8+) was observed in lymph node negative pN(-) as compared to lymph node positive cases pN(+) of OSCC.

Interpretation & Conclusion: CTLs (CD8+) are involved in modulating the immune response and can contribute to the dissemination or control of metastatic neoplastic cells. It can be considered that T-cell mediated adaptive immunity plays a key role in anti-tumour immunity.

KEYWORDS

Cytotoxic T lymphocyte, CD8, Immune response, Lymph node metastasis, OSCC.

INTRODUCTION:

The term 'Oral cancer' is used to describe any malignancy that arises from the oral cavity comprising of lip, tongue, buccal mucosa and oropharynx[1]There are an estimated half-a-million of cases of cancer of the oral cavity and pharynx occurring annually, and a quarter-of-a-million deaths[2]. India accounts for more than one-fourth of world's burden.[3] The high mortality rate may be due to the fact that oral carcinoma cells easily invade into territorial tissues and metastasize to the cervical lymph nodes. Hence it is necessary to know the mechanisms and factors involved for cancer progression, for it to be a metastatic disease. The process of metastasis in regional lymph nodes begins with invasive growth of tumor cells and its detachment from the primary lesion. This is followed by lymphogenous transport of cancer cells that results in lodgement and proliferation of cancer cells in and around lymph nodes.[4]

Cancer can be considered with regard to a step-wise development functionally grouped into three phases: initiation, promotion, and progression. Initiation is characterized by genomic changes within the "cancer cell," such as point mutations, gene deletion and amplification and chromosomal rearrangements leading to irreversible cellular changes. The concept that tumor development is the result of processes involving both the cancer cells themselves and non-cancer cells[5] consisting of tumour microenvironment which contains innate immune cells (including macrophages, neutrophils, mast cells, myeloid-derived suppressor cells, dendritic cells, and natural killer cells) an adaptive immune cells (T and B lymphocytes) and their surrounding stroma which consists of fibroblasts, endothelial cells, pericytes, and mesenchymal cells. These diverse cells communicate with each other by means of direct contact or cytokine and chemokine production and act in autocrine and paracrine manners to control and shape tumor growth. It is the expression of various immune mediators and modulators as well as the activation state of different cell types in the tumor microenvironment that dictate in which direction the balance is tipped and whether tumor-promoting inflammation or antitumor immunity will ensue. In established tumours, this balance is profoundly tilted toward protumor inflammation. However, it is safe to assume that tumor promoting inflammation and antitumor immunity coexist at different points along the path of tumor progression.[6]

Inflammatory elements like Activated memory- and cytotoxic tumor infiltrating T-lymphocytes (TILs) are considered to be manifestations of a specific host immune reaction against cancer cells, related to the cytotoxic activity and the production of growth modulating cytokines of TIL.[7]Tumor-infiltrating lymphocytes (TIL) were found to correlate with improved prognosis in several types of cancer. The presence of TIL is considered a reflection of the immune response to the tumor.[8] Hence TIL plays an important role in anti tumour immune response. Each T cell is genetically programmed to recognize a specific cell bound antigen by means of an antigen specific T cell receptor (TCR).[9] This complex helps in activation of T lymphocytes and release cytokines thereby mediating immune response.

T-lymphocytes can be T- helper cells and Cytotoxic T- cells. T- helper cells serves as a major regulator of all immune functions by forming lymphokines which act on other cells of immune system hence having no direct role.[10]Cytotoxic T lymphocytes(CTLs) formed from naïve T cells release cytotoxic granules, rich in perforin and granzyme B (GB) which cause apoptosis of neoplastic cells in lymph nodes and at tumour site.[11,12] Thereby CTLs (CD8+) are involved in modulating the immune response and can contribute to the dissemination or control of metastatic neoplastic cells.[13,14] A lower density of activated CTLs were seen in metastatic when compared to non metastatic lymph nodes in cases of OSCC.[11]

Thus, the aim of the present retrospective study is to evaluate the expression of Cytotoxic T lymphocyte in OSCC using immunohistochemical marker CD8+(CTLs) and correlate these findings with the status of lymphnode.

Dean
M. G. M. Dental College & Hospital
Kamothe, Navi Mumbai - 410 209.

ISSN No 2277 - 8179 | IF : 4.758 | IC Value : 93.98

ii) Independent t-test results:

	t-test value	df	p-value	Mean Difference
CD8+	-2.396	28	.023	-19.64000

*The p value is significant at the 0.05 level.

Table II illustrates the **descriptive statistics** correlating mean immunoexpression of Cytotoxic T lymphocytes (Cd8+) with lymphnode status(pN) in OSCC cases. The statistical test used was **independent student's't' test**. There was a statistically significant difference in the mean immunoexpression of CTLs(CD8+) (p value=0.023) between pN+ and pN- cases of OSCC.

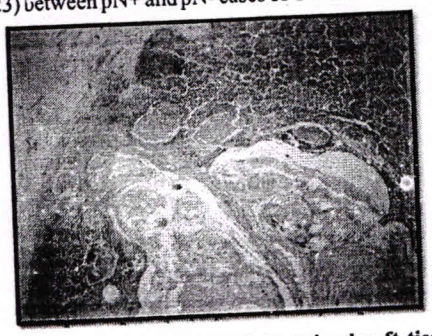


Figure 1: Photomicrograph of H & E stained soft tissue section showing pN(+) lymphnode. [Hematoxylin and Eosin, 40x]

Figure 2: Photomicrograph of H & E stained soft tissue section showing pN(-) lymphnode. [Hematoxylin and Eosin, 40x]

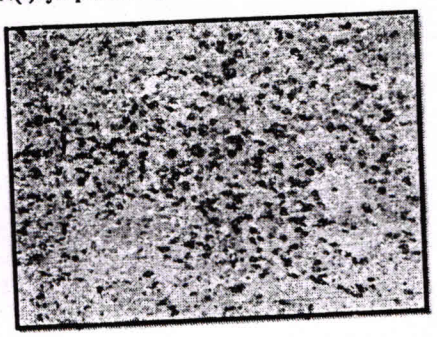
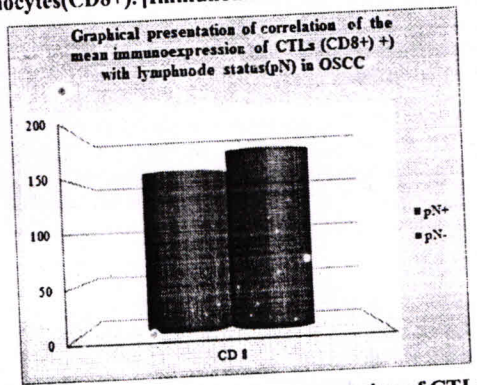


Figure 3: Photomicrograph of immunohistochemically stained soft tissue section of OSCC in primary tumour showing Cytotoxic T-lymphocytes(CD8+). [Immunohistochemical Stain, 400x]



Graph 1: correlates the mean immunoexpression of CTLs (Cd8+) with lymphnode status(pN) in OSCC cases. An increase in the count of CTLs(CD8+) was observed in lymph node negative pN(-) as compared to lymph node positive cases pN(+) of OSCC.

REFERENCE

1. Pawar HJ, Dhumale GB, Singh KK. Epidemiological determinants of oral cancer in a rural area of Maharashtra state, India. International J. of Healthcare and Biomedical Research. 2014; 2(4):186-94.
2. Gandini S, Negri E., Boffetta P, Vecchia C, Boyle P. Mouthwash and Oral Cancer Risk –Quantitative Meta-analysis of Epidemiologic Studies. Annals of Agricultural and Environmental Medicine 2012;19(2):173-80.
3. Dr. Cruz A, Dr. Chaukar D. Evidence based management of cancers in India. Vol XI. Part A. Tata memorial centre, Mumbai 2012.p-24.
4. Yamamoto E, Miyakawa A, Kohama G. Mode of invasion and lymph node metastasis in squamous cell carcinoma of the oral cavity. Head and neck surgery 1984;6(5):938-47.
5. Seth Rakoff- Nahoum. Why Cancer and Inflammation? Yale J Biol Med 2006;79(3-4):123-30.
6. Grivennikov S, Greten F, Karin M. Immunity, Inflammation, and Cancer. Cell 2010;140(6):883–899.
7. Rauser S, Langer R, Tschernitz S, Gias P, Jutting U, Feith M, et al. High number of CD45RO+ tumor infiltratinglymphocytes is an independent prognostic factor in non metastasized(stage I-IIA) esophageal adenocarcinoma .BMC Cancer 2010;10(608):1-9.
8. Leffers N, Gooden M, Jong R, Hoogeboom B, Hoor K, Hollema H, et al. Prognostic significance of tumor-infiltrating T-lymphocytes in primary and metastatic lesions of advanced stage ovarian cancer; Cancer Immunol Immunother 2009;58(3):449–59.
9. Cotran,Kumar,Collins. Robbins Pathologic Basis of Disease. 6th Edition. Elseveir 2003.p-189.
10. Guyton , Hall. Textbook of Medical Physiology. 10thEdition.W.B. Saunders 2000.p-409
11. Gonsalves A, Costa N, Deigo A, Alencar R, Silva T, Batista A.Immune response in cervical lymph nodes from patients with primary oral squamous cell carcinoma J of Oral Pathol Med 2013;42(7):535–40.
12. Trapani J, Smyth MJ. Functional Significance of the perforin/granzyme cell death pathway. Nat Rev Immunol 2002;2(10):735-47.
13. Almand B, Resser JR, Lindman B, Nadaf S, Clark JI, Kwon ED, et al. Clinical significance of defective dendritic cell differentiation in cancer. Clin Cancer Res 2000;6(5):1755–66.
14. Cella M, Sallusto F, Lanzavecchia A. Origin, maturation and antigen presenting function of dendritic cells. Curr Opin Immunol 1997;9(1):10–16.
15. Bancroft JD, Stevens A. Hematoxylin and Eosin. In: Suvarna K, Layton C, Bancroft JD, editors. Bancroft's Theory and Practice of Histological Techniques.6th ed. London: Churchill Livingstone; 2008.p.121-6.
16. Enomoto K, Sho M, Wataksuki K, Takayama T, Matsumoto S, Nakamura S, et al. Prognostic importance of tumour-infiltrating memory T cells in oesophageal squamous cell carcinoma. J Trans Immunol. 2012;168(2):186–91.
17. Snyderman CH, Heo DS, Chen K, Whiteside TL, Johnson JT .T-cell markers in tumor-infiltrating lymphocytes of head and neck cancer. Head Neck. 1989 ;11(4):331-6.
18. dos antos Pereira J, da Costa Miguel MC, Guedes Queiroz LM, da Silveira ÉJ Analysis of CD8+ and CD4+ cells in oral squamous cell carcinoma and their association with lymph node metastasis and histologic grade of malignancy. Appl Immunohistochem Mol Morphol. 2014; 22(3):200-5.
19. Balermpas P, Rödel F, Weiss C, Rödel C, et al.Tumor infiltrating lymphocytes favor the response to chemoradiotherapy of head and neck cancer, OncoImmunology 2014;3:(1):e27403
20. Koelzer V, Lungli A, Dawson H, Hadrich M, Berger M, Borner M. et al. CD8 / CD45RO Tcell infiltration in endoscopic biopsies of colorectal cancer predicts nodal metastasis and survival.J Transl Med. 2014; 12 :81
21. Jung IK, Kim S, Suh D, Kim KH,Lee CH, Yoon MS. Tumor-infiltration of T-lymphocytes is inversely correlated with clinicopathologic factors in endometrial adenocarcinoma. Obstet Gynecol Sci 2014; 57(4):266-273.
22. Ropponen KM, Eskelinen MJ, Lipponen PK, Alhava E, Kosma VM Prognostic value of tumour-infiltrating lymphocytes (TILs) in colorectal cancer. J Pathol.1997;182: 318–324
23. Naito Y, Saito K, Shiiba K, Ohuchi A, Saigenji K, Nagura H, and Ontani1H. CD8+ T Cells Infiltrated within Cancer Cell Nests as a Prognostic Factor in Human Colorectal Cancer. Cancer Research 1998;58. 3491-3494.
24. Chiba T, Ohtani H, Mizoi T, Naito Y, Sato E, Nagura H, Ohuchi A, Ohuchi K, Shiiba K, Kurokawa Y, Satomi S. Intraepithelial CD8+ T-cellcount becomes a prognostic factor after a longer follow-up period in human colorectal carcinoma: possible association with suppression of micrometastasis. Br J Cancer2004;91:1711–1717
25. Koch M, Beckhove P, Op den Winkel J, Autenrieth D, Wagner P, Nummer D, Specht S, Antolovic D, Galindo L, Schmitz-Winnenthal FH, Schirrmacher V, Buchler MW, Weitz J.Tumor infiltrating T lymphocytes in colorectal cancer: Tumor-selective activation and cytotoxic activity in situ. Ann Surg.2006;244: 986–992:992–3
26. Baker K, Zlobec I, Tornillo L, Terracciano L, Jass JR, Lugli A Differential significance of tumour infiltrating lymphocytes in sporadic mismatch repair deficient vs proficient colorectal cancers: a potential role for dysregulation of the transforming growth factor-beta pathway. Eur J Cancer.2007;43: 624–631
27. Zancope E, Costa NL, Junqueira-Kipnis AP, Valadares MC, Silva TA, Leles CR, et al . Differential infiltration of CD8+ and NK cells in lip and oral cavity squamous cell carcinoma. J Oral Pathol Med. 2010; 39(2):162-7.
28. Dunn GP, Old LJ, Schreiber RD The three Es of cancer immunoediting. Annu Rev Immunol.2004 ;22: 329–360
29. Ekert, P. G., and Vaux, D. L. Apoptosis and immune system. Br. Med. Bull., 1997; 53: 591–603
30. Henkart, P. A. Lymphocyte-mediated cytotoxicity: two pathways and multiple effector molecules. Immunity. 1994; 1: 343–346,
31. Berke, G. The CTL's kiss of death. Cell. 1995; 81: 9–12
32. Eerola AK, Soini Y, Pääkkö P. A high number of tumor-infiltrating lymphocytes are associated with a small tumor size, low tumor stage, and a favorable prognosis in operated small cell lung carcinoma. Clin Cancer Res. 2000;6(5):1875-81.
33. Cardi, G., Heaney, J. A., Schned, A. R., and Ernstoff, M. S. Expression of Fas(APO-1/CD95) in tumor-infiltrating and peripheral blood lymphocytes in patients with renal cell carcinoma. Cancer Res. 1998; 58: 2078–2080.
34. Strand, S., Hoffman, W. J., Hug, H., Mu¨ller, M., Otto, G., Strand, D., Mariani, S. M., Stremmel, W., Krammer, P. H., and Galle, P. R. Lymphocyte apoptosis induced by CD95 (APO-1/Fas) ligand-expressing tumor cells—a mechanism of immune evasion? Nat. Med. 1996.; 2: 1361–1366.
35. Bennett, M. W., O'Connell, J., O'Sullivan, G. C., Brady, C., Roche, D., Collins, J. K., and Shanahan, F. The fas counterattack in vivo: apoptotic depletion of tumor-infiltrating lymphocytes associated with fas ligand expression by human esophageal carcinoma. J. Immunol., 1998;160: 5669–5675.
36. Lee HE, Chae SW, Lee YJ, Kim MA, Lee HS, Lee BL, et al. Prognostic implications of type and density of tumourinfiltrating lymphocytes in gastric cancer. Br J Cancer. 2008 ;99: 1704–11.

ORIGINAL RESEARCH PAPER

Volume-7 | Issue-4 | April-2018 | ISSN No 2277 - 8179 | IF : 4.758 | IC Value : 93.98

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

EVALUATION AND CORRELATION OF DENSITY OF TUMOUR ASSOCIATED MACROPHAGES IN ORAL SQUAMOUS CELL CARCINOMA WITH OR WITHOUT LYMPH NODE INVOLVEMENT: A RETROSPECTIVE IMMUNOHISTOCHEMICAL ANALYSIS

Oncology

Dr. Rajshri Gurav* Resident, MDS, Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Kamothe, Navi Mumbai *Corresponding Author

Dr. Jigna Pathak Professor, MDS, Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Kamothe, Navi Mumbai

Dr. Shilpa Patel Professor and HOD, MDS, Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Kamothe, Navi Mumbai

Dr. Niharika Swain Reader, MDS, Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Kamothe, Navi Mumbai

Dr. Kehkashan Azmi Resident, MDS, Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Kamothe, Navi Mumbai

ABSTRACT

Objective: To immunohistochemically evaluate and correlate the density of tumour associated macrophages (TAMs) in Oral Squamous Cell Carcinoma (OSCC) cases with or without lymph node involvement.

Material and Methods: This retrospective study was conducted on formalin fixed paraffin embedded tissue blocks of OSCC cases (n=30) treated with neck dissection, which were retrieved from the archives of the Department of Oral Pathology and Microbiology. OSCC cases (n=30) were classified based on pathological lymph node status as with lymph node metastasis (pN+) (n=15) and without lymph node metastasis (pN-) (n=15). Immunohistochemical analysis was carried out using immunohistochemical marker CD68 for TAM. The density of CD68+ TAMs in primary tumour (OSCC) was evaluated and correlated with pathological lymph node status.

Results: The mean density of TAMs (CD68+) was increased in pN+ OSCC cases when compared to pN- OSCC cases. Statistically this difference was significant.

Conclusion: The result obtained suggested that the mean density of TAMs (CD68+) may have a predictive value in determining the metastatic potential of OSCC.

KEYWORDS

CD68, Macrophages, Oral squamous cell carcinoma, Pathological lymph node status, Tumour Associated Macrophages, Tumor microenvironment.

Introduction:

Oral squamous cell carcinoma (OSCC) comprises more than 95% of all Oral cancers.[1] It is estimated to be the sixth most common cancer and in India the prevalence is around 45%.[2,3] Despite the significant advances in therapeutic strategies, the five year survival rate is only 53%.[4] In addition, a high percentage of patients have a poor response to therapy and high recurrence rates.[5] Hence, there is a need to identify novel biological markers that predict patients at high risk of disease.

Carcinogenesis or Cancer development is a multistep process which can be summed up into 3 stages - initiation, promotion and progression.[6] Progression includes metastasis of oral cancer, which is a complex process involving detachment of cells from tumour tissue, regulation of cell motility, invasion, proliferation and evasion through the blood vessels or lymphatic system.[7] Locoregional lymph node metastasis is considered as one of the significant independent prognostic factors in OSCC.[8] One of the key factors in lymph node metastasis is tumour microenvironment.[9] Tumor microenvironment contains diverse cells including tumor cells and various population of stromal cells (non- neoplastic cells) such as fibroblasts, epithelial cells, endothelial cells and infiltrating immune cells (innate and adaptive), as well as the products of these cells such as growth factors, extracellular matrix, chemokines, cytokines, enzyme and various metabolites.[10] In tumor microenvironment, inflammatory immune cells such as macrophages are referred to as Tumour Associated Macrophages (TAMs)/ Tumor Infiltrating Macrophages/ Panmacrophages.[11,12]

Many studies have shown an association between mean density of TAMs and prognosis in a variety of human cancers. In patients with breast, oral, thyroid, gastric, uterine and bladder cancer, a high density of infiltrating TAMs are associated with poor clinical outcomes while, in patients with prostate, colorectal and brain cancer, a high density of infiltrating TAMs have been associated with increased survival and improved prognosis.[12,13,14,15] In oral cancer, however, TAMs infiltration correlated with lymphangiogenesis, increased lymph node metastasis and advanced stages of tumour invasion and consequently enhanced tumour aggressiveness.[16,17]

Very few studies are done in OSCC to evaluate and correlate the mean density of TAMs with pathological lymph node status. Thus, the purpose of present retrospective study was to immunohistochemically evaluate and correlate the mean density of TAMs in tumor microenvironment of OSCC cases with pathological lymph node status using the immunohistochemical marker Cd68.

Material and Methods:

This retrospective study was conducted on formalin fixed paraffin embedded tissue blocks of OSCC cases (n=30) treated with neck dissection which were retrieved from the archives of the Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Navi Mumbai. The study was carried out from December 2014 to August 2016. Based on pathological lymph node status OSCC cases were categorized as those with lymph node metastasis (pN+) (n=15) and without lymph node metastasis (pN-) (n=15). (Figure 1 and 2) Recurrent cases of OSCC were excluded from the study.

Sections from tumor proper of each case were subjected to immunohistochemical staining technique to detect TAMs using prediluted primary antibody against CD68 (Dako, USA).[18] The presence, distribution and density of TAMs (CD68+) in OSCC microenvironment were evaluated by using conventional light microscopy. The mean density of TAMs (CD68+) for each case in tumor proper, was calculated as the mean density of infiltrating macrophages in five chosen HPFs.[19] (Figure 3) All collected data was entered into SPSS 16.0 (statistical package for social sciences version 16.0) worksheet. Further analysis was performed using statistical test such as Independent student's't' test. A significance level of 0.05 was applied to decide the statistical significance of the hypothesis being tested.

TAM (CD68+) with pathological lymph node status. In our study on OSCC, we found that the mean density of TAMs (CD68+) was higher in lymph node metastasis cases as compared to without lymph node metastasis indicating that increase in mean density of TAMs (CD68+) has a key role in tumor progression and lymph node metastasis. However, more research work on a much larger sample size would further authenticate our observation.

Conclusion:

It is apparent from the results of present study that TAMs (CD68+) could be considered pro-tumour as they have a key role in tumor growth, tumor progression and metastasis. Further studies with larger sample size are required to ascertain the interaction between TAMs and cancer cells. This will emphasize the role of TAMs as an effective biomarker in predicting lymph node metastasis which is an independent prognostic marker and certainly shed new light on the development of efficient targeted anticancer therapy.

Financial support and sponsorship:
Nil.

Conflicts of interest:
There are no conflicts of interest.

References:

1. Bagan J, Sarrion G, Jimenez Y. Oral Cancer: Clinical Features. Oral Oncology. 2010; 46(6):414-7.
2. Shah JP, Gil Z. Current Concepts in Management of Oral Cancer-Surgery. Oral Oncology. 2009;45(4-5):394-401.
3. Siddiqui IA, Farooq MU, Siddiqui RA, Rafi SMT. Role of toluidine blue in early detection of oral cancer. Pak J Med Sci. 2006;22:184–7.
4. Parkin D, Bray F, Ferlay J, Pisani P. Global cancer statistics, 2002. CA Cancer J Clin. 2005;55:74–108.
5. Bettendorf O, Piffko J, Bankfalvi A. Prognostic and predictive factors in oral squamous cell cancer: important tools for planning individual therapy? Oral Oncol. 2004;40(2):110–9.
6. Barrett JC. Mechanisms of multistep carcinogenesis and carcinogen risk assessment. Environ Health Perspect.1995;100:9-20.
7. Noguti J, De Moura CF, De Jesus GP, Da Silva VH, Hossaka TA, Oshima CT, et al. Metastasis from Oral Cancer: An Overview. Cancer Genomics Proteomics. 2012; 9(5): 329-35.
8. Ran S, Montgomery KE. Macrophages- mediated lymphangiogenesis: The emerging role of macrophages as lymphatic endothelial progenitors. Cancers. 2012; 4(3): 618-57.
9. Chandrasekaran S, King MR. Microenvironment of Tumor-Draining Lymph Nodes: Opportunities for Liposome-Based Targeted Therapy. Int J Mol Sci. 2014;15(11):20209-39.
0. Zhang Y, Cheng S, Zhang M, Zhen L, Pang D, Zhang Q, et al. High-infiltration of tumor-associated macrophages predicts unfavorable clinical outcome for node-negative breast cancer. PLoS One. 2013;30;8(9):e76147.
1. Mori K, Hiroi M, Shimada J, Ohmori Y. Infiltration of M2 Tumor-Associated Macrophages in Oral Squamous Cell Carcinoma correlates with Tumor Malignancy. Cancers. 2011; 3(4): 3726-39.
2. Lin JY, Li XY, Tadashi N, Dong P. Clinical significance of tumor-associated macrophage infiltration in supraglottic laryngeal carcinoma. Chin J cancer. 2011; 30(4):280-6.
3. Zhang QW, Liu L, Gong CY, Shi HS, Zeng YH, Wang XZ, et al. Prognostic significance of tumor-associated macrophages in solid tumor: a meta-analysis of the literature. PLoS One. 2012;7(12):e50946.
4. Pantano F, Berti P, Guida FM, Perrone G, Vincenzi B, Amato MM, et al. The role of macrophages polarization in predicting prognosis of radically resected gastric cancer patients. J Cell Mol Med.2013; 17(11):1415-21.
5. Costa NL, Valadares MC, Souza PP, Mendonça EF, Oliveira JC, Silva TA, et al. Tumor-associated macrophages and the profile of inflammatory cytokines in oral squamous cell carcinoma. Oral Oncol.2013; 49(3): 216-23.
6. Marcus B, Arenberg D, Lee J, Kleer C, Chepeha DB, Schmalbach CE, et al. Prognostic factors in oral cavity and oropharyngeal squamous cell carcinoma. Cancer. 2004;101(12):2779–87.
7. Liu SY, Chang LC, Pan LF, Hung YJ, Lee CH, Shieh YS. Clinicopathologic significance of tumor cell-lined vessel and microenvironment in oral squamous cell carcinoma. Oral Oncol. 2008;44(3):277–85.
8. Bancroft JD, Stevens A. The Hematoxylins and Eosin. In: Suvarna K, Layton C, Bancroft JD, editors. Bancroft's Theory and Practice of Histological Techniques. 6th ed. London: Churchill Livingstone; 2008. p.121-6.
9. Lu CF, Huang CS, Tjiu JW, Chiang CP. Infiltrating macrophage count: a significant predictor for the progression and prognosis of Oral squamous cell carcinomas in Taiwan. Head Neck. 2010; 32(1): 18-25.
20. He KF, Zhang L, Huang CF, Ma SR, Wang YF, Wang WM, et al. CD163+ tumor-associated macrophages correlated with poor prognosis and cancer stem cells in oral squamous cell carcinoma. Biomed Res Int. 2014;2014:838632.
2. Hu Y, He MY, Zhu LF, Yang CC, Zhou ML, Wang Q, et al. Tumor-associated macrophages correlate with the clinicopathological features and poor outcomes via inducing epithelial to mesenchymal transition in oral squamous cell carcinoma. J Exp Clin Cancer Res. 2016;35:12.
22. Li C, Shintani S, Terakado N, Nakashiroki K, Hamakawa H. Infiltration of tumor associated macrophages in human oral squamous cell carcinoma. Oncol Rep 2002;9(6):1219–23.
23. Fujii N, Shomori K, Shiomi T, Nakabayashi M, Takeda C, Ryoke K, et al. Cancer-associated fibroblasts and CD163 positive macrophages in oral squamous cell carcinoma: their clinicopathological and prognostic significance. J Oral Pathol Med. 2012 Jul; 41(6):444-51.
24. Sun S, Pan X, Zhao L, Zhou J, Wang H, Sun Y. The Expression and Relationship of CD68-Tumor-Associated Macrophages and Microvascular Density With the Prognosis of Patients With Laryngeal Squamous Cell Carcinoma. Clin Exp Otorhinolaryngol. 2016 Sep;9(3):270-7.
25. Ding M, Fu X, Tan H, Wang R, Chen Z, Ding S. The effect of vascular endothelial growth factor C expression in tumor-associated macrophages on lymphangiogenesis and lymphatic metastasis in breast cancer. Mol Med Rep.2012;6(5):1023-9.
26. Chen SJ, Zhang QB, Zeng LJ, Lian GD, Li JJ, Qian CC. Distribution and clinical significance of tumour-associated macrophages in pancreatic ductal adenocarcinoma: a retrospective analysis in China. Curr Oncol. 2015;22(1):e11-9.
27. Zhang B, Yao G, Zhang Y, Gao J, Yang B, Rao Z, et al. M2-Polarized tumor associated macrophages are associated with poor prognoses resulting from accelerated lymphangiogenesis in lung adenocarcinoma. Clinics. 2011;66(11):1879-86.
28. Qing W, Fang WY, Ye L, Shen LY, Zhang XF, Fei XC, et al. Density of Tumor-Associated Macrophages Correlates with Lymph Node Metastasis in Papillary Thyroid Carcinoma. Thyroid. 2012 Sep;22(9):905-10.
29. Schoppmann SF1, Birner P, Stockl J, Kalt R, Ullrich R, Caucig C, et al. Tumor-Associated Macrophages Express Lymphatic Endothelial Growth Factors and Are Related to Peritumoral Lymphangiogenesis. Am J Pathol. 2002 Sep;161(3):947-56.
30. Kerjaschki D. The crucial role of macrophages in lymphangiogenesis. Journal of Clinical Investigation. 2005;115(9):2316–19.
3. Guruvayoorappan C. Tumor versus tumor-associated macrophages: how hot is the link?. Integrative Cancer Therapies. 2008;7(2):90–5.
32. Lynch CC, Hikosaka A, Acuff HB, Martin MD, Kawai N, Singh RK, et al. MMP-7 promotes prostate cancer-induced osteolysis via the solubilization of RANKL. Cancer Cell. 2005;7(5):485-96.
33. Luo JL, Tan W, Ricono JM, Korchynskyi O, Zhang M, Gonias SL, et al. Nuclear cytokine-activated IKKα controls prostate cancer metastasis by repressing Maspin. Nature. 2007;446(7136):690-4.
34. Zamarron BF, Chen W. Dual roles of immune cells and their factors in cancer development and progression. Int J Biol Sci.2011;7(5):651–8.

10.5005/jp-journals-10031-1217

Immunohistochemical Expression of MCM-2 for Evaluation of Proliferative Activity in the Epithelial Lining of Radicular Cyst, Dentigerous Cyst, and Odontogenic Keratocyst: A Retrospective Study

[1]Smita V Rathod, [2]Shilpa Patel, [3]Jigna Pathak, [4]Niharika Swain

ABSTRACT

Aim: To evaluate and compare immunohistochemical expression of minichromosome maintenance protein-2 (MCM-2) for proliferative activity in the epithelial lining of radicular cyst (RC), dentigerous cyst (DC) and odontogenic keratocyst (OKC)

Materials and methods: This retrospective study was conducted on 45 formalin-fixed paraffin-embedded tissue blocks of odontogenic cysts, which were retrieved from the archives. Sections from RC, DC, and OKC were subjected to MCM-2 nuclear staining technique. For each of the specimens, intensity and extent of MCM-2 expressions were evaluated by a comprehensive scoring formula; 100 nuclei were assessed in the epithelial lining of each specimen, under 400× magnification. Scoring was done for positively stained nuclei and expressed as percentage.

Results: The mean (%) MCM-2 immunopositive nuclei in the epithelial lining were higher in OKC cases (10.93%) as compared with RC (10.40%) and DC (3.07%), and the difference was statistically significant between the OKC and DC ($p = 0.046$). The mean (%) MCM-2 expression in different histopathological grades of inflammation in RC showed a statistically significant difference of MCM-2 expression (%) between cases of mild ($n = 4$) and severe ($n = 2$) degrees of inflammation ($p = 0.012$) and also between cases of moderate ($n = 9$) and severe (2) degrees of inflammation ($p = 0.008$). The mean (%) MCM-2 expression in inflamed ($n = 8$) and noninflamed ($n = 4$) cases of OKC showed a statistically significant difference. The mean (%) MCM-2 expression in cases of DC with ($n = 7$) and without ($n = 7$) inflammation also showed a statistically significant difference ($p = 0.033$).

Conclusion: The mean (%) MCM-2 expression was higher in OKC cases as compared with RC and DC, which shows that the epithelium of OKC has a higher proliferative capacity than RC and DC. In present samples, the MCM-2 expression in epithelial lining increased, with increasing grades of inflammation, thus supporting the carcinogenic role of inflammation.

[1]Postgraduate Student, [2]Professor and Head, [3]Professor [4]Lecturer

[1-4]Department of Oral Pathology and Microbiology, Mahatma Gandhi Mission's Dental College and Hospital, Navi Mumbai Maharashtra, India

Corresponding Author: Smita V Rathod, Postgraduate Student Department of Oral Pathology and Microbiology, Mahatma Gandhi Mission's Dental College and Hospital, Navi Mumbai, Maharashtra India, Phone: +917710078828, e-mail: smitarathod2012@gmail.com

Keywords: Dentigerous cyst, Minichromosome maintenance protein, Odontogenic keratocyst, Radicular cyst.

How to cite this article: Rathod SV, Patel S, Pathak J, Swain N. Immunohistochemical Expression of MCM-2 for Evaluation of Proliferative Activity in the Epithelial Lining of Radicular Cyst, Dentigerous Cyst, and Odontogenic Keratocyst: A Retrospective Study. J Contemp Dent 2018;8(1):20-26.

Source of support: Nil

Conflict of interest: None

INTRODUCTION

Odontogenic cysts develop from the residues of odontogenic epithelium, such as reduced enamel epithelium, cell rests of Serres or cell rests of Malassez, or epithelial cells of dental follicle (DF).[1-3] The coronal part of follicle of fully developed tooth is entitled as pericoronal sac or pericoronal follicle and that is occasionally present around impacted tooth.[4] It is an ectomesenchymal tissue and frequently comprises the epithelial remnants of odontogenesis, which might be the initiating point of pathology.[1,5] Occasionally, DF adjacent to impacted teeth will persist and lead to the development of cysts and tumors, such as RC, DC, OKC [as of 2017, the World Health Organization (WHO) has reclassified keratocystic odontogenic tumor (KCOT) as OKC].[6]

The RCs, DCs, and OKCs show different growth patterns and biological behaviors. It is well known that the lining epithelium of both inflammatory and developmental cysts is primarily comprised of squamous epithelium.[1] The differences in cell proliferation and the proliferation rate of this odontogenic epithelium play a significant role in numerous biological and pathological events in it.[1,7] Also, different literatures have shown that the chronic inflammation has a direct influence on these epithelial cells. The growth factors and cytokines of inflammatory infiltrates are also responsible for the higher proliferation ability of the residual epithelium.[8] Inflammation may also increase the squamous changes in good, healthy DFs. The incidence of malignancy developing in odontogenic cyst is believed to be caused by long chronic inflammation and continuous intracystic pressure.[9]

15. Kearsey SE, Labib K. MCM proteins: evolution, properties, and role in DNA replication. Biochim Biophys Acta 1998 Jun;1398(2):113-136.
16. Karimi, S.; Sadr, M. Mini-chromosome maintenance protein family: novel proliferative markers—the pathophysiologic role and clinical application. London: IntechOpen Ltd.; 2011. [cited 2011 Sep 26]. Available from: www.intechopen.com.
17. Liu Y, Richards TA, Aves SJ. Ancient diversification of eukaryotic MCM DNA replication proteins. BMC Evol Biol 2009 Mar;9:60.
18. Ayoub MS, Baghdadi HM, El-Kholy M. Immunohistochemical detection of laminin-1 and Ki-67 in radicular cysts and keratocystic odontogenic tumor. BMC Clin Pathol 2011 Mar;11:4.
19. Furuyama A, Hosokawa T, Mochitate K. Interleukin-1β and tumor necrosis factor-α have opposite effects on fibroblasts and epithelial cells during basement membrane formation. Matrix Biol 2008 Jun;27(5):429-440.
20. Kichi E, Enokiya Y, Muramatsu T, Hashimoto S, Inoue T, Abiko Y, Shimono M. Cell proliferation, apoptosis, and apoptosis-related factors in odontogenic keratocysts and in dentigerous cysts. J Oral Pathol Med 2005 May;34(5): 280-286.
21. Li TJ, Browne RM, Matthews JB. Epithelial cell proliferation in odontogenic keratocysts: immunocytochemical study of Ki67 in simple, recurrent, and basal cell naevus syndrome (BCNS)-associated lesions. J Oral Pathol Med 1995 May;24(5): 221-226.
22. Kaplan I, Hirshberg A. The correlation between epithelial cell proliferation and inflammation in odontogenic keratocyst. Oral Oncol 2004 Nov;40(10):985-991.
23. Baghaei F, Eslami M, Sadri D. Evaluation of Ki-67 antigen and protein P53 expression in orthokeratinized and parakeratinized odontogenic keratocyst. J Dent 2004 Jan;1(2): 53-58.

CASE REPORT

10.5005/jp-journals-10031-1210

16

Postsurgical Epidermal Inclusion Cyst in the Cheek Region

[1]Amit U Bhandarwar, [2]Shilpa Patel, [3]Jigna Pathak, [4]Niharika Swain, [5]Adil Gandevivala

ABSTRACT

Epidermal inclusion cyst (EIC) is one of the common conditions usually associated with trauma. This cyst commonly presents on the scalp, face, neck, trunk, and extremities. Epidermal inclusion cyst is believed to originate through implantation of epidermal element by either surgical or accidental trauma into deeper mesenchymal tissue and its subsequent cystic transformation. The EICs are indolent in nature, slow to progress, and remain asymptomatic unless secondarily infected. The authors report a case of EIC that occurred in a 35-year-old female after surgery of squamous cell carcinoma.

Keywords: Epidermal inclusion cyst, Epidermoid cysts, Mesenchymal tissue.

How to cite this article: Bhandarwar AU, Patel S, Pathak J, Swain N, Gandevivala A. Postsurgical Epidermal Inclusion Cyst in the Cheek Region. J Contemp Dent 2017;7(3):178-180.

Source of support: Nil

Conflict of interest: None

INTRODUCTION

Epidermal inclusion cysts are rare, slowly growing, benign, and developmental or acquired cysts which are derived from abnormally situated ectodermal tissue.[1] The terminology and nomenclature of EIC is numerous, which includes epidermal cyst, epithelial cyst, keratin cyst, follicular infundibular cyst, seborrheic cyst, milia, and so on.[2] The mainly reported cases are from the sites of face, trunk, neck, extremities and the scalp, genitals, behind the ear, fingers, palm, and soles.[3] About 7% of them are located in the maxillofacial region.

The EIC arises from traumatic implantation of epithelium or entrapment of epithelial remnants during embryonic fusion or by the surgical trauma.[4] The EIC is described as a dermal cystic enclosure of keratinizing squamous epithelium that is filled with keratin debris. The EIC usually presents as a firm, slow-growing, smooth, freely movable, painless mass or lump underneath the skin at the subcutaneous dermal level, with an intact skin surface but no apparent drainage point. It is indolent in nature, slow to progress, and remains asymptomatic, unless secondarily infected. It contains soft, cheesy-like skin secretions. The EICs are approximately twice as common in males than females, can occur at any age, but the third and fourth decade is the most common. Epidermoid cysts are the part of features of certain syndromes like Gardner syndrome, basal-cell nevus syndrome, pachyonychia congenita, which do not demonstrate cysts of the oral mucosa, but facial cysts may occur. They are treated by simple pericapsular excision.[2,3]

In the present study, we report a case of EIC, at surgically operated site of oral squamous cell carcinoma in left cheek region, whose features were rather unusual, in that, it presented as a painless fixed swelling, yellowish black in color, associated with foul smell mimicking an infection.

CASE REPORT

A 35-year-old female patient presented with swelling over left cheek area of face since 2 months (Fig. 1). It had gradually increased to the present size measuring approximately 2 × 1 cm. The patient gave the history of surgery of oral squamous cell carcinoma 9 months back in the same area. The lesion was a diffuse swelling over the left cheek, yellowish black in color with irregular overlying surface. The swelling was tender and firm on

Fig. 1: 35-year-old patient with diffuse swelling over the left cheek area of the face

[1]Postgraduate Student, [2]Professor and Head, [3]Professor [4]Reader, [5]Senior Lecturer

[1-4]Department of Oral Pathology and Microbiology, Mahatma Gandhi Mission's Dental College and Hospital, Navi Mumbai Maharashtra, India

[5]Department of Oral and Maxillofacial Surgery, Mahatma Gandhi Mission's Dental College and Hospital, Navi Mumbai, Maharashtra India

Corresponding Author: Amit U Bhandarwar, Postgraduate Student, Department of Oral Pathology and Microbiology Mahatma Gandhi Mission's Dental College and Hospital, Navi Mumbai, Maharashtra, India, Phone: +917506492789, e-mail: amitbhandarwar29@gmail.com

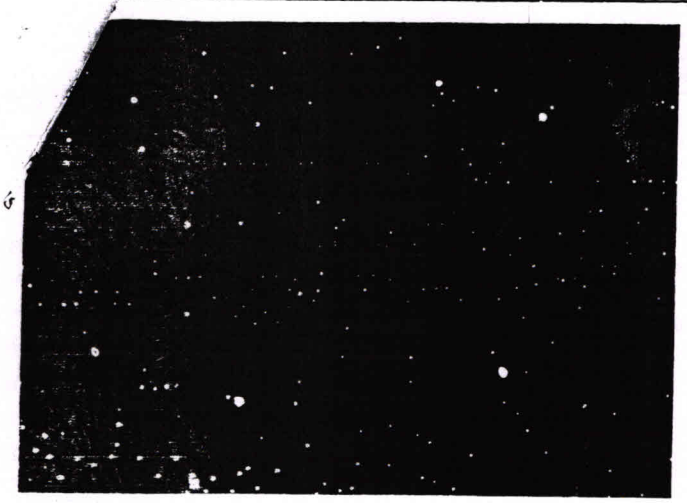


Fig. 4: H & E stained soft tissue section shows orthokeratinzed stratified sqamous epithelium, with a distinct Granular cell layer. The connective tissue capsule showed dense collagen with subepithelial layer of chronic inflammatory cells

range from 0.011 to 0.045%.[6] In our case, surgical excision was done and a 7-month follow-up showed no recurrence.

CONCLUSION

The EIC arises from epidermal inclusion secondary to postoperative trauma, which results in implantation and proliferation of squamous epithelium into the dermis. So care has to be taken to excise it *in toto*, along with the overlying skin and the punctum involved, in order to prevent recurrences from the residual keratin-producing lining of these cysts and to prevent possible malignant transformation.

REFERENCES

1. Nigam JS, Bharti JN, Nair V, Gargade CB, Deshpande AH, Dey B, Singh A. Epidermal cysts: a clinicopathological analysis with emphasis on unusual findings. Int J Trichol 2017 Jul-Sep:9(3):108-112.
2. Jeyaraj P, Sahoo NK. An unusual case of a recurrent seborrheic/epidermal inclusion cyst of the maxillofacial region. J Maxillofac Oral Surg 2015 Mar;14(Suppl 1):176-185.
3. Rajendran, R. Cyst of the orofaciai region. In: Rajendran R, Sivapathasundram B, editors. Shafer's textbook of oral pathology. 8th ed. Amsterdam: Elsevier; 2016. pp. 88-89.
4. Choi HJ, Lee JH, Lee YM. Traumatic epidermoid inclusion cyst on cheek area. J Craniofac Surg 2016 Jun;27(4):e343-e344.
5. Dutta M, Saha J, Biswas G, Chattopadhyay S, Sen I, Sinha R. Epidermoid cysts in head and neck: our experiences, with review of literature. Indian J Otolaryngol Head Neck Surg 2013 Jul;65(Suppl 1):14-21.
6. Mohan C, Srivastava A, Agarwal R, Bhardwaj P. A rare case of epidermoid cyst in neck. Int J Adv Integ Med Sci 2016;1(1): 15-17.

10.5005/jp-journals-10031-1211

CASE REPORT

Fibrolipoma of Lip in a Young Individual: A Rare Presentation

[1]Vishal H Punjabi, [2]Shilpa Patel, [3]Jigna Pathak, [4]Niharika Swain

ABSTRACT

Lipomas are tumors of mature adipose tissue. They are commonly seen in the upper extremities, neck, shoulders, and trunk region. However, oral lipomas are relatively rare. They particularly occur in the areas of fat accumulation, especially the cheek, followed by the tongue, floor of the mouth, buccal sulcus and vestibule, lip, palate, and gingiva. Lipomas can be histopathologically classified into classic lipoma and its variant forms, such as fibrolipomas, spindle cell lipomas, intramuscular lipomas, angiolipomas, salivary gland lipomas, pleomorphic lipomas, myxoid lipomas, and atypical lipomas. There have only been a few cases reported on fibrolipoma involving the lower lip in young individuals. Herein, we present a case report on oral fibrolipoma of the lower lip in a 20-year-old female.

Keywords: Fibrolipoma, Lipoma, Lower lip

How to cite this article: Punjabi VH, Patel S, Pathak J, Swain N. Fibrolipoma of Lip in a Young Individual: A Rare Presentation. J Contemp Dent 2017;7(3):181-184.

Source of support: Nil

Conflict of interest: None

INTRODUCTION

Lipomas are relatively rare in the oral cavity, accounting for 1 to 4.4% of all benign tumors.[1,2] Fibrolipoma is a variant of conventional lipoma. Most patients with this lesion are 40 years of age or older.[3,4] It usually presents as soft, smooth-surfaced nodular masses that can be sessile or pedunculated.[4] It is an uncommon histological variant of the classic lipoma, in which neoplastic fat cells are embedded within dense collagen.[5]

Their etiology and pathogenesis remain unclear, although mechanical, endocrine, and inflammatory influences have been reported.[6] It is suggested that the precursors of adipose cells resemble fibroblasts and that their fat content is acquired by the imbibition of soluble fat or by intracellular elaboration.[4] It is also believed that the fibroblastic component develops, independently from the fat cells, from mesenchymal cells as an intrinsic component of the lipomatous tumour. If fat cells and fibroblasts arise from the same prototype cell, this variant of a lipoma is explicable. Other combinations between lipoblastic tissues and mesenchymal structures are also possible.[4]

Histologically, lipomas are classified as simple lipoma or variants, such as fibrolipoma, spindle cell lipoma, intramuscular or infiltrating lipoma, angiolipoma, salivary gland lipoma (sialolipoma), pleomorphic lipoma, chondroid lipoma, osteoid lipoma, and atypical lipomas.[5,7]

Thus, in this article, we report a case of a fibrolipoma of the lower lip occurring in a young individual.

CASE REPORT

A 20-year-old female patient reported to the Department of Oral Pathology and Microbiology, Mahatma Gandhi Mission's Dental College and Hospital, Navi Mumbai, Maharashtra, India, with the chief complaint of a painless swelling in the right lower lip region. The swelling was noticed by the patient 2 to 3 years earlier, which gradually increased to the present size. The patient gave a history of lip biting; there was no relevant medical history. Intraoral examination revealed a solitary sessile swelling on the right labial mucosa, which was pink in color, soft-to-firm in consistency, and measuring approximately 3 × 2 cm (Fig. 1).

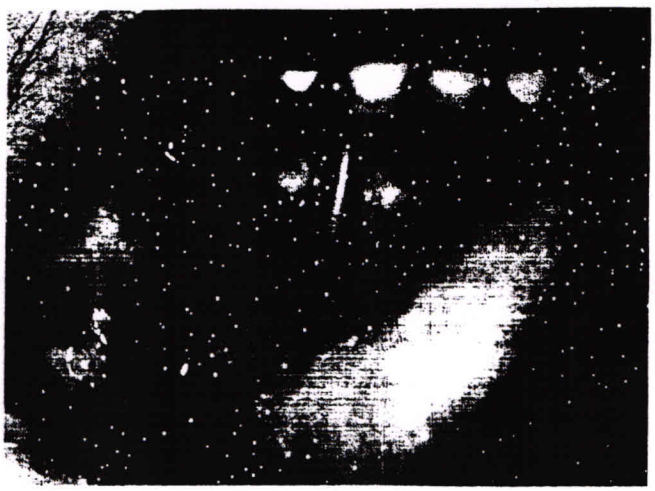


Fig. 1: Intraoral view of solitary sessile swelling on the right lower lip (arrow) showing traumatic ulcerations (arrowhead)

[1]Postgraduate Student, [2]Professor and Head, [3]Professor [4]Reader

[1-4]Department of Oral Pathology and Microbiology, Mahatma Gandhi Mission's Dental College and Hospital, Navi Mumbai Maharashtra, India

Corresponding Author: Vishal H Punjabi, Postgraduate Student, Department of Oral Pathology and Microbiology Mahatma Gandhi Mission's Dental College and Hospital, Navi Mumbai, Maharashtra, India, Phone: +912227436604, e-mail: drvishalpunjabi@gmail.com

REFERENCES

1. Lombardi T, Odell EW. Spindle cell lipoma of the oral cavity: report of a case. J Oral Pathol Med 1994 May;23(5): 237-239.
2. Fregnani ER, Pires FR, Falzoni R, Lopes MA, Vargas PA. Lipomas of the oral cavity: clinical findings, histological classification and proliferative activity of 46 cases. Int J Oral Maxillofac Surg 2003 Feb;32(1):49-53.
3. Vadvadgi VH, Saini R. Oral fibrolipoma of the oral cavity. Int J Experiment Dent Sci 2014 Jul-Dec;3(2):106-108.
4. de Visscher JG. Lipomas and fibrolipomas of the oral cavity. J Maxillofac Surg 1982 Aug;10(3).177-181.
5. Neville BW, Damm DD, Allen CM, Bouquot JE. Oral and maxillofacial pathology. 2nd ed. Philadelphia, PA: Elsevier Publication; 2004.
6. Aust MC, Spies M, Kall S, Gohritz A, Boorboor P, Kolokythas P, Vogt PM. Lipomas after blunt soft tissue trauma: are they real? Analysis of 31 cases. Br J Dermatol 2007 Jul;157(1):92-99.
7. Gnepp DR. Diagnostic surgical pathology of the head and neck. 1st ed. Philadelphia, PA: Saunders; 2010.
8. Gudi SS, Sikkerimath BC, Puranik RS, Kasbe SS. Swelling on lower lip...not always a mucocele !!! Ann Maxillofac Surg 2013 Jan;3(1):98-99.
9. Zhang M, Hayashi H, Fukuyama H, Nakamura T, Kurokawa H, Takahashi T. Traumatic neuroma in the lower lip arising following laser/cryosurgery to treat a mucocele. Oral Dis 2003 May;9:160-116.
10. Sengul I, Sengul D, Aribas D. Pleomorphic adenoma of the lower lip: a rare site of location. N Am J Med Sci 2011 Jun;3(6):299-301.
11. Phukan JP, Sinha A, Mukherjee S, Das UK. Mucoepidermoid carcinoma of the lower lip: a rare site of occurrence. Clin Cancer Investig J 2015 Jan;4(1):54-56.
12. Hatziotis JC. Lipoma of the oral cavity. Oral Surg 1971 Apr;31(4):511-524.
13. Bandéca MC, de Pádua JM, Nadalin MR, Ozório JE, Silva-Sousa YT, da Cruz Perez DE. Oral soft tissue lipomas: a case series. J Can Dent Assoc 2007 Jun;73(5):431-434.
14. Capodiferro S, Maiorano E, Scarpelli F, Favia G. Fibrolipoma of the lip treated by diode laser surgery: a case report. J Med Case Rep 2008 Sep;2:301.
15. Shi J, Zhang J, Ding M, Cao Q. Lower lip cleft, bifid tongue and fibrolipoma: a case report of rare congenital anomaly. Br J Oral Maxillofac Surg 2014 Sep;52(7):e36-e38.
16. Mishra R, Bhasin N, Sahu A, Ghate S. Oral fibrolipoma: a rare presentation case report and review of literature. J Adv Med Med Res 2017;23(10):1-5.
17. Rajendran, R.; Sivapathasundharam, B. Shafer's textbook of oral pathology. 7th ed. Elsevier India; 2012.
18. Iaconetta G, Friscia M, Cecere A, Romano A, Orabona GD, Califano L. Rare fibrolipoma of the tongue: a case report. J Med Case Rep 2015 Aug;9:177.
19. Kiehl RL. Oral fibrolipoma beneath complete denture. J Am Dent Assoc 1980 Apr;100(4):561-562.

CASE REPORT

10.5005/jp-journals-10031-1212

Diffuse Lipomatosis of Face

[1]Vibhuti S Mhatre, [2]Jigna Pathak, [3]Shilpa Patel, [4]Niharika Swain, [5]Adil Gandevivala

ABSTRACT

Congenital infiltrating lipomatosis is a distinct clinicopathological entity. It is a type of lipomatosis that is usually found at birth or early after birth. It is designated by a collection of nonencapsulated, mature adipocytes that infiltrate local tissues, leading to craniofacial deformities. Due to its diffuse infiltration and involvement of important facial structures, a complete surgical excision is often impossible. We report a case of a 5-year-old female patient presenting with a painless swelling on the left side of her face.

Keywords: Diffuse lipomatosis, Infiltrating lipomatosis, Lipomatous tumor.

How to cite this article: Mhatre VS, Pathak J, Patel S, Swain N, Gandevivala A. Diffuse Lipomatosis of Face. J Contemp Dent 2017;7(3):185-187.

Source of support: Nil

Conflict of interest: None

INTRODUCTION

Congenital infiltrating lipomatosis is a distinct clinicopathological entity. It is a type of lipomatosis that is usually found at birth or early after birth.[1] It comes under the subset of lipomatous tumor-like lesions and is designated by collection of nonencapsulated, mature adipocytes that infiltrate local tissues, leading to craniofacial deformities and is prone to recur postsurgery.[2] The tumor-like lesion is congenital in origin and presents in infancy or early childhood as unilateral facial asymmetry.[3] Until date, fewer than 50 cases have been reported in English literature.[2,4] We report a case of a 5-year-old female patient presenting with a painless swelling on the left side of her face.

[1]Postgraduate Student, [2]Professor, [3]Professor and Head [4]Reader, [5]Senior Lecturer

[1-4]Department of Oral Pathology and Microbiology, Mahatma Gandhi Mission's Dental College and Hospital, Navi Mumbai Maharashtra, India

[5]Department of Oral and Maxillofacial Surgery, Mahatma Gandhi Mission's Dental College and Hospital, Navi Mumbai Maharashtra, India

Corresponding Author: Vibhuti S Mhatre, Postgraduate Student, Department of Oral Pathology and Microbiology Mahatma Gandhi Mission's Dental College and Hospital, Navi Mumbai, Maharashtra, India, Phone: +919920900459, e-mail: vibsm.vm@gmail.com

CASE REPORT

A 5-year-old female patient reported to our department with a chief complaint of a painless swelling on the left middle and lower third of the face. The swelling was noticed by her parents at birth and was gradually increasing in size with age. Patient did not have any difficulty in speech or loss of hearing. There was no other relevant history.

Extraoral examination revealed gross facial asymmetry due to a solitary diffuse swelling on the left side of the face, extending from left zygomatic arch up to the left inferior border of the mandible (Fig. 1). Borders were indistinct and the skin over the swelling was normal. On palpation, there was no increase in surface temperature and the swelling was soft, nontender, noncompressible, and nonpulsatile. No lymph nodes were palpable. Magnetic resonance imaging of the patient showed hyperintense lesion obliterating left maxillary sinus in T1 images with subcutaneous fat deposition. Based on these findings, a provisional diagnosis of congenital diffuse lipomatosis was made. Patient was referred to the Department of Oral & Maxillofacial Surgery for biopsy.

Grossly, several bits of soft tissue were received with the largest rectangular bit, blackish white color, measuring 2.5 × 1.0 × 1.0 cm. Histopathological examination revealed hematoxylin and eosin-stained soft tissue section showing sinus lining comprising ciliated pseudostratified squamous epithelium with focal hyperplasia. The underlying connective tissue stroma showed varying degrees of inflammatory cell infiltration, minor salivary

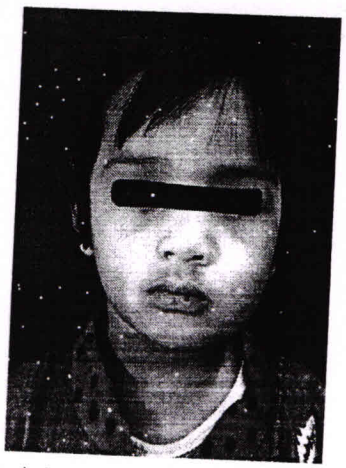


Fig. 1: Clinical photograph of a patient with unilateral facial swelling on the left side

The histopathologic features include a nonencapsulated lesion with proliferating mature adipose tissue, which is surrounded by a dense capillary network. There is diffuse infiltration of adjacent soft tissue and presence of fibrous tissue with various nerve bundles and thickened wall vessels. The absence of lipoblasts and signs of malignancy despite a rapid growth rate is observed. It may also show hypertrophy of subjacent bone.[1,3,7] Our case also revealed uncapsulated lesional tissue comprising lobular arrangement of adipocytes with infiltration to surrounding structures.

The treatment options available are liposuction and surgical excision. Treatment is primarily for esthetic reasons.[11,18,19] Although the tumors are benign, the rate of recurrence is very high, up to 58.6% after surgical excision.[10]

CONCLUSION

Congenital infiltrating lipomatosis of the face is a rare benign disorder of lipomatous tissue in infancy or childhood. When patients with facial asymmetry are reported, this should be considered. Thorough clinical examination, imaging studies, and histopathological examination help in the appropriate diagnosis. The chief motive of surgery is to improve the cosmetic appearance of the face rather than to eradicate the tumor.

REFERENCES

1. Chen CM, Lo LJ, Wong HF. Congenital infiltrating lipomatosis of the face: case report and literature review. Chang Gung Med J 2002 Mar;25(3):194-200.
2. Singh K, Sen P, Musgrove BT, Thicker N. Facial infiltrating lipomatosis: a case report and review of literature. Int J Surg Case Rep 2011 Jun;2(7):201-205.
3. Slavin SA, Baker DC, McCarthy JG, Mufarrij A. Congenital infiltrating lipomatosis of the face: clinicopathologic evaluation and treatment. Plast Reconstr Surg 1983 Aug;72(2):158-164.
4. Rajeswaran R, Murthy J, Chandrasekharan A, Joseph S. Case report: congenital infiltrating lipomatosis of face. Indian J Radiol Imaging 2008 Dec;18(4):306-309.
5. Weiss, S.; Goldblum, J. Enzinger and Weiss's soft tissue tumors. 5th ed. St Louis: Mosby; 2008. pp. 429-466.
6. Bouletreau P, Breton P, Friedel M. Congenital infiltrating lipomatosis of the face: case report. J Oral Maxillofac Surg 2000 Jul;58(7):807-810.
7. De Rosa G, Cozzolino A, Guarino M, Giardino C. Congenital infiltrating lipomatosis of the face. J Oral Maxillofac Surg 1987 Oct;45(10):879.
8. Donati L, Candiani P, Grappolini S, Klinger M, Signorini M. Congenital infiltrating lipomatosis of the face related to CMV infection. Br J Plast Surg 1990 Jan;43(1):124-126.
9. Patel RV, Gondalia JS. Congenital infiltrating lipomatosis of the face (comment letter). Br J Plast Surg 1991;44:157-158.
10. Balaji SM. Congenital diffuse infiltrating facial lipomatosis. Ann Maxillofac Surg 2012 Jul-Dec;2(2):190-196.
11. Heymans O, Ronsmans C. Congenital infiltrating lipomatosis of the face. Eur J Plast Surg 2005;28:186-189.
12. Urs AB, Augustine J, Kumar P, Arora S, Aggarwal N, Sultana N. Infiltrating lipomatosis of the face: a case series. J Nat Sci Biol Med 2013 Jan;4(1):252-257.
13. Shenoy AR, Nair KK, Lingappa A, Shetty KS. Congenital infiltrating lipomatosis of face: case report and review of literature. J Indian Soc Pedod Prev Dent 2015 Apr-Jun;33(2):156-160.
14. Padwa BL, Mulliken JB. Facial infiltrating lipomatosis. Plast Reconstr Surg 2001 Nov;108(6):1544-1554.
15. Mac Millan AR, Oliver AJ, Reade PC, Marshall DR. Regional macrodontia and regional bony enlargement associated with congenital infiltrating lipomatosis of the face presenting as unilateral facial hyperplasia. Int J Oral Maxillofac Surg 1990 Oct;19(5):283-286.
16. Kang N, Ross D, Harrison D. Unilateral hypertrophy of the face associated with infiltrating lipomatosis. J Oral Maxillofac Surg 1998 Jul;56(7):885-887.
17. Kim JE, Gottschall JA, Bachman RP, Nemzer L, Puligandla B, Schauer G. Facial infiltrating lipomatosis: physical, radiological, and histopathological findings. Arch Otolaryngol Head Neck Surg 2010 Mar;136(3):301-303.
18. Coffin, CM. Adipose and myxoid tumors. Pediatric soft tissue tumors: a clinical, pathological, and therapeutic approach. Baltimore: Williams and Wilkins Co; 1997. p. 245.
19. Mahadevappa A, Raghavan VH, Ravishankar S, Manjunath GV. Congenital infiltrating lipomatosis of the face: a case report. Case Rep Pediatr 2012;Article ID 134646:3 pages.

10.5005/jp-journals-10031-1195

ORIGINAL RESEARCH

Evaluation of Nuclear Morphometry in Oral Squamous Cell Carcinoma: A Retrospective Study

[1]Nikitha Narayanan, [2]Jigna Pathak, [3]Shilpa Patel, [4]Niharika Swain

ABSTRACT

Aim: To evaluate and compare various nuclear morphometric parameters in different histopathological grades of oral squamous cell carcinoma (OSCC) by using computerized image analysis and also to correlate it with regional lymph node metastasis.

Materials and methods: This retrospective study was conducted on paraffin blocks of 40 tissue specimens of OSCC cases treated with neck dissection, which were retrieved from the archives of the Department of Oral Pathology and Microbiology, Mahatma Gandhi Mission's Dental College and Hospital, Navi Mumbai, India. All cases were histopathologically graded as well, moderately, and poorly differentiated OSCC. Further, they were also categorized based on pathological lymph node status as with or without lymph node metastasis. Sections from tumor proper were subjected to Feulgen nuclear staining technique. Images of 10 microscopic fields at the deepest invading part of tumor were captured randomly and 100 nuclei of tumor cells with clear, complete, nonoverlapping outlines were selected in each case. Nuclear morphometric parameters, such as large diameter, small diameter, nuclear area, and nuclear perimeter were measured for each of the 100 cells.

Results: An increase in mean nuclear area coefficient of variation (NACV) was observed in OSCC cases with lymph node metastasis pN(+) than in those without lymph node metastasis pN(–), ($p = 0.67$). A significant increase in nuclear area and perimeter was observed in pN(+) cases ($p < 0.01$). A significant decrease in circular rate and increase in largest to smallest nuclear diameter (L/S) ratio ($p < 0.01$) was observed in pN(+) cases. On comparing the nuclear morphometric parameters with different histopathological grades of OSCC, we found an increase in mean NACV values from well-differentiated OSCC to moderately differentiated and poorly differentiated OSCC ($p = 0.612$). An increase in mean nuclear area and perimeter was noted as grades of OSCC advanced ($p > 0.01$). The mean circular rate was found to be lowest in poorly differentiated OSCC ($p = 0.362$). A significant increase in mean L/S ratio was observed within different histopathological grades of OSCC ($p = 0.044$), which when further confirmed using least significant difference (LSD) *post hoc* test, indicated a difference only between well-differentiated and poorly differentiated cases of OSCC ($p = 0.132$).

Conclusion: Our observations reveal that tumor cells with greater nuclear dimension and more elliptical shape tend to show increased incidence of nodal metastasis. Also, a positive inclination was observed in nuclear size and shape with increasing histopathological grades of OSCC. However, our data warrant a large-scale study to establish nuclear morphometry as a quantitative objective parameter and also for the rational application of the same.

Keywords: Image analysis, Lymph node, Nuclear morphometry, Oral squamous cell carcinoma.

How to cite this article: Narayanan N, Pathak J, Patel S, Swain N. Evaluation of Nuclear Morphometry in Oral Squamous Cell Carcinoma: A Retrospective Study. J Contemp Dent 2017;7(2):107-113.

Source of support: Nil

Conflict of interest: None

INTRODUCTION

Oral cancer is one of the most dreaded and aggressively combated diseases of this era. Worldwide, oral cancer accounts for 2 to 4% of all cancer cases.[1] The annual estimated incidence for oral cancer is around 275,000.[2] The prevalence of oral cancer is estimated to be around 45% in India.[3,4] Oral and pharyngeal cancer, grouped together, is the sixth most common cancer in the world.[5] Of all malignancies in the head and neck region, OSCC constitutes a majority of 90 percent.[6]

Despite the advances of therapeutic approaches, percentages of morbidity and mortality of OSCC have not improved significantly since the last 30 years.[1] This could be attributed to delay in diagnosis, the rapid advancement of the disease, and inaccessibility to tumor due to their metastasis.

Locoregional lymph node metastasis occurs frequently in patients with head and neck cancer.[7] In OSCC, tumor metastases in the regional lymph nodes at presentation is the most significant adverse prognostic factor and a major determinant of poor survival.[8] The process of metastasis in regional lymph nodes begins with invasive growth of tumor cells and its detachment from the primary lesion. This is followed by lymphogenous transport of cancer cells that results in lodgment

[1]Postgraduate Student, [2]Professor, [3]Professor and Head [4]Lecturer

[1-4]Department of Oral Pathology and Microbiology, Mahatma Gandhi Mission's Dental College and Hospital, Navi Mumbai Maharashtra, India

Corresponding Author: Nikitha Narayanan, Postgraduate Student, Department of Oral Pathology and Microbiology Mahatma Gandhi Mission's Dental College and Hospital, Navi Mumbai, Maharashtra, India, Phone: +9127436604, e-mail: nike.jn86@gmail.com

for evaluating the high metastatic potential of colorectal adenocarcinoma. Cancer 1999 Nov;86(10):1944-1951.

23. Dobroś W, Gil K, Ryś J, Stanisz-Wallis K. Nuclear morphometry for the prediction of regional lymph nodes metastases in patients with cancer of the larynx. Otolaryngol Head Neck Surg 2000 Dec;123(6):770-774.
24. Pienta KJ, Coffey DS. Correlation of nuclear morphometry with progression of breast cancer. Cancer 1991 Nov;68(9): 2012-2016.
25. Sekine J, Uehara M, Hideshima K, Irie A, Inokuchi T. Predictability of lymph node metastases by preoperative nuclear morphometry in squamous cell carcinoma of the tongue. Cancer Detect Prev 2003;27(6):427-433.
26. Pardoll DM, Vogelstein B, Coffey DS. A fixed site of DNA replication in eucaryotic cells. Cell 1980 Feb;19(2):527-536.
27. Vogelstein B, Pardoll DM, Coffey DS. Supercoiled loops and eucaryotic DNA replication. Cell 1980;22(1):79-85.
28. Nathanson SD. Insights into the mechanisms of lymph node metastasis. Cancer 2003 Jul;98(2):413-423.
29. Nandini DB, Subramanyam RV. Nuclear features in oral squamous cell carcinoma: a computer assisted microscopic study. J Oral Maxillofac Pathol 2011 Sep;15(2):177-181.
30. Pienta KJ, Partin AW, Coffey DS. Cancer as a disease of DNA organization and dynamic cell structure. Cancer Res 1989 May;49(10):2525-2532.
31. François C, Decaestecker C, Petein M, van Ham P, Peltier A, Pasteels JL, Danguy A, Salmon I, van Velthoven R, Kiss R. Classification strategies for the grading of renal cell carcinomas, based on nuclear morphometry and densitometry. J Pathol 1997 Oct;183(2):141-150.

RESEARCH ARTICLE

Adenosine Deaminase - a Novel Diagnostic and Prognostic Biomarker for Oral Squamous Cell Carcinoma

Deepak Chandrakant Kelgandre[1]*, Jigna Pathak[1], Shilpa Patel[2], Pramod Ingale[3], Niharika Swain[1]

Abstract

Background: The number of patients with oral cancer in India is increasing gradually (especially in younger people). Although the diagnostic modalities and therapeutic management of oral cancer are improving, the treatment outcome and prognosis of oral cancer remain poor. The absence of definite early warning symptoms for most head and neck cancers suggests that sensitive and specific biomarkers are likely to be important in screening for high-risk patients. Aims: To analyze serum adenosine deaminase (ADA) levels in oral squamous cell carcinoma (OSCC) cases who reported to our institute. Materials and Methods: A prospective study was performed on 100 histopathologically proven cases of OSCC (study group) and 100 normal healthy individuals (control group). Independent sample and one sample t-tests and one way ANOVA followed by Tuckey's POST HOC test were conducted for analysis. Results: Statistically significant increase in serum ADA levels was observed in OSCC cases compared to the control group. Also serum ADA level increased significantly with the histopathological grade. Conclusions: Serum ADA levels in OSCC may be a useful diagnostic and prognostic biomarkers in clinical practice and our findings suggest that a large-scale study is warranted to confirm clinical utility as a prognostic and diagnostic biomarker.

Keywords: Oral SCC - adenosine deaminase - biomarker - prognosis

Asian Pac J Cancer Prev, **17** (4), 1865-1868

Introduction

Failure or delay in the early diagnosis of oral squamous cell carcinoma (OSCC) is one of the primary causes of high mortality and morbidity in cancer patients (Ferlay et al., 2004). Further insights into the mechanisms leading to malignancy are prerequisite for identifying new biomarkers for OSCC from the serum. The idea of screening and following patients with malignancy by serum analysis is appealing from several points of view including its ease, economical advantage, non-invasiveness and possibility of repeated sampling.

Serum ADA level is shown to be increased in cancers of many tissues like colon, bladder, breast, esophagus and liver. Therefore aim of the study was to analyze Serum ADA level in OSCC and objectives of the study were to compare serum ADA level of OSCC patients with normal healthy individuals, to correlate serum ADA level of OSCC patients with histopathological grades of OSCC and to determine the diagnostic and prognostic implications of serum ADA level in OSCC patients.

Materials and Methods

In the present prospective study (n=90) OSCC patients who reported to our institute were selected as a study group. Detailed case history of all the patients was taken. Exclusion criteria includes cases having history of prior treatment for OSCC or other malignancies, Cases suffering from granulomatous diseases like tuberculosis and cases suffering from any other malignancy. Incisional biopsy was performed in clinically suspected cases of Oral carcinomas under local anesthesia by using 2% Lignocaine.

Tissue samples were processed by routine method and paraffin embedded blocks was made. Paraffin embedded specimen was cut into sections of 5 micrometer thickness by using the soft tissue microtome and sections were stained by using hematoxylin and eosin stain. According to descriptive criteria for OSCC grading provided by Royal College of Pathologists and WHO 2005, patients were categorized into 3 grades i.e. well differentiated OSCC, moderately differentiated OSCC and poorly differentiated

[1]*Oral Pathology and Microbiology, Dentistry, YCMM & RDFs Dental College and Hospital, Ahmednagar,* [2]*Dept Oral Pathology and Microbiology, , MGM Dental College and Hospital, Navi Mumbai, Navi,* [3]*Dept Biochemistry, LTMC, Mumbai, Mumbai, India*
**For correspondence: drdeepak144@gmail.com*

In our study mean serum ADA level in well, moderate and poorly differentiated grade were 27.3 IU/L, 31.8 IU/L and 33.5 IU/L respectively. We found that serum ADA level gradually increased from well, moderate to poorly differentiated OSCC grade. Statistically significant difference was present between well & moderate grade, and between well & poor grade, but insignificant difference between moderate & poor grade. In the literature previous studies mentioned the correlation of serum ADA level with different clinical stages of oral cancer. To the best of our knowledge no study correlated the serum ADA levels with different histopathological grades. Harbans Lal et al in 1987 evaluated the serum ADA in 40 OSCC patients of different stages and found the ADA activity is increased with advancement in the clinical stage of the cancer. Study done by R. Mishra et al. (2000) found significant correlation between increased levels of ADA with lymph node involvement and concluded that this may help in assessing the decrease in tumor mass and improvement in patient & clinical condition. Ashok et al in 2008 found that there was a highly significant correlation between the serum ADA level and the increasing disease stage (severity of the disease), the tumor status and metastasis of the tumor to the neck nodes. They concluded that Serum ADA levels can be used as one of the diagnostic tools in head and neck cancer.

ADA is an enzyme of the purine salvage pathway. ADA is present on the cell surface as well as intra-cellularly, but it does not have its own transmembrane domain and is associated with CD26, a surface glycoprotein with dipeptidyl peptidase IV activity (Franco, 1997). Due to the rapid growth, solid tumors routinely experience severe hypoxia and necrosis leading to adenine nucleotide degradation and adenosine release (Linden, 2006). The released adenosine constitutes supportive environment for tumor growth by means of protection against ischemia. At the same time it stimulates the growth and angiogenesis as well as suppresses immune response. So increase in adenosine production leads to increase in production of ADA. Also, in cancer there is an increased turnover of malignant cells and an associated increase in nucleotide metabolism leading to an increase in purine metabolizing enzymes. ADA is particularly sensitive to stimulation by growth factors and cytokines during rapid tissue proliferation such as IL-2, IL12 and INFγ which increases during malignancy (Ashok et al., 2008).

Results of our study showed that serum ADA level increases significantly in OSCC as compared to control group. It shows that nucleotide (purine & pyrimidine) metabolism increases in OSCC due to increase in DNA turnover, and serum level increases because of leakage of enzyme from primary malignant cells and lymphatic metastasis. In the literature it is mentioned that the prognosis of OSCC worsened from well to poorly differentiated grade, also serum ADA is known to increase with disease progression. In our study, serum ADA level increased from well to moderate to poorly differentiated OSCC. Therefore it can be suggested that serum ADA can be used as a diagnostic and also prognostic biomarker for OSCC. Also the simplicity of measuring ADA activity combined with its cost effectiveness gives an added advantage to consider ADA as a tumor marker in oral cancer. However, during evaluation of serum ADA level one has to keep in mind that the level of these markers altered in different systemic conditions such as various cancers, infections etc, and so these factors should be ruled out.

Thus, from our study observations it is seen that, serum ADA level increases in OSCC, also the level of these markers increases according to histopathological grade. Hence we propose that serum ADA can be used as a diagnostic as well as prognostic bio-marker in OSCC.

References

Ashok KJ, Pinto GJO, Kavitha AK, et al (2008). The diagnostic and prognostic value of serum adenosine deaminase levels in head and neck cancer. *J Clin Diag Res*, **3**, 833-37.

Balis EM (1985). Adenosine deaminase and malignant cells. *Ann NY Acad sci*, **451**, 142-9.

Franco R (1997). Cell surface adenosine deaminase: much more than an ectoenzyme. *Prog Neurobiol*, **52**, 283-94.

Ferlay J, Pisani P, Parkin DM (2004). Globocan. Cancer incidence, mortality and prevalence worldwide. Lyon: IARC Press Guesti G (1974). Adenosine deaminase In: Bergmeyer HU editor. Methods of enzymatic analysis, 2nd Ed, 2. New York: Academic press Inc, 1092-9.

Harbans lal, SK Munjal, Umesh Wig, et al (1987). Serum enzymes in head and neck cancer III. *J Laryngol Otol*, **101**, 1062-5.

Kalcioglu MT (2004). Adenosine deaminase, xanthine oxidase, superoxide dismutase, glutathione peroxidase activities and malondialdehyde levels in the serum of patients with head and neck carcinoma. *Kulak Burun Bogaz Ihtis Derg*, **12**, 16-22.

Kerawala CJ (2010). Complications of head and neck cancer surgery: Prevention and management. *Oral Oncol*, **46**, 433-5.

Linden J (2006). Adenosine metabolism and cancer. *Am J Physiol Cell Physiol*, **17**, 1-7.

Mini walia, Mridula Mahajan, K Singh (1995). serum adenosine deaminase, 5`- nucleotidase & alkaline phosphatase in Breast cancer Patients. *Indian J Med Res*, **101**, 247-9.

Suchitra MM, Prabhakar Reddy E, Muni Sudhakar G, et al (2009). Evaluation of serum adenosine deaminase as a tumor marker in gastric cancer. *Res J Medicine Med Sci*, **4**, 411-4.

Pragathi P, Bharath Kumar, Amar Kumar P, et al (2005). Evaluation of serum adenosine deaminase and 5`-nucleotidase activities as probable markers in ovarian cancer. *Indian J Clin Bio*, **20**, 195-7.

Mishra R, Agarwal MK, Chansuria JPN (2000). Serum adenosine deaminase levels as an index of tumor growth in head and neck malignancy. *Indian J Otolaryngol Head Neck Surg*, **52**, 360-3.

Rakesh Dhankhar, Kiran Dahiya, Tarun Kumar Sharma, et al (2011). Diagnostic significance of adenosine deaminase, uric acid and C-reactive protein levels in patients of head and neck carcinoma. *Clin Lab*, **57**, 795-8.

Dermirtas S (1996). Adenosine deaminase, 5`-nucleotidase, guanase and cytidine deaminase activites in gastric and breast-cancer. *SDV Tip Fakultesi Dergisi*, **3**, 1-4.

St. John, MAR, Li Y, Zhou X, et al (2004). Interleukin 6 and interleukin 8 as potential biomarkers for oral cavity and oropharyngeal squamous cell carcinoma. *Arch Otolaryngol Head Neck Surg*, **103**, 929-35.

Warnakulasuriya S (2009). Global epidemiology of oral and pharyngeal cancer. *Oral Oncol*, **45**, 309-16.

JCD

10.5005/jp-journals-10031-1160

CASE REPORT

Glandular Odontogenic Cyst Mimicker: A Novel Entity

[1]Kehkashan E Azmi, [2]Shilpa Patel, [3]Jigna Pathak, [4]Niharika Swain

ABSTRACT

Glandular odontogenic cyst (GOC) is an uncommon developmental odontogenic cyst of jaws with a frequency of approximately 0.2%. Because of its aggressive biological behavior including its malignant transformation potential, recently collaborative efforts by few researchers have laid down certain histopathological criteria segregating it from its mimickers which include dentigerous cyst, lateral periodontal cyst (LPC), or botryoid cyst, radicular cyst, and central low-grade mucoepidermoid carcinoma. Therefore, cautious histopathological evaluation is necessary of GOC mimickers in order to prevent its overdiagnosis. Here, we present a case of GOC mimicker in a 12-year-old male patient in left maxillary region.

Keywords: Glandular odontogenic cyst mimicker, Hobnail cell, Sialo-odontogenic cyst.

How to cite this article: Azmi KE, Patel S, Pathak J, Swain N. Glandular Odontogenic Cyst Mimicker: A Novel Entity. J Contemp Dent 2016;6(2):145-148.

Source of support: Nil

Conflict of interest: None

INTRODUCTION

Glandular odontogenic cyst (GOC) was first documented as "sialo-odontogenic cyst" by Padayachee and Van Wyk in 1987.[1] However, in 1988, Gardner et al[2] proposed it as a distinct entity and gave a term "GOC" whereas High et al[3] proposed the term "polymorphous odontogenic cyst" which is characterized by a potentially aggressive behavior with a high recurrence rate.[4] Glandular odontogenic cyst is a rare lesion comprising only 0.2% of all odontogenic cysts. The cyst is most frequently encountered in the age range of 14 to 75 years with male predilection (1.3:1), with predilection for mandible (70%) affecting both anterior and posterior areas. Radiographically, it is radiolucent, well-defined, either unilocular (53.8%) or multilocular (46.2%) with frequent perforation (61%) and thinning of cortical plates (24.4%). Furthermore, English literature revealed recurrence rate which varies from 21 to 55%[5,6] which indicates its aggressiveness.[7] Therefore, a confirmatory diagnosis is critical for treatment and follow-up. However, similar histopathological features are shared between GOC and other odontogenic cysts, such as botryoid odontogenic cyst, dentigerous cyst with metaplasia, radicular cyst, or central mucoepidermoid carcinoma (CMEC), thereby making the diagnosis more cumbersome.[7] However, even though the microscopic features of GOC have been described in detail, there is no consensus on its diagnostic features. It is sometimes difficult to discern whether a particular cyst having some but not all of the described features of GOC represents a true GOC or another cyst with GOC-like features imposing a need to introduce a new group "glandular odontogenic cyst mimickers" (GOC mimicker).

The most recent World Health Organization classification includes a definition of the GOC – "A cyst arising in the tooth bearing areas of the jaws and characterized by an epithelial lining with cuboidal or columnar cells, both at the surface and lining, with crypts or cyst-like spaces within the thickness of the epithelium."[8] Few authors have selected parameters based on previously reported microscopic features for GOC from the literature.[9,10] Kaplan et al[7] proposed major and minor microscopic criteria for GOC based on the frequency of each feature in reported cases from the literature. Fowler has emphasized on certain criteria which are mandatory in differentiating a true GOC from GOC mimicker. The purpose of this paper is to present a case report depicting the clinical, radiographic, and histopathologic features of a GOC mimicker with particular emphasis on microscopic parameters necessary for diagnosis.

CASE REPORT

A 12-year-old male patient reported in the Department of Oral Pathology, MGM Dental College and Hospital, Navi Mumbai with a chief complaint of painful swelling in upper left anterior region of the jaw since 3 months. Extraorally, a diffuse swelling was evident which resulted in facial asymmetry. Intraoral examination revealed a solitary diffuse buccal swelling in the left maxillary region extending from 21 to 25, approximately 3×2×1 cm in size (Figs 1A and B). The swelling extended superoinferiorly from attached gingiva obliterating the buccal vestibule. Displacement of 21 was also evident. Overlying mucosa was smooth and erythematous. On palpation, the swelling was firm, tender, nonmobile, and nonpulsatile. Radiographic examination

[1]Postgraduate Student, [2]Professor and Head, [3]Professor [4]Lecturer

[1-4]Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India

Corresponding Author: Kehkashan E Azmi, Postgraduate Student, Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India Phone: +918588912729, e-mail: drkehkashan.azmi@gmail.com

In our present case, clinical, radiological, and incisional biopsy report lead to the diagnosis of dentigerous cyst with metaplastic changes which include hobnail cells with eosinophilia, squamous epithelial lining with a flat interface with the connective tissue wall, lacking basal palisading and clear cells. These features are analogous with the mentioned criteria.[4,7,14] There was presence of pseudomicrocysts (microcyst lined by flattened cells) in contrast to the true microcysts (cuboidal to columnar cells lining) which are seen in GOC lining, thereby validating our histopathological diagnosis of GOC mimicker.

Glandular odontogenic cyst mimickers are an intermediate between conventional lesions and GOC, so persistent efforts should be carried out for their seclusion, so that their exact biological behavior could be traced out.

CONCLUSION

This paper aims to depict a clear picture toward histopathological diagnosis so as to differentiate GOC mimickers from GOC to prevent overdiagnosis, as GOC has a higher recurrence rate. Further studies need to be undertaken to have a cumulative data to explore the exact biological behavior of this budding entity.

REFERENCES

1. Padayachee A, Van Wyk CW. Two cystic lesions with features of both the botryoid odontogenic cyst and the central mucoepidermoid tumor: sialo-odontogenic cyst? J Oral Pathol 1987 Nov;16(10):499-504.
2. Gardner DG, Kessler HP, Morency R, Schaner DL. The glandular odontogenic cyst: an apparent entity. J Oral Pathol 1988 Sep;17(8):359-366.
3. High AS, Main DM, Khoo SP, Pedlar J, Hume WJ. The polymorphous odontogenic cyst. J Oral Pathol Med 1996 Jan;25(1):25-31.
4. Kaplan I, Gal G, Anavi Y, Manor R, Calderon S. Glandular odontogenic cyst: treatment and recurrence. J Oral Maxillofac Surg 2005 Apr;63(4):435-441.
5. Rasmusson LG, Magnusson BC, Borrman H. The lateral periodontal cyst. A histopathologic and radiographic study of 32 cases. Br J Oral Maxillofac Surg 1991 Feb;29(1):54-57.
6. Hussain K, Edmondson HD, Browne RM. Glandular odontogenic cysts. Diagnosis and treatment. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1995 May;79(5):593-602.
7. Kaplan I, Anavi Y, Hirshberg A. Glandular odontogenic cyst: a challenge in diagnosis and treatment. Oral Dis 2008 Oct;14(7):575-581.
8. Kramer IRH, Pindborg JJ, Shear M. Histological typing of odontogenic tumors. In: WHO, editor. International histological classification of tumors. 2nd ed. Berlin: Springer-Verlag; 1992.
9. Koppang HS, Johannessen S, Haugen LK, Haanaes HR, Solheim T, Donath K. Glandular odontogenic cyst (sialo-odontogenic cyst): report of two cases and literature review of 45 previously reported cases. J Oral Pathol Med 1998 Oct;27(9):455-462.
10. de Sousa SO, Cabezas NT, de Oliveira PT, de Araújo VC. Glandular odontogenic cyst: report of a case with cytokeratin expression. Oral Surg Oral Med Oral Pathol Radiol Endod 1997 Apr;83(4):478-483.
11. Shear M. Cysts of the oral region. 3rd ed. Mumbai: Varghese Publishing House; 1992.
12. Browne RM. Metaplasia in odontogeni cysts. J Dent Res 1971;50(Suppl):1177.
13. Gorlin RJ. Potentialities of oral epithelium manifest by mandibular dentigerous cysts. Oral Surg Oral Med Oral Pathol 1957 Mar,10(3):271-284.
14. Fowler C, Brannon R, Kessler H, Castle J, Kahn M. Glandular odontogenic cyst: analysis of 46 cases with special emphasis on microscopic criteria for diagnosis. Head Neck Pathol 2011 Dec;5(4):364-375.

JCD

CASE REPORT

10.5005/jp-journals-10031-1161

Giant Aneurysmal Bone Cyst of the Mandible

[1]Rajshri U Gurav, [2]Jigna Pathak, [3]Shilpa Patel, [4]Niharika Swain

ABSTRACT

An aneurysmal bone cyst (ABC) is a benign osteolytic bony lesion that commonly affects the long bones with rare presentation in the jaws. The etiopathogenesis of ABC is unsure. Several theories have been suggested like trauma, intramedullary hematoma, alterations in local hemodynamics, reactive malformation, and genetic predisposition. Though ABCs are considered as secondary phenomenon in preexisting benign and malignant bony lesions, intermittent reports of ABCs with primary/denovo origin are generating perplexity in the scenario.

Here, we describe a rare case of giant ABC involving mandible extending from right angle of mandible to left canine region which crosses midline, in a 10-year-old female patient, without any evidence of preexisting bony lesion.

Keywords: Aneurysmal bone cyst, Mandible, Primary/denovo, Secondary phenomenon.

How to cite this article. Gurav RU, Pathak J, Patel S, Swain N. Giant Aneurysmal Bone Cyst of the Mandible. J Contemp Dent 2016;6(2):149-153.

Source of support: Nil

Conflict of interest: None

INTRODUCTION

Aneurysmal bone cyst (ABC) was first reported by Van Arsdale in 1893, who described it as ossifying hematoma.[1] Jaffe and Lichtenstein in 1942 were the first to recognize ABC as an intraosseous, osteolytic lesion, chiefly affecting the metaphyseal region of long bones and vertebrae.[1] Bernier and Bhaskar in 1958 described the first case of ABC in the jaws.[2,3] In 1972 Schajowicz in his Histopathological Classification of Primary Bone Tumors placed ABC in group IX tumor-like lesions, which was latter modified by World Health Organization (WHO) in 1993.[4]

The WHO defined ABC as a benign tumor-like lesion with an expanding osteolytic lesion consisting of blood-filled spaces of variable size separated by connective tissue septa containing trabeculae or osteoid tissue and osteoclast-like giant cells.[5] Aneurysmal bone cyst is considered a pseudocyst because it is not lined by epithelium.[2] Fifty percent of ABCs arise in the long bones and 20% in the vertebral column; and only about 12% affect the head and neck region of which 2% occurs in jaws.[6,7] Mandible is more frequently affected than the maxilla, the proportions varying from 2:1 to 11:9. The body, ramus, and angle of the mandible are the main locations; rarely it occurs in mandibular condyle and coronoid process.[7] It generally affects young adults below 20 years of age and there is no definite gender predilection.[6,8]

Various theories have been proposed for etiopathogenesis of ABCs including post traumatic, reactive malformation, genetic predisposition, and dilatation of local vascular network due to increased venous pressure caused by local circulatory abnormalities.[9,10] Consistent finding of ABC, like changes in preexisting primary bony lesions, gave rise to the well-accepted theory that it is a secondary phenomenon. Cumulative evidences of ABCs without any association of preexisting lesions are now raising a question on exact etiopathogenesis suggesting a primary/denovo variant.[8,10]

We hereby report a case of giant ABC without any histological evidence of preexisting bony lesion, involving mandible extending from right angle of mandible to left canine region which crosses midline in 10-year-old female patient.

CASE REPORT

A 10-year-old female patient reported to Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Navi Mumbai, with a chief complaint of pain and swelling in lower right back region of the jaw since last 8 months. The patient was apparently well 8 months back until she noticed the swelling in the same region, which had suddenly increased to the present size. There was no history of trauma or any other disease affecting the jaw and other bones. Extraoral examination revealed mild swelling was present on the right side of the mandible (Fig. 1). Temperature of the overlying skin was normal and there was no sign of inflammation. An intraoral examination revealed a diffuse bony swelling from right retromolar region to left deciduous canine region, crossing the midline. There was bicortical expansion with vestibular obliteration. On palpation, swelling was firm to hard in consistency and tender.

Orthopantomogram (OPG) and lateral oblique view radiograph revealed a large multilocular radiolucent,

[1]Postgraduate Student, [2]Professor, [3]Professor and Head [4]Lecturer

[1-4]Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India

Corresponding Author: Rajshri U Gurav, Postgraduate Student, Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India
Phone: +919769828886, e-mail: rajshrigurav91@gmail.com

12. Verma RK, Kumar R, Bal A, Panda NK. Aneurysmal bone cyst of maxilla with ectopic molar tooth – a case report. Otolaryngol Pol 2013 Nov-Dec;67(6):302-307.
13. Levy WM, Miller AS, Bonakdarpour A, Aegerter E. Aneurysmal bone cyst secondary to other osseous lesions. Report of 57 cases. Am J Clin Pathol 1975 Jan;63(1):1-8.
14. Kransdorf MJ, Sweet DE Aneurysmal bone cyst: controversy, clinical presentation, imaging. Am J Roentgenol 1995 Mar;164(3):573-580.
15. Tillman BP, Dahlin DC, Lipscomb PR, Stewart JR. Aneurysmal bone cyst: an analysis of ninety-five cases. Mayo Clin Proc 1968 Jul;43(7):478-495.
16. Ruiter DJ, van Rijssel TG, van der Velde EA. Aneurysmal bone cysts. A clinicopathological study of 145 cases. Cancer 1977 May;39(5):2231-2239.
17. Steiner GC, Kantor EB. Ultrastructure of aneurysmal bone cyst. Cancer 1978 Dec;40(6):2967-2978.
18. Harnandez GA, Castro A, Castro G, Amador E. Aneurysmal bone cyst versus hemangioma of the mandible. Oral Surg Oral Med Oral Pathol 1993 Dec;76(6):790-796.
19. Sun ZJ, Zhao YF, Yang RL, Zwahlen RA. Aneurysmal bone cysts of the jaws: analysis of 17 cases. J Oral Maxillofac Surg 2010 Sep;68(9):2122-2128.
20. Behal SV. Evolution of an aneurysmal bone cyst: a case report. J Oral Sci 2011 Dec;53(4):529-532.

CASE REPORT

10.5005/jp-journals-10031-1145

Osteosarcoma of Mandible

[1]Shikha Satish Bhatt, [2]Shilpa Patel, [3]Jigna Pathak, [4]Niharika Swain

ABSTRACT

Osteosarcomas (OS) are malignant neoplasms of the bone that commonly affect the long bones with rare presentation in jaws. Osteosarcomas of jaws represent about 6 to 8% of all OS, with an incidence of approximately 1 in 1.5 million persons per year. Although the exact cause of OS is still unknown, defects in the retinoblastoma (RB) and p53 genes play an important role in the process. It is characterized histologically by anaplastic stroma with direct osteoid production. Here, we report a case of OS in a 30-year-old female, who came with a massive bony swelling in the right mandibular region.

Keywords: Osteoblastic variant, Osteosarcoma, Sunburst appearance.

How to cite this article: Bhatt SS, Patel S, Pathak J, Swain N. Osteosarcoma of Mandible. J Contemp Dent 2016;6(1):70-74.

Source of support: Nil

Conflict of interest: None

INTRODUCTION

The term "osteosarcoma", also known as osteogenic sarcoma (OS), refers to a heterogeneous group of primary malignant neoplasms affecting bone forming or mesenchymal tissue that is characterized by formation of osteoid tissue.[1] It occurs most commonly in long bones of extremities near metaphyseal growth plate. Osteogenic sarcoma of jaw is rare and represents only 6 to 8% of all OS. Jaw OS usually presents themselves in the 3rd and 4th decades of life, almost a decade after their presentation in long bone tumors with a slight predilection for the mandible.[2] The exact etiology is unknown. Three main factors generally may play an important role in their development—irradiation, preexisting benign bone disorders, and genetic predisposition. Biologically, OS of the jaw is considered to be less aggressive with a lower incidence of metastasis and hence better prognosis than that occurring in long bones.[3] Despite modern treatment protocols that combine chemotherapy, surgery, and sometimes radiotherapy, the 5-year survival rate for patients diagnosed with OS remains at 50 to 70%.[4,5] Here, we report a case of a 30-year-old female with mandibular bony swelling diagnosed as OS.

[1]Postgraduate Student, [2]Professor and Head, [3]Professor [4]Lecturer

[1-4]Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India

Corresponding Author: Shikha Satish Bhatt, Postgraduate Student, Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Navi Mumbai-410209, Maharashtra India, Phone: +9127436604, e-mail: shikh-abhatt24@gmail.com

CASE REPORT

A 30-year-old female reported to Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Navi Mumbai, with a chief complain of swelling in lower right back region since 8 months. Patient was asymptomatic 8 months ago but started experiencing a swelling with respect to lower right half of the face. Swelling was small initially. Patient visited a local dentist who extracted a tooth from lower right back region. Swelling had gradually increased to its present size. No history of trauma was noted. There was no history of any other disease affecting the jaw or other bones. Medical and family history were noncontributory.

On extraoral examination, a diffuse swelling was present on the right side of face, extending from the zygoma inferiorly 2 to 3 cm below the chin. Anteriorly, it extended from just beyond the midline (left side) to the right preauricular region (Fig. 1). Anteroposteriorly, it measured about 12 × 9 cm in size. Overlying skin was normal but tensed and not associated with any sinus or fistula. Borders of the swelling were poorly demarcated (Fig. 2). There was no evidence of extraoral draining sinus. Temperature of the overlying skin was normal. An intraoral examination revealed an ill-defined, diffuse, ulceroproliferative lesion showing bicortical expansion obliterating the buccal and lingual vestibular spaces on the right side of the mandible. The soft tissue growth showed indentations of the maxillary teeth. Mucosa over the swelling was inflamed (Fig. 3). Floor of the mouth was raised on the right side. On palpation, the swelling was tender and hard to firm in consistency. Grade II mobility was seen in all teeth from 33 to 47. Orthopantomograph (OPG) showed a diffuse radiopacity with a sunburst appearance on right side of the mandible extending from 45 to 48 region (Fig. 4). Periodontal widening of the lower anterior teeth along with two root pieces and two missing teeth was seen. Computed tomography (CT) scan showed bone forming malignant mass of 8 × 4 cm approximately arising from right hemimandible with sunburst periosteal reaction and not breaking the lingual border of the mandible. Externally, it extended up to subcutaneous tissue. Superiorly, the lesion extended up to the level of maxillary alveolus and inferiorly up to submandibular

3. Ong ST, Shim CK, Ng Kok-Han, Siar CH. Osteosarcoma presenting as an aggressive nodular mass in the region of the mandible. J Oral Sci 2004 Mar;46(1):55-59.
4. Marx ER, Stern D. Oral and Maxillofacial Pathology: a Rationale for Diagnosis and Treatment, volume 2. 2nd ed. Illinois: Quintessence Publishing Company; 2012. p. 859.
5. Bielack SS, Kempf-Bielack B, Delling G, Exner GU, Flege S, Helmke K, Kotz R, Salzer-Kuntschik M, Werner M, Winkelmann W, et al. Prognostic factors in high-grade osteosarcoma of the extremities or trunk: an analysis of 1,702 patients treated on neoadjuvant cooperative osteosarcoma study group protocols. J Clin Oncol 2002;20(3); 776-790.
6. Barnes L, Eveson JW, Reichart P, Sidransky D. World Health Organization classification of tumours, pathology and genetics of tumors of head and neck tumours. Lyon: IARC Press; 2005. p. 52.
7. Jordan RS. Oral Pathology: Clinical Pathologic Correlation. 6th ed. Chapter 14. Malignancies of jaw. Missouri: Elsevier. pp. 328-335.
8. Neville BW, Damm DD, Allen CM, Bouquot JE. Bone pathology In: Neville BW, Damm DD, Allen CM, Bouquot JE, editors. Oral and maxilofacial pathology. Philadelphia: Saunders; 2002. pp. 574-577.
9. Kundu ZS. Classification, imaging, biopsy and staging of osteosarcoma. Indian J Orthop 2014 May;48(3):238-246.
10. Rajendran R. Benign and Malignant Tumors of the Oral Cavity. In: Rajendran R, Sivapathasundaram B, editors. Shafer's Textbook of Oral Pathology. 6th ed. Amsterdam: Elsevier; 2009. pp. 169-173.
11. Picci Piero. Osteosarcoma (Osteogenic sarcoma). Orphanet J Rare Dis 2007;2:6.
12. Gnepp DR. Diagnostic surgical pathology of the Head and Neck. 2nd ed. Philadelphia: Elsevier; 2009. p. 667.
13. Garrington GE, Scofield HH, Cornyn J, Hooker SP. Osteosarcoma of the jaws. Analysis of 56 cases. Cancer 1967 Mar;20(3):377-391.
14. Paparella ML, Olvi LG, Brandizzi D, Keszler A, Santini-Araujo E, Cabrini RL. Osteosarcoma of the jaw: an analysis of a series of 74 cases. Histopathology 2013 Oct;63(4):551-557.
15. Forteza G, Colmenero B, Lopez-Barea F. Osteogenic sarcoma of maxilla and mandible. Oral Surg Oral Med Oral Pathol 1986 Aug;62(2):179-184.
16. Chaudhary M, Chaudhary SD. Osteosarcoma of jaws. J Oral Maxillofac Pathol 2012 May;16(2):233-238.
17. Pogrel MA. Inferior hemi-maxillectomy for treatment of palatal tumors. J Oral Maxillofac Surg 1988 Jan;46(1): 85-87.

JCD 31

10.5005/jp-journals-10031-1150

CASE REPORT

4 copies

Adenomatoid Odontogenic Cyst: A Rare Case Report

[1]Nikitha Narayanan, [2]Jigna Pathak, [3]Shilpa Patel, [4]Niharika Swain

ABSTRACT

Adenomatoid odontogenic cyst (AOC) is a benign, slow growing, relatively uncommon lesion of odontogenic origin. Histogenesis of AOC is still uncertain; however, it is often considered as a hamartomatous lesion rather than a true neoplasm. It is described as a cyst that has a hamartomatous intraluminal proliferation of epithelial cells derived from Hertwig's epithelial root sheath. It usually presents as an expansile lesion in maxillary anterior region. Adenomatoid odontogenic cyst is characterized histopathologically as well-demarcated cysts that typically appear with intraluminal masses. In the present paper, we report a rare case of AOC, thereby emphasizing the terminology and the histoarchitectural spectrum.

Keywords: Adenomatoid odontogenic cyst, Hamartoma, Hertwig's epithelial root sheath, Odontogenic.

How to cite this article: Narayanan N, Pathak J, Patel S, Swain N. Adenomatoid Odontogenic Cyst: A Rare Case Report. J Contemp Dent 2016;6(1):96-99.

Source of support: Nil

Conflict of interest: None

INTRODUCTION

Adenomatoid odontogenic cyst (AOC) is a benign, slow growing, relatively uncommon lesion of odontogenic origin.[1] Adenomatoid odontogenic cyst accounts for about 1 to 9% of all odontogenic lesions. It is best considered as a hamartomatous proliferation rather than neoplasm.[2] It is known to arise from Hertwig's epithelial root sheath as a hamartomatous intraluminal proliferation of epithelial cells.[3] The lesion is most frequently encountered in the second decade of life (68.6%) and 53.1% of cases occur within 13 to 19 years of age. It has a female predilection in almost 2:1 ratio.[1] The cyst presents as an expansile lesion most commonly in the anterior maxillary region.[3,4] The lesion is asymptomatic but may cause cortical expansion and displacement of the adjacent teeth. Adenomatoid odontogenic cyst is usually associated with an impacted tooth, most often involving unerupted permanent canine.[1,2]

[1]Postgraduate Student, [2]Professor, [3]Professor and Head [4]Lecturer

[1-4]Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India

Corresponding Author: Nikitha Narayanan, Postgraduate Student, Department of Oral Pathology and Microbiology MGM Dental College and Hospital, Navi Mumbai-410209, Maharashtra India, Phone: +01127436604, e-mail: nike.jn86@gmail.com

In the present paper, we report a case of AOC in anterior maxillary region, thereby emphasizing the terminology and the histoarchitectural spectrum.

CASE REPORT

A 19-year-old male patient presented at our institute with a firm, nontender swelling of right maxillary region, since 1 month (Fig. 1). The patient was under medication for epilepsy since past 10 years. The lesion presented as a diffuse swelling extending superoinferiorly from the infraorbital region to alae of right nasal cavity and anteroposteriorly till the zygomatic process. Intraorally, a diffuse swelling was observed extending from 11 to 15 region obliterating the buccal vestibule (Fig. 2). Orthopantomography (OPG) showed radiopacity in relation to maxillary sinus with over retained 53 and impacted 13. Computed tomography showed a well-defined radiolucency with spicules of radiopaque structure and the associated impacted tooth. Obliteration of maxillary

Fig. 1: Extraoral view shows diffuse swelling extending over the right maxillary region

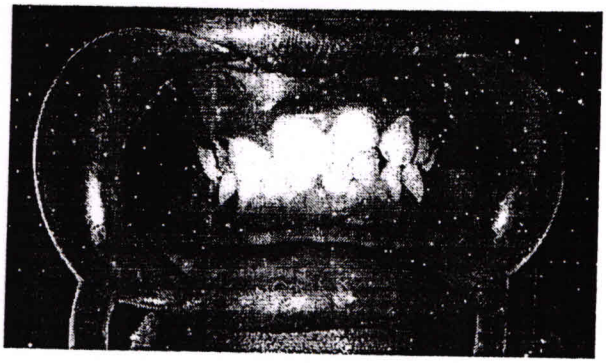


Fig. 2: Intraoral view shows diffuse swelling extending from 11 to 15, obliterating the buccal vestibule

Adenomatoid odontogenic cyst is considered to be a slowly growing benign lesion; hence, a conservative surgical enucleation or curettage is sufficient. The lesion is encapsulated by a thick connective tissue capsule that readily separates from its bony crypt, and hence recurrences are rare.[3] However, Xiang and Yan,[9] in their review of 16 cases, reported one case that recurred twice over a period of 20 years. Gadewar et al[6] in their review have also reported four cases of recurrences. Although the prognosis is considered excellent, regular follow-up is necessary.[2] Our case was treated with surgical enucleation with no recurrence reported in 2 years follow-up period.

CONCLUSION

Herein, the goal of this paper is to add on one more case of this rare entity to the literature. The rarity of AOC may be associated with its slowly growing pattern and benign behavior. Therefore, it should be always distinguished from more common lesions of odontogenic origin in routine dental examination. Also, uncommonness of such cases hinders any lasting conclusions regarding the lesions behavior. Thus, it necessitates a periodic review of these cases so as to understand the actual incidence, biological behavior, and outcome associated.

REFERENCES

1. Gupte S, Shetye A, Chhadva S, Malusare P. Adenomatoid odontogenic cyst of the mandible: report of a case. Int J Sci Res 2015;4(7):656-658.
2. Kumar R, Hashmi GS, Amjad SM. Adenomatoid odontogenic cyst of mandible: a rare case report. Int J Health Sci Res 2015;5(5):490-494.
3. Marx RE, Stern D. Oral and maxillofacial pathology: a rationale for diagnosis and treatment. Hanover Park: Quientessence Publishing; 2003. p. 609-612.
4. Regezi JA, Sciubba JJ, Jordan RCK, editors. Odontogenic tumours. Oral pathology clinical pathologic correlation, Fourth edition. St Louis, Missouri: Saunders; 1999. p. 276-277.
5. Harbitz F. On cystic tumours of the maxilla, and especially on adamantine cystadenomas (adamantomas). Dental Cosmos 1915;57:1081-1093.
6. Gadewar DR, Srikant N. Adenomatoid odontogenic tumour: tumour or a cyst, a histopathological support for the controversy. Int J Pediatr Otorhinolaryngol 2010 Apr;74(4):333-337.
7. Kurra S, Gunupati S, Prasad P R, Raju. Y S, Reddy BVR. An adenomatoid odontogenic cyst (aoc) with an assorted histoarchitecture: a unique entity. J Clin Diagn Res 2013 Jun;7(6):1232-1235.
8. Uppada UK, Salavadi R, Agarwal A, Paul D. Adenomatoid odontogenic cyst mimicking dentigerous cyst: a case report. J Cranio Max Dis 2015;4(1):90-94.
9. Xiang ZC, Yan G. Adenomatoid odontogenic tumour: a report of a rare case with recurrence. J Oral Pathol Med 2007 Aug;36(7): 440-443.

Vol. ■ No. ■ Month 2015

5 copies

Asymptomatic, blue, dome-shaped lesion on buccal mucosa

Janaki Iyer, MDS,[a] Jigna Pathak, MDS,[b] Shilpa Patel, MDS,[c] and Niharika Swain, MDS[d]
Mahatma Gandhi Mission's Dental College & Hospital, Maharashtra, India
(Oral Surg Oral Med Oral Pathol Oral Radiol 2015;■:1-5)

CLINICAL PRESENTATION

A 34-year-old female patient reported to a private dental practitioner with a complaint of a painless, slow-growing swelling on the right cheek region for the past 5 months. Extra-oral examination revealed no relevant abnormality. Intraoral examination disclosed a well-defined, dome-shaped, firm, nontender, noncompressible, bluish, sessile swelling measuring 1 cm in diameter, which did not blanch on pressure, on the right buccal mucosa adjacent to the maxillary right second molar. The overlying mucosa was smooth and intact (Figure 1). The patient was apparently healthy, with no signs of regional lymphadenopathy. Her medical and family histories were noncontributory.

DIFFERENTIAL DIAGNOSIS

Based on the clinical features, an array of asymptomatic, benign pathologic conditions affecting the buccal mucosa were included as the differential diagnoses. The signs and symptoms presented here could fall under pathologies of either a reactive, developmental lesion, benign neoplasm, or, rarely, a malignant variant.

Reactive lesions

Hematoma, a common pathosis of the oral cavity, is caused by a collection of extravasated blood in the connective tissue. It could present on traumatized buccal mucosa. Clinically, hematoma may appear as a painless, blue, nodular, smooth-surfaced swelling that does not blanch on pressure.

[a]Postgraduate Resident, Department of Oral and Maxillofacial Pathology, Mahatma Gandhi Mission's Dental College & Hospital, Maharashtra, India.
[b]Professor, Department of Oral and Maxillofacial Pathology, Mahatma Gandhi Mission's Dental College & Hospital, Maharashtra, India.
[c]Professor and Head, Department of Oral and Maxillofacial Pathology, Mahatma Gandhi Mission's Dental College & Hospital, Maharashtra, India.
[d]Lecturer, Department of Oral and Maxillofacial Pathology, Mahatma Gandhi Mission's Dental College & Hospital, Maharashtra, India.
Received for publication Mar 31, 2015; returned for revision Sep 12, 2015; accepted for publication Sep 20, 2015.

2212-4403/$ - see front matter
http://dx.doi.org/10.1016/j.oooo.2015.09.014

Developmental lesions

Pathoses such as blue nevi, varicosities and thrombi, vascular malformations, and hemangiomas were considered as differential diagnoses because of similar clinical presentations. Blue nevus, a common variant of oral melanotic nevus, could appear as a well-circumscribed, deep blue, nodular, slow-growing lesion. Buccal mucosa is the second common site of involvement, after the palatal region.[1] Oral varicosities appear as a bluish, purple painless macule or nodule, usually on the lingual mucosa at the elderly. However, buccal mucosa and lip show lower predilection for the same.[2] If the varix contains a thrombus, it may present as a firm, blue, noncompressible nodule. Buccal mucosa is a common site of thrombus presentation. Benign proliferation of endothelial lining of blood vessels may clinically present as a raised, bluish hemangioma of the oral mucosa. Hemangiomas are soft, dark red to blue, sessile or pedunculated lesions, with smooth or lobulated surfaces that blanch on compression. They present by the first month of life, show rapid growth until puberty, and generally involute at a later stage. Vascular malformations are similar structural anomalies but lack endothelial proliferation and generally present at birth; they are compressible and progress in size, without any involution.

Benign tumors of salivary gland origin

Benign neoplastic entities of salivary gland origin contributed to the differential diagnoses. Pleomorphic adenoma of the minor salivary glands, although most commonly affecting the junction of the hard and soft palates as a painless submucousal swelling, has been reported to involve the buccal mucosa (5.5% of cases).[3] Canalicular adenoma has been documented to show about 9.5% occurrence on the buccal mucosa, in some cases as bluish nodular swellings.[4] Pleomorphic adenoma shows an age predilection from the first to 10th decades, with a mean of 46 years,[5] whereas canalicular adenoma is reported to occur in age the range of 33-87 years, with a mean of 65 years.[4] Both these benign salivary gland tumors show higher occurrence in females.[1,5] Our presented case was a female patient of younger age than the expected mean ages of these aforementioned diseases.

SCO 5.4.0 DTD ■ OOOO1313_proof ■ 16 October 2015 ■ 5:41 pm ■ ce

16. Miura K, Ishimaru Y, Yoshimura T. Light and electron microscopic study of mucoepidermoid tumour of the clear cell. *Acta Pathol Jpn.* 1986;36:1419-1427.
17. Triantafillidou E, Dimitrakopoulos I, Skordalaki A. Clear cell carcinoma of minor salivary glands: report of a case. *Aust Dent J.* 1997;42:8-10.
18. Tinoco P, Pereira JCO, Filho RCL, et al. Mucoepidermoid carcinoma of minor salivary glands. *Intl Arch Otorhinolaryngol.* 2011;15:99-101.

Reprint requests:
Janaki Iyer, MDS (Post graduate resident)
Department of Oral and Maxillofacial Pathology
Mahatma Gandhi Mission's Dental College & Hospital
Junction of NH - 4 and Sion Panvel Expressway
Sector - 18, Kamothe
Navi Mumbai - 410 209, Maharashtra
India.
drjanakisiyer@gmail.com

CASE REPORT

Basaloid squamous cell carcinoma of the maxilla

Jigna Pathak,[1] Shilpa Patel,[1] Janaki Subramanian Iyer,[1] Neeta Mohanty[2]

[1]Department of Oral Pathology, MGM Dental College & Hospital, Navi Mumbai, Maharashtra, India
[2]Department of Oral Pathology and Microbiology, Institute of Dental Sciences, Siksha 'O' Anusandhan University, Bhubaneswar, Odisha, India, Institute of Dental Sciences, Bhubaneswar, Odisha, India

Correspondence to
Dr Neeta Mohanty,
dr.neetamohanty@gmail.com

Accepted 14 May 2015

SUMMARY

Basaloid squamous cell carcinoma (BSCC) is a distinctive aggressive variant of squamous cell carcinoma. We present a case of a 60-year-old man with tender swelling in the right cheek region for 6 months and continuous unilateral nasal discharge for 2 months. Extraoral examination revealed an ovoid, well-defined swelling from the right infraorbital rim to the angle of the mouth superoinferiorly and the right lateral wall of the nose to preauricular region anteroposteriorly. Intraorally, an ulceroproliferative growth from right upper gingivobuccal sulcus to mid palatine raphe with bicortical expansion was evident. CT revealed a hypodense mass obliterating the right maxillary sinus. Histopathology showed closely packed basaloid cells, with hyperchromatic palisading nuclei, arranged in a solid pattern with a lobular configuration. Prominent areas of comedo necrosis and keratin pearl formation were seen. These features suggested BSCC. The patient underwent surgical excision with adjuvant radiation but was lost to follow-up after 6 months of radiation therapy.

BACKGROUND

Basaloid squamous cell carcinoma (BSCC) is defined by WHO (2005) as an aggressive, high-grade variant of squamous cell carcinoma (SCC), and composed of basaloid as well as squamous components.[1] The basaloid variety of SCC is uncommon in the head and neck region, with an incidence of 2%,[2] and shows predilection to the upper aerodigestive tract.

To the best of our knowledge, the previous literature indicates BSCC of the nose and paranasal sinus as a relatively rare occurrence[3] with only 33 cases reported to date. We report a case of a 60-year-old man with maxillary sinus swelling and nasal discharge diagnosed as BSCC, highlighting the clinicopathological and therapeutic aspect of sinonasal tract BSCC.

CASE PRESENTATION

A 60-year-old man reported to the department of oral and maxillofacial surgery with pain and swelling in the right cheek for 6 months and continuous unilateral nasal discharge for 2 months. He had a 15-year history of tobacco consumption using mishri (powdered burnt tobacco leaves) about 6–7 times a day, and a 10-year history of bidi (a thin cigarette made of tobacco wrapped in a leaf) smoking about 4–5 times a day.

Extraoral clinical examination revealed an ovoid, well defined, palpable, firm swelling of 8×6 cm extending from the right infraorbital rim to the angle of the mouth superoinferiorly and to the right lateral wall of the nose to preauricular region anteroposteriorly (figure 1). Right-sided infraorbital nerve paraesthesia and deviated nasal septum were evident. On intraoral examination, an ulceroproliferative growth measuring 7×5 cm was seen, extending from the upper gingivobuccal sulcus up to the mid-palatine raphe mediolaterally and from 13 to 17 anteroposteriorly with evident bicortical expansion (figure 2). On palpation, the ulcer exhibited everted and rolled out margins with tenderness of the adjacent mucosa. The patient was systemically healthy with no signs of regional lymphadenopathy.

INVESTIGATIONS

CT scan imaging at the level of the zygomatic arch revealed a hypodense mass completely obliterating the right maxillary sinus with expansion of the buccal and palatal cortical plates, eroded lateral nasal wall and deviated nasal septum at a lower level (figure 3). Clinical and radiological features suggested an aggressive lesion suspicious of malignancy. An incisional biopsy was performed, after obtaining consent. Histopathologically, H&E-stained sections showed closely packed basaloid cells, with scanty cytoplasm and hyperchromatic nuclei, arranged in a solid pattern with a lobular configuration (figure 4A). Nuclear palisading along the periphery of the neoplastic nest was evident (figure 4B). Prominent areas of comedo type necrosis within the islands were evident (figure 4C). There was an abrupt association of these basaloid cells with foci of squamous differentiation showing keratin pearl

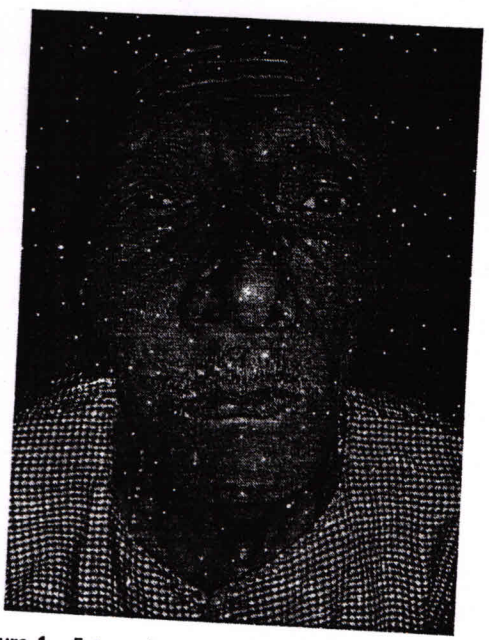


Figure 1 Extraoral view revealing an ovoid, well-defined swelling of right zygomatic region.

To cite: Pathak J, Patel S, Iyer JS, *et al*. *BMJ Case Rep* Published online: [*please include* Day Month Year] doi:10.1136/bcr-2014-209038

- Presence of abrupt foci of squamous differentiation with or without keratin pearls, and surface mucosal epithelium showing dysplastic features.

Sinonasal tract BSCC is uncommon and only limited studies have been reported by authors (as mentioned in table 1). In the previously reported cases, the nasal cavity and paranasal sinus were the most commonly affected sinonasal sites.

Nodal metastasis is not uncommon in BSCC. Winzenburg *et al*[8] showed significant differences in the survival of BSCC with and without lymph node metastases, with survival of 18.6 and 47.6 months, respectively, which was seconded by Ereno *et al* in 2008.[9] Regional and distant metastases have been reported as 64% and 44%, respectively, by Oikawa *et al*,[10] and 75% and 35–50%, respectively, of cases in the series by Winzenburg *et al*,[8] with 38% mortality and median survival of 17 months.[7] BSCC requires aggressive multimodality therapy, including radical surgical excision, neck dissection, radiotherapy and, often, chemotherapy. The aggressive biological behaviour of BSCC is well documented with 3-year and 5-year overall survival rates of 53% and 32%, respectively.[11]

Learning points

- Basaloid squamous cell carcinoma (BSCC) is an uncommon but distinct malignant tumour considered to be very aggressive, and it often presents as an advanced stage lesion with locoregional and distant metastases.
- Sino nasal BSCC, though rare, mimics BSCC of common mucosal sites of the upper aerodigestive tract, requiring equally aggressive intervention with multimodality therapy.
- Careful histological differentiation from other tumours with overlapping features holds significance for early accurate diagnosis, treatment planning and prognosis.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

1 Cardesa A, Zidar N, Ereno C. Basaloid squamous cell carcinoma. In: *World Health Organization classification of tumours*. Lyon, France: IARC Press, 2005:124–5.
2 Bhanja KR, Mallick SK, Sharma S. Cutaneous basaloid squamous cell carcinoma of the face, a rare variant: a case report. *Int J Case Rep Image* 2012;3:20–3.
3 Lee SJ, Ko IJ, Jun SY, *et al*. Basaloid squamous cell carcinoma in nasal cavity. *Clin Exp Otorhinolaryngol* 2009;2:207–10.
4 Ide F, Shimoyama T, Horie N, *et al*. Basaloid squamous cell carcinoma of the oral cavity: case report and review of 45 cases. *Oral Oncol* 2002:120–4.
5 Wain SL, Kier R, Volmer RT. Basaloid squamous cell carcinoma of the tongue, hypopharynx and larynx report of 10 cases. *Hum Pathol* 1986;17:1158–66.
6 Jacqueline AW, Thompson LDR, Wenig BM. Basaloid squamous cell carcinoma of the sinonasal tract. *Cancer* 1999;85:841–54.
7 Rachel JR, Kumar NS, Jain NK. Basaloid squamous cell carcinoma of retromolar trigone: a case report with review of literature. *J Oral Maxillofac Pathol* 2011;15:192–6.
8 Winzenburg SN, Niehans GA, George E, *et al*. Basaloid squamous cell carcinoma: a clinical comparison of two histological types with poorly differentiated squamous cell carcinoma. *Otolaryngol Head Neck Surg* 1998;119:471–5.
9 Cosme E, Ayman G, Maddi G, *et al*. Basaloid squamous cell carcinoma of the head and neck. *Head Neck Pathol* 2008;2:83–91.
10 Oikawa K, Tabuchi K, Nomura M. Basaloid squamous cell carcinoma of the maxillary sinus: a report of two cases of the head and neck. *AurisNasus Larynx* 2007;34:119–23.
11 Yu GY, Gao Y, Peng X, *et al*. A clinicopathologic study on basaloid squamous cell carcinoma in the oral and maxillofacial region. *Int J Oral Maxillofac Surg* 2008;37:1003–8.
12 Wan SK, Chan JKC, Tse KC. Basaloid squamous cell carcinoma of the nasal cavity. *J Laryngol Otol* 1992;106:370–1.
13 Wedenberge C, Jesslen P, Lundqvist G, *et al*. Basaloid squamous cell carcinoma of the maxilla. *Oral Oncol* 1997;33:141–4.
14 Paulino AFG, Singh B, Shah JP, *et al*. Basaloid squamous cell carcinoma of the head and neck. *Laryngoscope* 2000;110:1479–82.
15 Lu SY, Eng HL, Huang CC, *et al*. Basaloid squamous cell carcinoma of the sinonasal tract: report of two cases. *Otolaryngol Head Neck Surg* 2006;134:883–5.
16 Vasudev P, Tadross OB, Radhi J. Basaloid squamous cell carcinoma: two case reports. *Cases J* 2009;2:1–4.
17 Gu X, Eskandari F, Fowler M. Sphenoid sinus basaloid squamous cell carcinoma presenting as a sellar mass: report a case with review of the literature. *Head and Neck Pathol* 2011;5:81–5.
18 Stanculescu L, Vermesan O, Grintescu I, *et al*. A rare case of basaloid squamous cell carcinoma of the maxilla. *Rom J Morphol Embryol* 2012;53:1081–5.
19 Ishida M, Okabe H. Basaloid squamous cell carcinoma of the maxillary sinus: report of two cases in association with cathepsin K expression. *Oncol Lett* 2013;5:1755–9.

10.5005/jp-journals-10031-1120

CASE REPORT

4 copies

Subperiosteal Osteoid Osteoma: A Rare Entity

[1]Smita Vishwanath Rathod, [2]Shilpa Patel, [3]Jigna Pathak, [4]Niharika Swain

ABSTRACT

Osteoid osteoma (OO) is a relatively frequent benign tumor of the bone which has seldom been described in the jaws. It was originally described by Bergstand in 1930 and recognized as a distinct clinical entity by Jaffe in 1935. It is composed of osteoid and woven bone, and surrounded by a halo of reactive sclerotic bone, with an average size of the nidus less than 1.5 cm. The lesion occurs predominantly in children, adolescents and young adults. It accounts for 3% of all primary bone tumors, and about 10% of benign bone tumors. About 80% cases of OO occur in long bones while less than 1% occurs in head and neck region. The most common site is in the skull. When affecting the facial bones, they are frequently found in the mandible, the most common location being the posterior lingual surface and the mandibular angle area.

Herein, we report a rare case of OO of the mandible in a 16-year-old male patient with a brief literature review.

Keywords: Mandible, Nidus, Osteoid osteoma, Subperiosteal osteoid osteoma.

How to cite this article: Rathod SV, Patel S, Pathak J, Swain N. Subperiosteal Osteoid Osteoma: A Rare Entity. J Contemp Dent 2015;5(2):118-121.

Source of support: Nil

Conflict of interest: None

INTRODUCTION

Osteoid osteoma (OO) is a distinct benign entity. The etiology of this lesion is unknown. Jaffe and Lichtenstein have suggested that the lesion is a true neoplasm of osteo-blastic derivation,[1] but other workers have reported that the lesion occurs as a result of trauma or inflammation.[6] Jaffe refers to the OO as 'Sui Genris', denoting the lesion is small with self-limiting nature.[4] The lesion is typically less than 1.5 cm in diameter with distinct margins surrounded by a sclerotic zone of reactive new bone. The pain produced by a very small lesion is the most pathognomic and distingushing clinical feature of OO. This pain is initially dull, that may worsen at night and is relieved by nonsteroidal anti-inflammatory drugs (NSAIDS), mainly aspirin.[7] Occurrence of OO in head and neck region is very rare, Green et al reviewed the literature and reported the total number of cases of OO of the jaws to be seven, of these four have occurred in the mandible (three were in the body and one in the condyle) and three in the maxilia (one in the antrum).[6]

Here, we report an unusual case of OO in the mandible of a 16-year-old male patient with a brief literature review.

CASE REPORT

A 16-year-old male patient reported to department of oral pathology and microbiology, MGM Dental College and Hospital, with the chief complaint of pain and swelling in lower left back region of the oral cavity since last 6 months. Patient was apparently all right 6 months back after which he started experiencing pain in lower left back region. He noticed a small nodular swelling in the same region, which had gradually increased to the present size. Pain associated with the swelling was sharp, continuous and was relieved on taking analgesics and recurred after sometimes. There was no relevant medical and family history. On general physical examination, the patient was moderately built and nourished and all the vital signs were in the normal limits. No extraoral abnormality was detected. Intraoral examination revealed a round to oval solitary swelling on lingual cortical plate in relation to 34 and 35 region with obliteration of the lingual vestibule (Fig. 1). Swelling was approximately 1 cm in diameter. Surface of the swelling was eroded. Borders of the swelling were indistinct. On palpation, it was firm to hard in consistency, tender non-pulsatile, nonfluctuant, noncompressible and showed no evidence of discharge on digital pressure. For the presenting complaint, an orthopantomogram (OPG) was taken which showed no pathological changes in the bone except divergent roots of 34 and 35 (Fig. 2). An excisional biopsy was done from the lesional tissue in region of 34 and 35, and sent for histopathological analysis.

Histopathologically, hematoxylin and eosin (H&E) stained decalcified tissue section showed osteoid and woven bone in trabecular arrangement of irregular length, width and rimmed by osteoblasts (Fig. 3). Some of the trabecula showed mosaic or pagetoid pattern of reversal lines (Fig. 4). The connective tissue matrix

[1]Postgraduate Student, [2]Professor and Head
[3]Professor, [4]Lecturer

[1-4]Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India

Corresponding Author: Smita Vishwanath Rathod, Postgraduate Student, Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India
Phone: 02227436604, e-mail: smitarathod2012@gmail.com

of bone resorption are also usually evident. The overlying periosteum exhibits new bone formaton, and in this interstitial tissue collections of lymphocytes may be noted. Multinucleated giant calls are rarely seen. Our microscopic findings were in agreement with the literature.[6]

In the differential diagnosis, we considered fibro-osseous lesions and tumors of bony origin. One of the challenging issues with OO is differentiating the tumor from osteoblastoma (OB), because both are closely related entities. There are no specific histological criteria to distinguish OO from OB. Some authors believe that, in spite of the similarity in histopathologic features, they are distinguishable by clinical symptoms, size, site and radiological appearance. Ossifying fibroma has some similar clinical and radiographic features to OO, it is usually painless and lacks the nidus. Fibrous dysplasia is a poorly defined lesion showing characteristic chinese letter pattern histopathologically and being continuous with the host bone; neither of which were seen in this case. Paget's disease shows pathognomonic histologic feature of a mosaic pattern of bone formation but is mostly seen in older patients unlike in our case which showed the mosaic pattern in a young adult.

There are three main approaches for the treatment of OO, namely: conservative treatment, surgical excision and percutaneous excision. Osteoid osteoma is first treated conservative methods (NSAID) followed by surgical excision or curettage, but newly developed minimally invasive techniques, such as CT-guided percutaneous radiofrequency thermal ablation and laser photocoagulation, have become the methods of choice for the treatment. There is general agreement in the literature that the complete excision of an OO is the treatment of choice. Incomplete excision of the lesion leads to the recurrence of symptoms. Curettage of the cavity is not advised, because if it is not thoroughly performed the lesion is liable to recur. The tumor has no malignant potential. There is fairly good circumstantial evidence that spontaneous regression may occur in at least some untreated cases.[6] Our case was treated with surgical excision with no recurrence reported in a 3 years follow-up period.

CONCLUSION

Osteoid osteoma is a benign bone lesion that occurs very rarely in the jaw bones. Herein, we reported a case of subperiosteal OO that occurred in the mandible of a young adult. The lack of knowledge concerning the genesis of OO and its confusion with similar looking lesions in the bone make accurate compilation of data concerning the lesion a difficult task. It is mandatory to correlate the proper clinical, radiographic and histopathological examination to arrive at the correct diagnosis. It is obvious that the small number of reported cases of OO in jaw bone hinders any lasting conclusions concerning this lesion's behavior in the jaws. Thus, it necessitates an increased awareness among the dentists worldwide to report additional cases in literature.

REFERENCES

1. Golding JSR. The natural history of osteoid osteoma: with a report of twenty cases. J Bone Joint Surg 1954;36(2):218-229.
2. Rahsepar B, Nikgoo A, Fatemitabar SA. Osteoid osteoma of subcondylar region: case report and review of the literature. J Oral Maxillofac Surg 2009;67(4):888-893.
3. Kitsoulis P, Mantellos G, Vlychou M. Osteoid osteoma. Acta Orthop Belg 2006;72(2):119-125.
4. Walia C, Devi P, Thimmarasa VB, Jayadev S. Osteoid osteoma of the mandible: a rare entity. J Indian Acad Oral Med Radiol 2010 Jul-Sep;22(3):162-164.
5. Chaudhary M, Kulkarni M. Osteoid osteoma of mandible. J Oral Maxillofac Pathol 2007;11(2):52-55.
6. Shafer WG, Hine MK, Levy BM. A Textbook of Oral Pathology. 7th ed. Elsevier. p. 155-156.
7. Rashmishree K, Kruthika R, Venkatesh SG, Naikmasur G, Burde K. Osteoid osteoma of the mandible. E Journal of Dentistry 2014;4(1):565-570.
8. Ida M, Kurabayashi T, Takahashi Y, Takagi M, Sasaki T. Osteoid osteoma in the mandible. Dentomaxillofac Radiol 2002;31(3):385-387.
9. Jaffe HL. Osteoid osteoma. Arch Surg 1935;31(5):709-728.
10. Lee EH, Shafi M, Hui JH. Osteoid osteoma, a current review. J Pediatr Orthop 2006;26(5):965.
11. Singh A, Solomon MC. Osteoid osteoma of the mandible: a case report with review of the literature. J Dental Sciences (2012). Available at: http://dx.doi.org/10.1016/j.jds.2012.10.002.
12. Kayser, et al. Evidence of the subperiosteal origin of osteoid osteomas in tubular bones: analysis by CT and MR imaging. AJR: 170, March 1998.

CASE REPORT

Pagetoid polyostotic fibrous dysplasia

Rashmi Maruti Hosalkar,[1] Jigna Pathak,[1] Niharika Swain,[1] Neeta Mohanty[2]

[1]Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India
[2]Department of Oral Pathology and Microbiology, Institute of Dental Sciences Siksha O Anusandhan University, Bhubaneswar, Odisha, India

Correspondence to
Dr Neeta Mohanty,
dr.neetamohanty@gmail.com

Accepted 20 April 2015

SUMMARY

Fibrous dysplasia (FD) is a benign skeletal lesion occurring due to mutation of Gs α gene and involves one or multiple bones. We present a case of a 30-year-old female patient, with a 1-year history of swelling under her right eye that had gradually increased in size. Extraoral examination revealed a diffuse swelling extending anteroposteriorly from preauricular region to nasolabial fold, frontonasal region and superoinferiorly from zygoma to body of mandible, causing ipsilateral proptosis and contralateral deviation of nose. Intraoral examination showed obliteration of right upper and lower buccal vestibule. CT disclosed expansile lytic lesions involving multiple skull bones, jaws, sternum, rib and thoracic vertebrae. Histopathology displayed broad and interconnected trabeculae connected to the host bone exhibiting reversal lines resembling the mosaic pattern of Paget's disease. These features suggested pagetoid polyostotic FD. The patient underwent cosmetic recontouring and is under regular follow-up postoperatively.

BACKGROUND

Depending on the number of bone involved, fibrous dysplasia (FD) is generally classified as monostotic FD (MFD; single bone affected) and polyostotic FD (PFD; multiple bones affected). Although craniofacial bone involvement can occur in both types of FD, polyostotic variant is more prevalent.[1] The most affected bones in decreasing order for MFD are cranial bones, facial bones, rib, femur, tibia, humerus and those in PFD are femur, tibia, cranial bones, facial bones, pelvis, rib, upper extremities and clavicle. Only 1.4–5.5% of all FD lesions reported spinal involvement of which, lumbar spine is the most commonly affected followed by thoracic, sacral and cervical.[2 3] Based on the diverse histopathological features in correlation with the clinical occurrence and event of prenatal genetic mutation, FD can be classified into three types: Chinese letter pattern, pagetoid pattern and hypercellular pattern.[4]

To the best of our knowledge, on rigorous PubMed/MEDLINE search, the present case is the first of its kind to report occurrence of sternum, rib and cervical vertebra involvement in histopathological pagetoid variant of polyostotic FD.

To cite: Hosalkar RM, Pathak J, Swain N, *et al*. *BMJ Case Rep* Published online: [*please include* Day Month Year] doi:10.1136/bcr-2014-209149

CASE PRESENTATION

A 30-year-old woman presented with a 1-year history of swelling under her right eye that had gradually increased in size. On extraoral examination, a large, ill-defined swelling measuring approximately 8×6 cm was noted on the right side of face extending anteroposteriorly from preauricular region to nasolabial fold and frontonasal region and superoinferiorly from zygoma to body of mandible (figure 1), causing ipsilateral proptosis (figure 2) and contralateral deviation of nose. Functional deformity was absent. On intraoral examination, the swelling caused obliteration of the right maxillary and mandibular buccal vestibule in relation to 14–17 and 44–47 regions.

Figure 1 Extraoral view showing unilateral swelling involved the right side of face.

INVESTIGATIONS

Radiologically, the posteroanterior view and lateral view of skull revealed expansile lytic lesions involving right frontal, parietal, temporal, zygomatic, maxillary and mandibular bones. The lesions show ground glass attenuation in diploic spaces of calvarium; medulla could not be differentiated from the cortex (figures 3 and 4). CT of the head and neck revealed diffuse ill-defined expansile ground glass

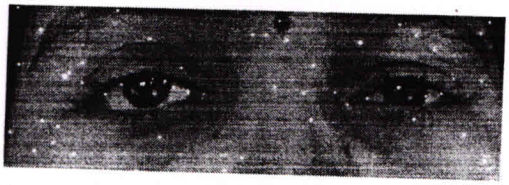


Figure 2 Extraoral view showing proptosis of right eye.

Cases of FD that do not present any functional distress, deformity or fractures should be monitored periodically. Surgical intervention should be considered if the lesion is encroaching on the adjacent structures and causing distress.[17] The present case was managed by cosmetic recontouring of facial bones. The patient is on routine follow-up since 1 year and has not reported any distress.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

1 Feller L, Wood NH, Khammissa R, *et al*. The nature of fibrous dysplasia. *Head Face Med* 2009;5:22.
2 Riddle ND, Bui MM. Nature of fibrous dysplasia. *Arch Pathol Lab Med* 2013;137:134–8.
3 Wu FL, Liu ZJ, Liu XG, *et al*. Polyostotic fibrous dysplasia involving the thoracic spine with myelopathy: case report and review of the literature. *Spine J* 2014;14:11–15.
4 Riminucci M, Liu B, Corsi A, *et al*. The histopathology of fibrous dysplasia of bone in patients with activating mutations of the Gs αgene: site-specific patterns and recurrent histological hallmarks. *J Pathol* 1999;187:249–58.
5 Naik MN, Tourani KL, Chandra Sekhar G, *et al*. Interpretation of computed tomography imaging of the eye and orbit. A systematic approach. *Indian J Ophthalmol* 2002;50:339–53.
6 Eversole R, Su L, ElMofty S. Benign fibro-osseous lesions of the craniofacial complex. A review. *Head Neck Pathol* 2008;2:177–202.
7 Neville BW, Damm DD, Allen CM, *et al*. Oral and maxillofacial pathology. *Chapter. 14, Bone pathology*. 2nd edn. Philadelphia: W.B. Saunders Company, 2002;563–4.
8 Lichtenstein L. Polyostotic fibrous dysplasia. *Arch Surg* 1938;36:874–98.
9 Kruse A, Pieles U, Riener MO, *et al*. Craniomaxillofacial fibrous dysplasia: a 10-year database 1996–2006. *Br J Oral Maxillofac Surg* 2009;47:302–5.
10 Bianco P, Riminucci M, Majolagbe A, *et al*. Mutations of the GNAS1 gene, stromal cell dysfunction, and osteomalacic changes in non–McCune–Albright fibrous dysplasia of bone. *J Bone Miner Res* 2000;15:120–8.
11 Weinstein LS. G(s) alpha mutations in fibrous dysplasia and McCune-Albright syndrome. *J Bone Miner Res* 2006;21:120–4.
12 Abdelkarim A, Green R, James Startzell S, *et al*. Craniofacial polyostotic fibrous dysplasia: a case report and review of the literature. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2008;106:49–55.
13 Leet AI, Magur E, Lee JS, *et al*. Fibrous dysplasia in the spine: prevalence of lesions and associations with scoliosis. *J Bone Joint Surg Am* 2004;86-A:531–7.
14 DiCaprio MR, Enneking WF. Fibrous dysplasia. Pathophysiology, evaluation, and treatment. *J Bone Joint Surg Am* 2005;87:1848–64.
15 Brown EW, Megerian CA, Mckenna MJ, *et al*. Fibrous dysplasia of temporal bone: imaging findings. *AJR* 1995;164:679–82.
16 Hanifi B, Samil KS, Yasar C, *et al*. Craniofacial fibrous dysplasia. *Clin Imaging* 2013;37:1109–15.
17 Menon S, Venkatswamy S, Ramu V, *et al*. Craniofacial fibrous dysplasia: surgery and literature review. *Ann Maxillofac Surg* 2013;3:66–71.

CASE REPORT

Malignant peripheral nerve sheath tumour (MPNST) of mandible: solving the perplexity.

Shilpa Patel,[1] Jigna Pathak,[1] Kamlesh Dekate,[2] Neeta Mohanty[3]

[1]Department of Oral Pathology, MGM Dental College & Hospital, Navi Mumbai, Maharashtra, India
[2]MGM Dental College & Hospital, Navi Mumbai, Maharashtra, India
[3]Department of Oral Pathology and Microbiology, Institute of Dental Sciences, Bhubaneswar, Odisha, India

Correspondence to
Dr Neeta Mohanty,
dr.neetamohanty@gmail.com

Accepted 9 January 2015

SUMMARY

We present an extremely rare case of malignant peripheral nerve sheath tumour (MPNST) in a 30-year-old woman without associated neurofibromatosis 1. The patient presented with an 8 cm×4 cm lesion extending from 46 to the retro molar region involving the ramus of the right mandible associated with regional paraesthesia. Incisional biopsy revealed spindle cells with vesicular nuclei arranged in fascicles leading to a diagnosis of spindle cell lesion. Posterior segmental mandibulectomy was performed under general anaesthesia. On excisional biopsy, a definitive diagnosis of low-grade MPNST was established on the basis of immunohistochemistry. The patient was then lost to follow-up.

BACKGROUND

To the best of our knowledge, only two cases of malignant peripheral nerve sheath tumour (MPNST) of the mandible without association of neurofibromatosis have been reported in the literature to date. The aim of this paper is to highlight, in depth, its various clinicopathological characteristics and the role of immunohistochemistry findings that differentiate MPNST from other commonly encountered spindle cell malignancies.

CASE PRESENTATION

A 30-year-old woman was referred to our department of oral and maxillofacial pathology with a tender swelling on the right side of the mandible of about 2 months' duration. The swelling had gradually increased in size since she first noticed it, and had abruptly increased postextraction of 47 1 month prior (figure 1). She presented no relevant family, medical or personal history.

The swelling on the right side of the mandible extended horizontally from first molar up to the sigmoid notch, and vertically to the inferior border of the mandible, pushing 48 inferiorly. Intraoral examination revealed a swelling from 46 to retro molar region. Mild cortical expansion was noticed (figure 2). The lesion was covered with normal-appearing mucosa. On palpation, the lesion was slightly tender, non-fluctuant and soft-to-firm in consistency. Paraesthesia of the same side was noted. The cervical lymph nodes were apparently normal. Incisional biopsy revealed spindle cells with vesicular nuclei arranged in fascicles leading to a diagnosis of spindle cell lesion.

Posterior segmental mandibulectomy was performed under general anaesthesia. Histopathological examination aided with immunohistochemistry of the resected specimen confirmed the diagnosis of low-grade MPNST. The patient was advised chemotherapy and radiotherapy; however, the patient was subsequently lost to follow-up.

INVESTIGATIONS

Radiographic analysis showed a well-defined radiolucency involving body and ramus of the right side of the mandible extending from 46 up to the sigmoid notch (figures 3 and 4).

Histopathological findings showed a partially encapsulated lesion having a fasciculated growth pattern with alternate hypocellular and hypercellular areas. Under higher magnification, fusiform or spindle-shaped cells were arranged in short fascicles in a haphazard fashion. These cells had scanty cytoplasm with hyperchromatic nuclei demonstrating mild-to-moderate pleomorphism and minimal mitotic activity. The tumour cells were also seen invading into the capsule and surrounding soft tissues (figures 5–7).

Figure 1 Extraoral view.

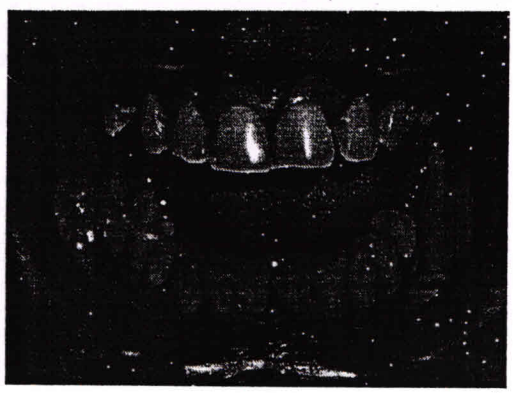


Figure 2 Intraoral view.

To cite: Patel S, Pathak J, Dekate K, *et al. BMJ Case Rep* Published online: [*please include* Day Month Year] doi:10.1136/bcr-2014-207790

Table 1 Summary of clinical data of patients with intraosseous (mandible) malignant peripheral nerve sheath tumour without neurofibromatosis

Author	Year	Age/sex	Site/radiological features	Treatment	Clinical outcome/follow-up period
Che *et al*[5]	2006	15/F	Anterior mandible/ill-defined radiolucent lesion	Wide excision	Not mentioned
Sham *et al*[4]	2009	18/F	Body and angle multilocular/irregular radiolucent of mandible lesion	Segmental mandibulectomy and tumour resection with wide margin Bilateral comprehensive functional neck dissection (type-III)	No recurrences/17 months

surrounding bone.[4 5 7] In the present case, the 30-year-old patient presented a solitary intraosseous MPNST as a well-defined radiolucent lesion in the body and ramus of the mandible.

A diagnosis of MPNST should be made on the basis of following widely accepted criteria, such as origin from a peripheral nerve, arising from a pre-existing benign nerve sheath tumour (neurofibroma) or the tumour displaying a constellation of histological features, including the dense and hypodense fascicles alternating in a marble-like pattern consisting of asymmetrically tapered spindled cells with irregular buckled nuclei. Other subtle and less-specific features such as nuclear palisading, increased perivascular cellularity and storiform or herringbone, curlicue growth patterns, may also be seen. Areas of geographic necrosis may be present. The most striking feature of MPNST is its degree of morphological heterogeneity, not only in terms of cellular pleomorphism and nuclear atypia but also in its abrupt transition of patterns, cellularity and grade. Certain histological variants such as epithelioid, glandular, rhabdomyosarcomatous (malignant triton tumour), pigmented and perineural have also been described in the literature but have little effect on prognosis.[1] Diagnosis of MPNSTs is usually based on histopathology aided by immunohistochemistry, which reflects the Schwann cell differentiation in this neoplasm. Approximately 50–90% of MPNSTs are positive for S-100 protein. Occasionally, they may also express glial fibrillary acidic protein, EMA and CK expression.[1 2 4 5] In the present case, histopathological features of the lesion, which included fasciculated growth pattern with alternate hypodense and hyperdense areas, and spindle cells with minimal cytological atypia, were seen infiltrating into the surrounding hard and soft tissue structures. These features were suggestive of spindle cell malignancy. The major challenge was to differentiate this neoplasm from other mimicking spindle cell malignancy, necessitating the aid of immunohistochemistry for a definitive diagnosis. The tumour cells showed immune reactivity for S-100, vimentin and Bcl-2, suggestive of neural origin. Immunonegativity of desmin and SMA ruled out the possibility of muscle-specific spindle cell malignancies. Non-reactivity to CD99, EMA, CK-7 and melanocytic markers eliminated other plausible neoplasms including synovial sarcoma and melanoma.

The treatment of MPNSTs of the jaws is wide surgical excision; however, local recurrence is common. The most common metastatic site for MPNST is the lung followed by bone and pleura. Regional lymph node metastasis is also not uncommon. Haematogenous metastasis occurs in at least half of cases.[1 7–11]

Prognosis of MPNST is generally poor. Prognostic variables for MPNST are the size of lesion, location, stage and grade, status of margins, necrosis and use of adjuvant radiation. Among these, the status of surgical margin and history of irradiation are independent negative prognostic factors.[1 5] Overall survival rate is 40–70%.[4]

Learning points

- Diagnosis of malignant peripheral nerve sheath tumours (MPNSTs) in the head and neck region is difficult as there are no standardised radiological and histological criteria demarcating them from other spindle cell neoplasms and this often poses a great challenge for pathologists.
- Definitive diagnosis of MPNSTs is usually based on histopathology aided by immunohistochemistry.
- Surgical margin status and history of irradiation are strong negative predictors for prognosis of MPNST.

Contributors SP contributed to the review of literature and design. SP, JP and KD participated in the preparation of the manuscript. NM and KD performed editing of the manuscript.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

1 Weiss SW, Goldblum JR. *Enzinger and Weiss's soft tissue tumors*. 5th edn. St. Louis: Mosby, 2008:903–41.
2 Hirose T, Hasegawa T, Kudo E, *et al*. MPNST an immuno-histochemical study in relation to ultrastructural features. *Hum Pathol* 1992;23(8):865–870.
3 Ellis GL, Abrams AM, Melrose RJ. Intraosseous benign neural sheath neoplasms of the jaws. Report of seven new cases and review of the literature. *Oral Surg Oral Med Oral Pathol* 1977;44:731–43.
4 Sham ME, Ghorpande, Shetty A, *et al*. Malignant peripheral nerve cell tumor. *J Maxillofac Oral Surg* 2009;9:68–71.
5 Che Z, Nam W, Park W-S, *et al*. Intraosseous nerve sheath tumors in the jaws. *Yonsei Med J* 2006;47:264–70.
6 Ferner RE, Huson SM, Thomas N, *et al*. Guidelines for the diagnosis and management of individuals with neurofibromatosis 1. *J Med Genet* 2007;44:81–8.
7 Bullock MJ, Bedard YC, Bell RS, *et al*. Intraosseous malignant peripheral nerve sheath tumor. Report of a case and review of the literature. *Arch Pathol Lab Med* 1995;119:367–70.
8 Polak M, Polak G, Brocheriou C, *et al*. Solitary neurofibroma of the mandible: case report and review of the literature. *J Oral Maxillofac Surg* 1989;47:65–8.
9 Fernandes AM, Johann ACBR, Da Silveira-Juniour JB, *et al*. Malignant peripheral nerve cell tumour of tongue. *Oral Oncology Extra* 2006;42:210–12.
10 Conley J, Janecka IP. Neurilemmoma of the head and neck. *Trans Sect Otolaryngol Am Acad Ophthalmol Otolaryngol* 1975;80:459–64.
11 Dorfman HD, Czerniak B. *Bone tumors*. St Louis: Mosby, 1998:839–41.

ORIGINAL ARTICLE

10.5005/jp-journals-10031-1125

41

Serum Albumin, Globulin and Albumin/Globulin Ratio in Oral Squamous Cell Carcinoma: A Prospective Study

4 copies

[1]Rashmi Maruti Hosalkar, [2]Shilpa Patel, [3]Jigna Pathak, [4]Niharika Swain, [5]Leela Poonja

ABSTRACT

Background: Detection of oral squamous cell carcinoma (OSCC) at an early stage would be a paramount for any successful clinical treatment, and thus better prognosis. Serum proteomics, a minimally invasive procedure being simple, safe and accessible, is one of the methods that are used for detection of various biomarkers that could be of diagnostic and prognostic importance for diseases including OSCC. The aim of this prospective study was to determine the role of serum albumin, globulin levels and albumin/globulin (A/G) ratio as a reliable diagnostic and prognostic biomarker in OSCC.

Materials and methods: The study was conducted on 30 clinically diagnosed OSCC patients and 10 normal healthy patients of the control group. Blood samples were collected from all patients under necessary precautions and processed further to obtain serum. Biopsies were obtained from OSCC patients and were histopathologically graded into well, moderate and poorly differentiated OSCC. Serum sample from both groups was evaluated and statistically analyzed for albumin, globulin and A/G ratio.

Results: Serum albumin, globulin and A/G ratio levels did not show any statistically significant increase in OSCC patients as compared to the control group. However, A/G ratio decreased with the advancement of disease.

Conclusion: The results obtained suggested that serum albumin, globulin and A/G ratio cannot be used as serum markers for diagnosis of OSCC. However, there have been studies suggesting significant increase in the levels, hence we emphasize on the need for more studies with larger sample sizes to be conducted for determining the role.

Keywords: Albumin/globulin ratio and serum proteomics, Albumin, Globulin, Oral squamous cell carcinoma.

How to cite this article: Hosalkar RM, Patel S, Pathak J, Swain N, Poonja L. Serum Albumin, Globulin and Albumin/Globulin Ratio in Oral Squamous Cell Carcinoma: A Prospective Study. J Contemp Dent 2015;5(3):149-152.

Source of support: Nil

Conflict of interest: None

[1]Postgraduate Student, [2]Head, [3,5]Professor, [4]Lecturer

[1-5]Department of Oral Pathology and Microbiology, MGM's Dental College and Hospital, Navi Mumbai, Maharashtra, India

Corresponding Author: Rashmi Maruti Hosalkar, Postgraduate Student, Department of Oral Pathology and Microbiology MGM's Dental College and Hospital, Navi Mumbai, Maharashtra India, Phone: 09029985467, e-mail: drrashmi009@gmail.com

INTRODUCTION

The term 'oral squamous cell carcinoma (OSCC)' or more frequently used term 'oral cancer' in general depicts any malignancy arising from the oral cavity. Oral cancer still poses a major health problem in many parts of the world and is also a leading cause of death among the most common cancers.[1]

In diseases including OSCC, due to continuous perfusion, blood proteomes show constant changes within. These changes could result in serum protein levels to over-express and/or abnormally shed, add, subtract and modify as the disease advances. Alterations in the blood proteomes could be implored through various methods of which serum-based proteomic pattern analysis is a minimally invasive procedure being simple, safe and accessible.[2]

Many researchers have studied the implications of various serum markers for OSCC including serum albumin, globulin and albumin/globulin A/G ratio.[3-5] In the present study, we evaluated the serum albumin, globulin levels and A/G ratio in different grades of OSCC to determine if they could be used as diagnostic or prognostic markers.

MATERIALS AND METHODS

The study was carried out in department of oral and maxillofacial pathology for a period of 1 year after obtaining ethical clearance from college and hospital ethical committee. The study was conducted on 30 clinically as well as histopathologically diagnosed OSCC cases comprising the diseased group and 10 normal healthy patients forming the control group all aged between 32 and 80 years. The exclusion criteria included previously treated cases of carcinoma or sarcomas, metastatic tumors to the jaw, recurrent OSCC and no other systemic diseases, such as renal, hepatic or muscle related, etc.

Five milliliters venous blood was collected from all patients under aseptic conditions. Blood was centrifuged for obtaining serum which was used then used for analysis. An informed consent was taken from patient prior to conducting the study. The serum obtained from the blood sample was analyzed for albumin, globulin levels and A/G ratio using colorimeter by biuret test. The serum levels were calculated and expressed in gm/dl.

Separate studies have evaluated the role of serum albumin, globulin and A/G ratio in OSCC and suggested that serum albumin levels constituting the major part of serum proteins have decreased significantly as the disease progressed, thus affecting the serum globulin levels by increasing it and in turn the A/G ratio. These results have led the authors to conclude that serum albumin and globulin can be used as diagnostic markers while serum A/G ratio could be a prognostic marker. The present study showed no significant changes in serum levels of albumin and globulin in different histopathological grades of OSCC. However, A/G ratio did show increase in MDOSCC. Nevertheless, we assume that this significant change observed was due to compensatory reaction of serum levels for each other as the disease progressed. Hence, we conclude that in present study there was no significant change in serum albumin, globulin levels and A/G and thus cannot be used as reliable markers in OSCC. However, limited sample size may have been a deterrent in establishing these serum levels as potential serum biomarkers.

REFERENCES

1. Elango JK, Gangadharan P, Sumithra S, Kuriakose MA. Trends of head and neck cancers in urban and rural India. Asian Pac J Cancer Prev 2006;7:108-112.
2. Petricoin EF, Zoon KC, Kohn EC, Barrett JC, Liotta LA. Clinical proteomics: translating benchside promise into bedside reality. Nat Rev Drug Discov 2002;1(9):684-695.
3. Nayyar AS, Khan M, Vijayalakshmi KR, Anitha M. Serum albumin implications in oral squamous cell carcinoma. Acad J Cancer Res 2011;4(2):56-60.
4. Silvia CR, Delphine W, Vasudevan DM, Sudhakar PK. Alteration of serum β_2 microglobulin in oral carcinoma. Ind J Clin Biochem 2002;17(2):104-107.
5. Joudi FS Al, Wahab NA. The utilization of an index for serum globulin compensation in diseases associated with decreased serum albumin. Med J Malaysia 2004;59(4):495-501.
6. Barnes L, Eveson JW, Reichart P, Sidransky D. World Health Organization classification of tumors. Pathology and genetics head and neck tumours. IARC Press. Lyon, 2005.
7. Royal College of Pathologists. Datasets and tissue pathways RCP. Available at: http://www.repath.org/index.asp? Page ID = 254.2009.
8. Jemal A, Bray F, Center MM, Ferlay J, Ward E, Forman D. Global cancer statistics. CA Cancer J Clin 2011;61(2):69-90.
9. Warnakulasuriya S. Global epidemiology of oral and oropharyngeal cancer. Oral Oncol 2009;45:309-316.
10. Murray RK, Granner DK, Rodwell VW. 2006. Harper's Illustrated. Biochemistry. 27th ed. McGraw-Hill Publications, USA p. 588-605.
11. Lawal AO, Kolude B, Adeyemi BF, Lawoyin JO, Akang EEU. Relationship between serum albumin and oral epithelial cancers in patients seen at a nigerian tertiary hospital. Afr J Biomed Res 2010;13(3):225-229.
12. Soriani M, Pietraforte D, Minetti M. Antioxidant potential of anaerobic human plasma: role of serum and thiols as scavengers of carbon radiclas. Arch Biochem Biophys 1994;312(1): 180-188.
13. Seaton K. Albumin concentration controls cancer. J Natl Med Assoc 2001;93(12):490-493.
14. Nayyar AS, Khan M, Vijayalakshmi KR, et al. Serum total protein, albumin and advanced oxidation protein products implications in oral squamous cell carcinoma. Malays J Pathol 2012;34(1):47-52.
15. Metgud R, Patel S. Serum and salivary levels of albumin as diagnostic tools for oral pre-malignancy and oral malignancy. Biotech Histochem 2014;89(1):8-13.
16. Shabana AHM. Expression of Beta-2 microglobulin by normal, benign and malignant oral epithelia. Saudi Dent J 1991;3(3): 92-98.
17. Du XJ, Tang LL, Mao YP, et al. The pretreatment albumin to globulin ratio has predictive value for long-term mortality in nasopharyngeal carcinoma. PLoS ONE 2014;9(4):e94473.
18. Suh B, Park S, Shin DW, et al. Low albumin to globulin ratio associated with cancer incidence and mortality in generally h althy adults. Ann Oncol 2014;25(11):2260-2266.
19. Joudi FS Al. Prognostic value of an index for serum globulin compensation in colon and breast cancers. Singapore Med J 2005;46(12):710.

ORIGINAL ARTICLE

10.5005/jp-journals-10031-1115

Comparative Evaluation of Micronuclei in Exfoliated Urothelial Cells in Patients with Smoking and Smokeless Tobacco-associated Lesions: A Prospective Quantitative Study

[1]Janaki Subramanian Iyer, [2]Jigna Pathak, [3]Shilpa Patel, [4]Leela Poonja, [5]Niharika Swain

ABSTRACT

Objective: To clinically evaluate various tobacco-associated lesions and to evaluate and compare the micronucleus (MN) assay in exfoliated urothelial cells in patients with smoking and smokeless tobacco-associated lesions.

Materials and methods: This study was conducted in the Department of Oral Pathology and Microbiology, Mahatma Gandhi Mission's Dental College and Hospital, Navi Mumbai, from October 2012 to June 2013. One hundred cases having tobacco habits (smoking or smokeless) and clinically detectable tobacco-associated lesions were included. Exfoliated urothelial cytosmears were prepared, stained with Papanicolaou and slides were scored for MN.

Results: All cases (n = 100) were found to have tobacco-associated lesions that were clinically detectable. Voided urine samples were collected from all cases (n = 100) who indulged in smoking and smokeless tobacco habit, with males (n = 71) and females (n = 29), whose ages ranged from 19 to 75 years. We observed that out of the 100 cases evaluated, 12 cases showed the presence of MN in the urine cytosmear. Of these 12 cases, n = 10 were bidi smokers and n = 2 were betel quid chewers. Owing to insufficient population of urothelial cells in the cytosmear, MN evaluation could not be statistically proved.

Conclusion: Although, MN score in the urothelial cells could not be statistically assessed, due to insufficient number of urothelial cells, our observations reveal that MN count seems to be increased in smokers than smokeless tobacco users. Thus, we urge the need for further studies to highlight the comparative evaluation of MN score between smokers and smokeless tobacco on urothelial cells.

Keywords: Cytosmear, Genotoxicity, Micronucleus, Tobacco, Urothelial.

How to cite this article: Iyer JS, Pathak J, Patel S, Poonja L, Swain N. Comparative Evaluation of Micronuclei in Exfoliated Urothelial Cells in Patients with Smoking and Smokeless Tobacco-associated Lesions: A Prospective Quantitative Study. J Contemp Dent 2015;5(2):93-97.

Source of support: Nil

Conflict of interest: None

[1]Postgraduate Student, [2,4]Professor
[3]Professor and Head, [5]Lecturer

[1-5]Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India

Corresponding Author: Janaki Subramanian Iyer, Postgraduate Student, Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India
Phone: 022226365378, e-mail: drjanakisiyer@gmail.com

INTRODUCTION

Oral cancer is the sixth most common cancer worldwide and the most common in India.[1] Approximately, 70,000 cases and over 48,000 oral cancer-related deaths occur yearly.[2] Tobacco usage is one of the prime factors for the escalating cancer rates. Epidemiological studies show that the risk of developing oral cancer is 5 to 9 times greater for tobacco consumers than non-consumers.[3] Tobacco is most commonly smoked as cigarettes, bidis, cigars and loose tobacco in pipes. Smokeless tobacco is usually placed in the oral and nasal cavities against the mucosal sites that permit the absorption of nicotine into the body. In India, tobacco is commercially available in bidi, cigarette, cigar smoking tobacco forms, or gutkha, mawa, mishri, admixed with arecanut and slaked lime—smokeless tobacco forms.[4,5]

Benzo[a]pyrene and other polycyclic aromatic carcinogens are the most important carcinogenic agents in smoke, while unburnt tobacco contains 28 carcinogens of which the most harmful are the tobacco specific nitrosamines (TSNAs). The metabolites of nitrosamines, such as carcinogenic TSNAs N'-nitrosonornicotine (NNN), 4-(N-methyl-N-nitrosamino)-1-(3-pyridyl)-1-butanone (NNK) and N-nitrosoanabasine as well as the volatile nitrosamines N-nitrosodimethylamine and N-nitrododiethylamine, have been detected in saliva of tobacco chewers.[4-6] Other cancer causing substances include formaldehyde, acetaldehyde, cortonaldehyde, hydrazine, arsenic, nickel, cadmium and benzopyrene.[6] Tobacco smoke contains more than 60 carcinogenic combustion products. In particular, NNN, NNK and polycyclic aromatic hydrocarbons have been linked with genotoxicity.[5] Nitrosamines are also produced by the alkaloids in arecanut which are also carcinogenic.[7] These agents are known to cause adverse effects at a cellular level, either directly at the site of placement or by indirect systemic effects. Various tobacco-associated clinically detectable oral lesions are tobacco keratosis, leukoplakia, oral lichen planus and oral submucous fibrosis. These mucosal changes are well documented to predispose carcinoma.[8]

8. Altini M, Coleman H, Doglioni C, Favia G, Maiorano E. Calretinin expression in ameloblastoma. Histopathol 2000;37(1):27-32.
9. Kumamoto H, Yoshida M, Ooya K. Immunohistochemical detection of amelogenin and cytokeratin 19 in epithelial odontogenic tumors. Oral Diseases 2001;7(3):171-176.
10. Stoll C, Stollenwerk C, Riediger D, Mittermayer C, Alfer J. Cytokeratin expression patterns for distinction of odontogenic keratocysts from dentigerous and radicular cysts. J Oral Pathol Med 2005;34(9):558-564.
11. Chu P, Weiss L. Keratin expression in human tissues and neoplasms. Histopathol 2002;40(1):403-439.
12. Chatterjee S. Cytokeratins in health and disease. JOMP 2012; 3(1):198-202.
13. Chaitanya Babu N, Dawra G, Sindura CS. Immunohistochemical evaluation of Bcl2 and Cytokeratin 14 and Cytokeratin 19 in ameloblastoma. IJCD 2010;1(1):36-39.
14. Bancroft J, Gamble M. Theory and practice of histological techniques. 6th ed. Churchill Livingstone Elsevier. p. 432-472.
15. Piattelli A, Lezzi G, Rubini C. Calretinin expression in odontogenic cysts. J Am Asso Endo 2003;29:6.
16. Shear M, Paul M. Speight, cyst of the oral and maxillofacial regions. 4th ed; 2006 Oct.
17. Kanth KS, Kumarl TD, Kumar AR. Immunohistochemical analysis of dentigerous cyst and ameloblastoma using cytokeratin 19 and 14, p53, p63 and ki-67. SRM J Res Dent Sci 2012 Oct-Dec;3(4):4.

CASE REPORT

10.5005/jp-journals-10031-1100

National, II, 2.5 43 4 4 copies

Odontogenic Myxoma of Maxilla

[1]Rashmi Maruti Hosalkar, [2]Shilpa Patel, [3]Jigna Pathak, [4]Niharika Swain

ABSTRACT

Odontogenic myxoma (OM) is a locally aggressive, benign, slow-growing tumor arising from mesenchyme and/or odontogenic epithelium. It is the third most common odontogenic tumor predominantly affecting females and posterior mandible. Radiographically, OM demonstrates unilocular or multilocular, well or ill-defined radiolucency showing 'honeycomb', 'soap-bubble' or 'tennis-racket' pattern with cortical expansion and tooth displacement. Histologically, OM shows loosely arranged stellate or spindle-shaped cells interspersed in myxoid matrix. Various treatment modalities, such as wide excision, enucleation and curettage, curettage with/without electrical or chemical cautery, enbloc resection and wide resection with/without immediate grafting can be considered based on the extent of lesion. Here, we present a case of a 17-year-old female, with OM in the left maxilla and the maxillary sinus.

Keywords: Odontogenic myxoma, Maxilla, Myxoid, Reticulin.

How to cite this article: Hosalkar RM, Patel S, Pathak J, Swain N. Odontogenic Myxoma of Maxilla. J Contemp Dent 2015;5(1):27-30.

Source of support: Nil

Conflict of interest: None

INTRODUCTION

In 2005, World Health Organization (WHO) has classified odontogenic myxoma (OM) as a tumor arising from mesenchyme and/or odontogenic ectomesenchyme with/without odontogenic epithelium.[1] It is believed to arise from primitive mesenchymal portion of tooth germ.[2] OM is a benign, slow growing but locally aggressive neoplasm affecting jaw bones that accounts for about 3 to 6% of all odontogenic tumors, affecting females more than males. It is the third most common odontogenic tumor. While OMs predominantly involve the mandible, maxillary tumors are said to be more aggressive. Majority of lesions that are without pain reach a large size and cause displacement of the teeth and asymmetry of the mandible or maxilla prior to discovery. Certain lesions spread with progressive pain through the maxillary sinus and nasal cavity, and severe cases result in exophthalmos, nasal obstruction and neurological disturbance.[1]

Here, we present a case of a 17-year-old female, with OM in the left maxilla and maxillary sinus.

CASE REPORT

A 17-year-old female reported to the department with complain of swelling on left upper region of the jaw since 3 months, which gradually increased to the current size. On extraoral clinical examination, a diffuse swelling was seen in left maxillary region obliterating the nasolabial fold (Fig. 1). Intraoral examination revealed a diffused, painless swelling present in 21 to 24 region, obliterating the buccal vestibule which appeared to be firm in consistency (Fig. 2). Radiological examination by computed tomography showed well defined, mixed radiopaque-radiolucent swelling causing bicortical expansion of left maxillary region involving the anterior wall of maxillary sinus and extending into maxillary antrum (Fig. 3). Orthopantomograph revealed ill-defined multilocular mixed radiopaque-radiolucent lesion with fine bony trabeculae within its interior structure (Fig. 4). Provisional diagnosis of ameloblastoma, ameloblastic fibroma, ameloblastic fibro-odontoma, odontogenic fibroma, odontogenic myxoma, central giant cell granuloma and fibro-osseous lesion was made based on clinicoradiographic features. The lesion was surgically excised under local anesthesia with informed consent. Macroscopically, the lesion appeared to be well defined, lobulated and with a gelatinous texture (Fig. 5). Microscopically, the cut sections demonstrated abundant loose myxoid stroma

Fig. 1: Extraoral photograph of the patient

[1]Postgraduate Student, [2]Professor and Head
[3]Professor, [4]Lecturer

[1-4]Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India

Corresponding Author: Rashmi Maruti Hosalkar, Postgraduate Student, Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India
Phone: 09029985467, e-mail: drrashmi009@gmail.com

'soap-bubble' or 'tennis-racket' pattern with cortical expansion and tooth displacement.[14] Zhang et al in their study described six different types on basis of the conventional radiographs as type I—unilocular; type II—multilocular (including honeycomb, soap bubble and tennis racket patterns); type III—involvement of local alveolar bone; type IV—features of type III and involvement of the maxillary sinus; type V—osteolytic destruction and type VI—a mix of osteolytic destruction and osteogenesis.[15] Our case shows type IV radiographic pattern which has been observed in 22% of the cases in previous study.[15]

Histologically, OM shows anastomosing processes of spindle, wedge or stellate-shaped cells lying loosely in an abundant background of acid mucopolysaccharide. Islands of inactive odontogenic epithelium may or may not be observed.[16] Sections stained with special stains, such as reticulin methods, showed that the extracellular matrix contained abundant acid and neutral mucopolysaccharides as well as reticulin fibers. Other stains that can be used are alcian blue and periodic acid schiff. The histological features were in accordance with the literature. A special stain, such as reticulin, was also positive. Histological differential diagnosis considered for the lesion was odontogenic fibroma, oral focal mucinosis. Odontogenic fibroma is more fibrous, is encapsulated and lacks the stellate-shaped cells typical of myxoma while oral focal mucinosis lacks reticulin fibers.

A number of treatment modalities can be considered for OM depending upon the extent of the lesion and includes wide excision, enucleation and curettage, curettage with/without electrical or chemical cautery, en bloc resection and wide resection with/without immediate grafting.[18] The lesion was surgically excised with patients consent in our case. The overall recurrence rate ranges from 10 to 33%.[11] Noffeke et al suggested that the higher recurrence rate was related to the tumor spillage during the operative procedure due to the gelationous consistency and poorly defined margins.[13] As the recurrence rate is high in OM, it is a must that the patient should be followed for 5 years to establish a diseased free state.[19] However, our patient was lost to follow-up postoperatively.

CONCLUSION

Histopathological diagnosis of OM becomes a necessity as it is very difficult for a diagnosis of OM to be made based on clinicoradiological parameters. At the same time, recurrence rate of OM is high and mandates a follow-up in all the cases. The present report attempts to throw a light for proper understanding and knowledge on OM.

REFERENCES

1. Barnes L, Eveson JW, Reichert P, et al. World Health Oragnisation Classification of Tumours. Pathology and Genetics of Head and Neck Tumours. Lyon: IARC Press; 2005. p. 1-430.
2. White DK, Chen SY, Mohnac AM and Miller AS. Odontogenic myxoma: a clinical and ultrastructural study. OOO 1975;39(6):901-917.
3. Virchow R. Die cellular pathologie in ihrer beegrundung auf physiologische and pathologische Gewebelehre. Berlin, Germany, Verlag von August Hirschwald 1871;563.
4. Stout AP. Myxoma, the tumor of primitive mesenchyme. Ann Surg 1948;127(4):706-719.
5. de Melo GM, et al. Myxoma of cervical soft tissue: case report and literature review. Int Arch Otorhinolaryngol 2008;12(4): 587-590.
6. Munjal M, et al. Odontogenic myxoma of the maxilla: a clinical case report and review of literature. Otolaryngology Online Journal 2013;3(2):ISSN:2250-0359a.
7. Chrcanovic BR, Figueiredo do Amaral MB, Marigo HA, Freire-Maia B. An expanded odontogenic myxoma in maxilla. Stomatologija, Baltic Dental and Maxillofac J 20[illegible] 12(4):122-128.
8. Mehendiratta M. The histological spectrum of myxoma, myxofibroma/fibromyxoma and odontogenic fibroma: a chicken and egg situation. IOSRJDMS 2012;1(1):3-5.
9. Taylor AM. New findings and controversies in odontogenic tumors. Med Oral Pathol Oral Cir Bucal 2008 Sep 1;13(9):E555-558.
10. Simon ENM, Merkx MAW, Vuhahula E, Ngassapa D, Stoelinga P. Odontogenic myxomas: a clinicopathological study of 33 cases. Int J Oral Maxillofac Surg 2004;33(4):333-337.
11. Reichart PA, Philipsen HP. Odontogenic tumors and allied lesions. Quintessence; Chapter 20. Odontogenic myxomas or myxofibroma 2004. p. 189-196.
12. Kaffe I, Naor H, Buchner A. Clinical and radiological features of odontogenic myxoma of the jaws. Dentomaxiliofac Radiol 1997;26(5):299-303.
13. Noffke CE, Raubenheimer EJ, Chabikuli NJ, Bouckaert MM. Odontogenic myxoma: review of the literature and report of 30 cases from South Africa. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007 July;104(1):101-109.
14. Barros RE, Dominguez FV, Cabrini RL. Myxoma of the jaws. Oral Surg Oral Med Oral Pathol 1969;27(2):225-235.
15. Zhang J, Wang H, He X, Niu Y, Li X. Radiographic examination of 41 cases of odontogenic myxomas on the basis of conventional radiographs. Dentomaxillofac Radiol 2007 Mar;36(3):160-167.
16. Rajendran R, Sivapathasundaram B. Shafer's Textbook of Oral Pathology. 6th ed. New Delhi: Elsevier; 2009. p. 148.
17. James L, Shetty A, Jaypal N, Okade D. Oral soft tissue myxomas. J Ind Aca Oral Med Radiol 2012;24(2):152-154.
18. Brannon RB. Central odontogenic fibroma, myxoma (odontogenic myxoma, fibro-myxoma) and central odontogenic granular cell tumor. Oral Maxillofac Surg Clin North Am 2004;16(3):359-374.
19. Thomas P, Suresh Babu G, Mishra C, Anusha RL, Shetty S. Odontogenic myxoma: a report of two cases with review of literature. JIAOMK 2011 Apr-June;23(2):143-146.

JCD

National, III 2.5

10.5005/jp-journals-10031-1101

CASE REPORT

44 3

Low Grade Central Mucoepidermoid Carcinoma

4 copies

[1]Janaki Subramanian Iyer, [2]Kartik Poonja, [3]Jigna Pathak, [4]Shilpa Patel, [5]Leela Poonja

ABSTRACT

Mucoepidermoid carcinoma (MEC) is the most common malignant salivary gland tumor, comprising about 15% of all salivary gland tumors and 30% of all salivary malignancies. Most of the MEC arise in the parotid gland. Rarely, it originates in the mandible and maxilla as an intraosseous variant, referred to as 'central mucoepidermoid carcinoma' or 'intraosseous mucoepidermoid carcinoma'. Central mucoepidermoid carcinomas (CMECs) are extremely rare, but well-known entity, comprising 2 to 3% of all MECs reported. Histopathologically, this malignant neoplasm is characterized by mucous, intermediate and epidermoid cells. In this report, we present a case of a male patient diagnosed as low grade CMEC.

Keywords: Central mucoepidermoid carcinoma, Intraosseous, Mandible, Low grade.

How to cite this article: Iyer JS, Poonja K, Pathak J, Patel S, Poonja L. Low Grade Central Mucoepidermoid Carcinoma. J Contemp Dent 2015;5(1):31-34.

Source of support: Nil

Conflict of interest: None

INTRODUCTION

Mucoepidermoid carcinoma (MEC) is the most common malignant salivary gland tumor, comprising about 15% of all salivary gland tumors and 30% of all salivary malignancies.[1] This entity was initially proposed by Masson and Berger in 1924.[2] Stewart et al, in 1945, described its mucus secreting and epidermal cellular elements, thus, establishing it as a distinct pathologic entity.[3] The involvement of parotid glands predominate, representing 45%.[4] Palate is the most frequently involved minor salivary gland site.[3] Mucoepidermoid carcinoma can also arise from ectopic salivary gland tissue in periparotid lymph nodes as well as in the larynx, lacrimal gland, nose, paranasal sinuses, lung and trachea. Rarely, it originates in the mandible and maxilla as an intraosseous variant, referred to as 'central mucoepidermoid carcinoma (CMEC)' or 'intraosseous mucoepidermoid carcinoma'.[3] Central mucoepidermoid carcinomas are extremely rare, but a well-known entity, comprising 2 to 3% of all MECs reported.[5] Lepp in 1939 first reported of a CMEC of the mandible in a 66-year-old woman, and Bhaskar (1963) analyzed the criteria for their central origin, histology and pathogenesis.[6] Waldron and Mustoe (1989) suggested that CMEC should be included in primary intraosseous carcinoma of jaws as type 4.[7]

Classification of primary intraosseous carcinomas (Waldron and Mustoe)[7] is as follows:

- *Type 1:* Primary intraosseous odontogenic carcinoma ex odontogenic cyst
- *Type 2A:* Malignant ameloblastoma
- *Type 2B:* Ameloblastic carcinoma
- *Type 3:* Primary intraosseous odontogenic carcinoma developed *de novo*
  a. Keratinizing type
  b. Nonkeratinizing type
- *Type 4:* Central intraosseous mucoepidermoid carcinoma

Mucoepidermoid carcinoma is defined by WHO (2005) as a malignant glandular epithelial neoplasm characterized by mucous, intermediate and epidermoid cells, with columnar, clear cell and oncocytoid features.[4] Previous literature indicates over 150 reported cases of CMEC. Here, we report a case of a 27-year-old male patient with a chief complaint of painful slow-growing swelling on left posterior aspect of lower jaw diagnosed as low grade CMEC.

CASE REPORT

Clinical and Radiographic Features

A 27-year-old male patient reported to the Department of Oral and Maxillofacial Surgery, Mahatma Gandhi Missions Dental College and Hospital, with a complaint of tender, slow-growing swelling on the left posterior region of lower jaw since 2 months. He gave history of dull, continuous pain related to fully erupted 38 which gradually increased over 2 months. He was advised extraction of 38 by a general dental physician. Patient noticed subsequent increase in pain postextraction, with no change in swelling. He gave no history of consumption of any deleterious substances. Extraoral clinical examination revealed a diffused, tender, nonfluctuant, noncompressible and nonreducible swelling measuring

[1]Postgraduate Student, [2]Lecturer, [3,5]Professor
[4]Professor and Head

[1,3-5]Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India

[2]Department of Oral and Maxillofacial Surgery, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India

Corresponding Author: Janaki Subramanian Iyer, Postgraduate Student, Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India
Phone: 9869040237, e-mail: drjanakisiyer@gmail.com

mucous cells population. Based on these findings, in the present case, a diagnosis of low grade central mucoepidermoid carcinoma was established.

Central mucoepidermoid carcinoma should be distinguished from various pathological entities, like residual cyst, chronic suppurative osteomyelitis, dentigerous cyst, odontogenic keratocyst, aneurysmal bone cyst, traumatic bone cyst, ameloblastoma, central giant cell granuloma and malignancies. Though there exist several systems to diagnose and stage this neoplasm, histopathological diagnosis remains the universally accepted golden standard.

Metastases are reported in 9% of CMEC, mainly to the regional lymph nodes. Nodal metastases have been described from 1 to 24 years after the time of initial management.[6] The widely practiced main treatment for CMEC is wide local excision with sectional or radical neck dissection in case of cervical involvement. Cervical nodal involvement was ruled out in our case. Documented recurrence rate for radical excision is 13%.[6] The present case underwent a posterior enblock resection with no neck dissection. The histopathological findings of the excisional specimen confirmed the diagnosis of low grade CMEC. Prognosis is difficult to evaluate due to the inadequacy of the staging and the disparity of treatment reported in the literature, whereas a well-differentiated, low-grade tumor without perineural invasion and with tumor-free margin indicates a good prognosis.[11] The CMEC that are graded in the intermediate to high grade range imply a worse prognosis than low-grade tumors. The presented case has been disease free for past 14 months.

REFERENCES

1. Azevedo RS, de Almeida OP, Kowalski LP, Pires FR. Comparative cytokeratin expression in the different cell types of salivary gland mucoepidermoid carcinoma. Head and Neck Pathol 2008;2(4):257-264.
2. Bansal A, Shetty DC, Rai HC, Singh HP. Primary intraosseous mucoepidermoid carcinoma of maxilla: a rare occurrence. E-Journal of Dentistry 2011;1(1):14-17.
3. Barnes L. Surgical pathology of the head and neck. 3rd ed. Informa healthcare. Diseases of Salivary Glands. p. 546-552.
4. Barnes L, Eveson J, Reichart P, Sidransky D. Pathology and genetics of head and neck tumours. WHO classification of tumours. Tumours of Salivary Glands. Lyon: IARC Press; 2005. p. 219-220.
5. Tucci R, Antonio LFM, de Carvalhosa AA, de Sousa Castro PH, Nunes FD, Santos Pinto DD Jr. Central mucoepidermoid carcinoma: report of a case with 11 years' evolution and peculiar macroscopical and clinical characteristics. Med Oral Pathol Oral Cir Bucal 2009 Jun 1;14(6):E283-E286.
6. Maremonti P, Califano L, Mangone GM, Zupi A, Guida C. Intraosseous mucoepidermoid carcinoma: report of a long-term evolution case. Oral Oncology 2001;37(1):110-113.
7. Waldron CA, Mustoe TA. Primary intraosseous carcinoma of mandible with probable origin in an odontogenic cyst. Oral Surg Oral Med Oral Pathol 1989;67(6):716-724.
8. Varma S, Shameena PM, Sudha S, Nair RG, Varghese IV. Clear cell variant of intraosseous mucoepidermoid carcinoma: report of a rare entity. J Oral Maxillofac Pathol 2012;16(1):141-144.
9. Alexander RW, Dupuis RH, Holton H. Central mucoepidermoid tumour (carcinoma) of the mandible. J Oral Surg 1974; 32(7):541-547.
10. Browand BC, Waldron CA. Central mucoepidermoid tumours of the jaws. Oral Surg Oral Med Oral Pathol 1975;40(5):631-643.
11. Li Y, Li LJ, Huang J, Han B, Pan J. Central malignant salivary gland tumours of the jaw: retrospective clinical analysis of 22 cases. J Oral Maxillofac Surg 2008;66(11):2247-2253.

Category I
4 copies

Oral Oncology 51 (2015) e26

Contents lists available at ScienceDirect

Oral Oncology

journal homepage: www.elsevier.com/locate/oraloncology

Letter to the editor

Comment on "Tumour thickness as a predictor of nodal metastases in oral cancer: Comparison between tongue and floor of mouth subsites" by Balasubramanian D et al.

Sir,

We would like to comment on the article "Tumour thickness as a predictor of nodal metastases in oral cancer: Comparison between tongue and floor of mouth subsites" by Balasubramanian D et al.

First of all, we appreciate the extensive single institution comparison study conducted by the authors on 343 cases of oral squamous cell carcinoma of two subsites i.e. tongue and floor of mouth of diverse prognostic importance. On reviewing previous literature of the OSCC of the same oral subsite and our institutional experience, we would like to further comment on certain issues as following:

(1) In present study, authors have chosen tumor thickness as the predictive parameter for nodal metastases. For tumor thickness, they have measured from the adjacent mucosa to level of deepest point of invasion. By definition, tumor thickness is defined as a direct micrometer measurement of vertical bulk of tumor regardless of histologic structure of an ulcerative or exophytic form of the tumor growth, whereas depth of invasion (DOI) means the extent of cancer growth into the tissue beneath an epithelial surface. In cases in which the epithelium is destroyed, some investigators reconstruct a surface line and measure from this line [1,2]. As stated by Moore et al., depth of invasion (DOI) and tumor thickness are not the same [3]. In this scenario, measurement of tumor thickness performed by Balasubramaniam D et al. does not seem to satisfy the aforementioned criteria.

(2) Authors have not mentioned about the selection criteria with regards to the nature or variants of OSCC sample. As in case of exophytic (verrucous squamous cell carcinoma) and endophytic lesions (ulcerative lesion), measurement of tumor thickness and DOI differs [2,4,5].

Minute variations in Cut-off values for both tumor thickness and DOI can influence the neck management (elective/selective neck dissection) and clinical outcome. So the need of hour is a definitive, reproducible and universally accepted distinct measurement technique for both.

Conflict of interest

None declared.

References

[1] Batsakis JG. The histology of oral cancer. In: Shah JP, Johnson NW, Batsakis JG, editors. Oral cancer. 1st ed. London: Martin Dunitz; 2003.

[2] Pentenero M, Gandolfo S, Carrozzo M. Importance of tumor thickness and depth of invasion in nodal involvement and prognosis of oral squamous cell carcinoma: a review of the [illegible] Head Neck [illegible]

[3] Moore C, [illegible] Thickness as prognostic aid in upper aerodigestive tract cancer. Arch Surg [illegible]

[4] Breslow A. Thickness, cross-sectional areas and depth of invasion in the prognosis of cutaneous melanoma. Ann Surg 1970;172:902–8. [illegible]

[5] Breslow A. Prognostic factors in the treatment of cutaneous melanoma. J Cutan Pathol [illegible]

Niharika Swain *

Shilpa Patel

Jigna Pathak

Department of Oral Pathology, Mahatma Gandhi Mission Dental College & Hospital, Navi Mumbai, India

* Corresponding author at: 304, Siddhivinayak Residency, Sector-1, Plot No.: 40, Kamothe, Navimumbai, Maharashtra, India. Tel.: +91 9324701036; fax: +91 22 27433185.

E-mail address: niharikadec30@gmail.com (N. Swain)

Available online 3 March 2015

http://dx.doi.org/10.1016/j.oraloncology.2015.[illegible]

ORIGINAL ARTICLE

10.5005/jp-journals-10031-1116

Immunohistochemical Evaluation of Calretinin and Cytokeratin-19 in Odontogenic Keratocyst and Ameloblastoma: A Retrospective Study

[1]Vipul Mohan Pawar, [2]Shilpa Patel, [3]Jigna Pathak, [4]Niharika Swain, [5]Rashmi Hosalkar, [6]Janaki Iyer

ABSTRACT

The odontogenic epithelial remnants, i.e. cell rests of Serre and Malassez, are formed from dental lamina and Hertwig's epithelial root sheath respectively, may proliferate and have role in pathogenesis of odontogenic cysts and tumors. Odontogenic keratocyst (OKC) is the most common and aggressive cyst of the dental lamina origin. Ameloblastoma, the second most common odontogenic tumor (OT), is a clinically benign and locally invasive polymorphic neoplasia. Differentiation of OKC from ameloblastoma sometimes poses a diagnostic dilemma, thus necessitating the need to differentiate between the two (especially unicystic ameloblastoma and OKC). Calretinin, a calcium binding protein, functions as a calcium buffer and a regulator of apoptosis. Some studies have shown its expression in parakeratinized OKC, unicystic and solid ameloblastoma, but not in other OTs. Calretinin may thus provide a better understanding of the biological behavior and tumorogenesis of ameloblastoma. Cytokeratin (CK)-19 is a type I cytokeratin, has been found to be a reliable marker of epithelial differentiation. The intense expression of CK-19 is useful for identification of odontogenic epithelial components, thus suggesting their potential for proliferation to form epithelial odontogenic cysts and tumors. The aim of this study is to evaluate calretinin and CK-19 in OKC and ameloblastoma. For this retrospective study, 20 formalin fixed paraffin embedded tissue samples of histopathologically proven OKC and ameloblastoma each, retrieved from the department of oral pathology was used. The results will be evaluated by using immunohistochemical analysis.

Keywords: Ameloblastoma calretinin, Cytokeratin, Immunohistochemistry, Orthokeratinized odontogenic cyst.

How to cite this article: Pawar VM, Patel S, Pathak J, Swain N, Hosalkar R, Iyer J Immunohistochemical Evaluation of Calretinin and Cytokeratin-19 in Odontogenic Keratocyst and Ameloblastoma: A Retrospective Study. J Contemp Dent 2015;5(2):98-103.

Source of support: Nil

Conflict of interest: None

INTRODUCTION

The dental lamina that forms the enamel organs as well as Hertwig's epithelial root sheath (HERS), which forms the roots of the teeth disintegrates and gives rise to odontogenic epithelial remnants called cell rests of Serre and cell rests of Malassez respectively. Although a vast majority of these epithelial residues persist throughout life in an inactive state, some of these are triggered by hitherto unknown mechanisms to proliferation and, thus, produce a variety of pathogenesis comprising of odontogenic cysts and tumors.[1]

Odontogenic keratocyst (OKC) is one of the common odontogenic cysts arising from the dental lamina. It may grow to a large size before it manifests clinically and it has a striking tendency to recur.[2] Various studies are performed to ascertain the aggressive nature of OKC, thus considering OKC as a benign cystic neoplasm.[3] Odontogenic tumors (OTs) are a group of lesions arising from the tooth producing apparatus or its remnants. They may originate from odontogenic epithelium and/or ectomesenchyme with varying degree of inductive tissue interaction.[4,5] Ameloblastoma is the second most common odontogenic neoplasm. World Health Organization (WHO) defines ameloblastoma as 'a locally invasive, polymorphic neoplasia that often has a follicular or plexiform pattern in a fibrous stroma'.[6] Ameloblastoma can also be classified into solid/multicystic (SMA) and unicystic ameloblastoma (UA). The biological behavior, aggressiveness, treatment and prognosis between SMA and UA varies, thus necessitating the need to differentiate between the two.

Differentiation of OKC from ameloblastoma sometimes poses a diagnostic dilemma, thus compelling the need to differentiate between the two (especially UA and OKC). Various immunohistochemical studies have been carried out in OKC and ameloblastomas. Of these markers, calretinin and cytokeratin (CK)-19 have proven to be important in differentiating and determining the proliferative potential of OKC and ameloblastoma. Calretinin, a calcium binding protein with a molecular weight of 29 kilodalton (kDa), is a member of EF-hand proteins. In humans, it is enclosed by CLAB-2 gene. The exact biological function of calretinin remains unknown, but possible roles as calcium buffer and/or calcium sensor and regulator of apoptosis have been postulated.[6,7] Intracellular Ca^{2+} ions are considered to be important second

[1,5,6]Postgraduate Student, [2]Professor and Head
[3]Professor, [4]Lecturer

[1-6]Department of Oral Pathology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India

Corresponding Author: Vipul Mohan Pawar, Postgraduate Student Department of Oral Pathology, MGM Dental College and Hospital Navi Mumbai, Maharashtra, India, Phone: 9822387600, e-mail: vipul242428@gmail.com

8. Altini M, Coleman H, Doglioni C, Favia G, Maiorano E. Calretinin expression in ameloblastoma. Histopathol 2000;37(1):27-32.
9. Kumamoto H, Yoshida M, Ooya K. Immunohistochemical detection of amelogenin and cytokeratin 19 in epithelial odontogenic tumors. Oral Diseases 2001;7(3):171-176.
10. Stoll C, Stollenwerk C, Riediger D, Mittermayer C, Alfer J. Cytokeratin expression patterns for distinction of odontogenic keratocysts from dentigerous and radicular cysts. J Oral Pathol Med 2005;34(9):558-564.
11. Chu P, Weiss L. Keratin expression in human tissues and neoplasms. Histopathol 2002;40(1):403-439.
12. Chatterjee S. Cytokeratins in health and disease. JOMP 2012; 3(1):198-202.
13. Chaitanya Babu N, Dawra G, Sindura CS. Immunohistochemical evaluation of Bcl2 and Cytokeratin 14 and Cytokeratin 19 in ameloblastoma. IJCD 2010;1(1):36-39.
14. Bancroft J, Gamble M. Theory and practice of histological techniques. 6th ed. Churchill Livingstone Elsevier. p. 432-472.
15. Piattelli A, Lezzi G, Rubini C. Calretinin expression in odontogenic cysts. J Am Asso Endo 2003;29:6.
16. Shear M, Paul M. Speight, cyst of the oral and maxillofacial regions. 4th ed; 2006 Oct.
17. Kanth KS, Kumarl TD, Kumar AR. Immunohistochemical analysis of dentigerous cyst and ameloblastoma using cytokeratin 19 and 14, p53, p63 and ki-67. SRM J Res Dent Sci 2012 Oct-Dec;3(4):4.

CASE REPORT

10.5005/jp-journals-10031-1093

Verruciform Xanthoma-Histopathologically: A Distinct Entity

[1]Vipul Mohan Pawar, [2]Shilpa Patel, [3]Jigna Pathak, [4]Niharika Swain

ABSTRACT

Verruciform xanthoma (VX) is an uncommon benign mucocutaneous lesion of unknown etiology. It is essential to diagnose this lesion as a varied entity of utmost importance as clinically their appearance could range from a simple leukoplakia or papilloma to as grave as squamous cell carcinoma SCC. Although this lesion is of multifactorial pathogenesis, a viral etiology like human papilloma virus (HPV) has been suggested in some cases. This rare, harmless lesion usually presents as a sessile or pedunculated, pale yellowish-to-red, papillary, granular or verrucous mucosal growth. Histopathologically, VX is characterized by the presence of parakeratinzed epithelium showing papillary or verrucous growth with thin rete ridges and connective tissue papillae extending up to the surface. The papillae characteristically consist of foam cells, also called xanthoma cells. We report two cases of VX of varied clinical appearance but very similar and characteristic histopathological presentation to be diagnosed as VX. The clinical diagnosis, though may be challenging; the histopathological features are diagnostic and well-defined. It is also noteworthy that in an improper biopsy, xanthoma cells may be scanty and their presence can be missed, especially if one is unfamiliar with the existence of this lesion.

Keywords: Verruciform xanthoma, Foam cells, Masticatory mucosa.

How to cite this article: Pawar VM, Patel S, Pathak J, Swain N. Verruciform Xanthoma-Histopathologically: A Distinct Entity. J Contemp Dent 2014;4(3):181-184.

Source of support: Nil

Conflict of interest: None

INTRODUCTION

Verruciform xanthoma (VX) is an uncommon benign, hyperplastic lesion primarily of the oral mucosa, first described by Shafer in 1971.[1] Verruciforms xanthomas are usually asymptomatic, solitary and slow-growing lesions. Verruciforms xanthoma appears as a well-defined, sessile growth having smooth margins with papillary, granular or verrucous appearance.[2,3] Males are affected slightly more than females and the most frequently affected intraoral sites are gingiva, alveolus and hard palate.[2-4] The lesion probably represents an unusual reaction or immune response to localized epithelial trauma or damage. The hypothesis of an unusual reaction or immune response to localized epithelial trauma is supported by cases of VX that have developed in association with disturbed epithelium like lichen planus, lupus erythematosus, epidermolysis bullosa, epithelial dysplasia, pemphigus vulgaris, warty dyskeratoma, graft-vs host disease.[5] Clinically, it is seen as a well demarcated, soft, painless, sessile, elevated mass with papillary or roughened surface with an average size of 2 cm.[4] Histopathologically, foam cells are the characteristic of the lesion along with hyperplastic parakeratinzed squamous epithelium with elongated rete ridges of relatively uniform depth.[3,4]

Here, we report two cases of VX of varied clinical appearance but very similar and characteristic histopathological presentation.

CASE REPORTS

Case I

A 30-year-old male patient complained of gingival growth in his upper front region of the jaw since 2 to 3 months. Patient was apparently normal 2 to 3 months back after which he developed a painless small triangular gingival growth on the labial aspect of interdental papilla of 11 and 21 which had gradually increased to the present size. There was no pain, bleeding or trauma associated with the gingival growth. No aggravating or relieving factors were associated with the growth. Root canal treatment was done with 11 and 21 and porcelain fused metal bridge given from 14 to 22, 4 to 5 years back. All vital signs were within the normal range. Medical history was noncontributory. Intraorally, on inspection a localized gingival enlargement was present in the interdental papilla of 11 and 21 region approximately 1 × 2 cm in size and reddish-pink in color (Fig. 1). The localized gingival enlargement was well-defined, sessile having smooth margins and rough pebbly surface. On palpation, the enlargement was nontender, soft to firm in

[1]Postgraduate Student (3rd year), [2]Professor and Head Professor, [3]Lecturer

[1-4]Department of Oral Pathology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India

Corresponding Author: Vipul Mohan Pawar, Postgraduate Student (3rd year), Department of Oral Pathology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India, Phone: 09822387600, e-mail: vipul242428@gmail.com

The differential diagnosis of VX includes: squamous papilloma, verruca vulgaris, condyloma acuminatum, verrucous carcinoma (VC) and squamous cell carcinoma (SCC). The presence of foamy macrophages, distinctive papillary epithelial proliferation, brightly eosinophilic parakeratin with keratin squames and neutrophilic infiltration are the characteristic features of VX that would help it in distinguishing from the above mentioned lesions.[10] Histologically, the differential diagnosis from VC is clearly an important one. The marked acanthosis with minimal or no atypia, and the presence of keratin-filled crypts are among the shared features. Squamous papilloma does not contain lipid-laden macrophages (foam cells) like VX, thus can be differentiated histologically. Xanthoma cells are also not a feature of either verruca vulgaris or condyloma acuminatum. The vacuolation of epithelial cells in the upper epidermis that is prominent in verruca vulgaris and condyloma acuminatum, is either absent or inconspicuous in VX. Presence of invasive epithelial proliferation, parakeratin plugging, pushing border and the lack of foamy histiocytic infiltrate would help in distinguishing VC from VX. Absence of cellular architectural atypia and breach in basement membrane as in SCC, exclude the possibility of VX.[3]

Surgical excision is treatment of choice for VX. The prognosis for VX is excellent and recurrence is extremely rare.

CONCLUSION

We report two cases of VX of varied clinical appearance but very similar and characteristic histopathological presentation. The clinical diagnosis, though may be challenging; the histopathological features are diagnostic and well-defined. It is also noteworthy that in an improper biopsy, xanthoma cells may be scanty and their presence can be missed, especially if one is unfamiliar with the existence of this lesion.

REFERENCES

1. Shafer WG. Verruciform xanthoma. Oral Surg 1971 Jun;31(6):784-789.
2. Santa Cruz DJ, Martin SA. Verruciform xanthoma of the vulva: report of two cases. Am J Clin Pathol 1979;71:224-228.
3. Philipsen HP, Reichart PA, Takata T, Ogawa I. Verruciform xanthoma—biological profile of 282 oral lesions based on a literature survey with nine new cases from Japan. Oral Oncol 2003 Jun;39(4):325-336.
4. Polonowita AD, Firth NA, Rich AM. Verruciform xanthoma and concomitant lichen planus of the oral mucosa. A report of three cases. Int J Oral Maxillofac Surg 1999 Feb;28(1):62-66.
5. Iamaroon A, Vickers RA. Characterization of verruciform xanthoma by in situ hybridization and immunohistochemistry. J Oral Pathol Med 1996 Aug;25(7):395-400.
6. Palestine RF, Winkelmann RK. Verruciform xanthoma in an epithelial nevus. Arch Dermatol 1982 Sep;118(9):686-691.
7. Yu CH, Tsai TC, Wang JT, Liu BY, Wang YP, Sun A, Chiang CP. Oral verruciform xanthoma: a clinicopathologic study of 15 cases. J Formos Med Assoc 2007 Feb;106(2):141-147.
8. Matakeyame M, Alonso Juliana MS, Marinaldo G, Brandao Adriana AH, Cavalcante Ana SR. Verruciform xanthoma located in anterior gingival. J Clin Exp Dent 2010;2(2):82-84.
9. Regezi, Scuibba. Oral pathology: clinical pathological correlation. 1st ed. p. 180-182.
10. Farahani SS, Treister NS. Oral verruciform xanthoma associated with chronic graft-versus- host disease: a report of five cases and a review of the literature. Head Neck Pathol 2011;5:193-198.

Tumor Biol. (2014) 35:8407–8413
DOI 10.1007/s13277-014-2266-5

REVIEW

Podoplanin—a novel marker in oral carcinogenesis

Niharika Swain · Shwetha V. Kumar · Samapika Routray · Jigna Pathak · Shilpa Patel

Received: 19 April 2014 / Accepted: 19 June 2014 / Published online: 27 June 2014

Abstract Podoplanin, a transmembrane sialoglycoprotein, is a specific marker for lymphatic endothelial cells which in recent years has gained prominent notoriety for its role in tumor progression and metastasis. It is an extensively studied biomarker for predictive assessment of malignant transformation as well as biologic behavior in both human precancer and cancer, respectively. This review summarizes the association of podoplanin overexpression in oral potentially malignant disorders and oral cancer with special emphasis on its putative role in carcinogenesis as well as its prospective use in targeted therapy.

Keywords Podoplanin · Biomarker · Carcinogenesis · Oral potentially malignant disorder · Oral cancer · Epithelial mesenchymal transition

Introduction

Podoplanin, though initially detected in murine osteosarcoma cell lines and lymphatic endothelial cells (LECs), was so named due to its peculiar expression in podocytes or foot processes of renal corpuscles and its potential influence in maintaining the unique shape of podocytes [1]. Subsequently, several PDPN like homologous proteins such as, the oncofetal antigen M2A recognized by the D2-40 antibody and the type I alveolar cell marker hT1α-2 and gp36, a sialoglycoprotein of vascular endothelium and alveolar epithelium, were indentified in human body [2].

PDPN primarily belongs to the family of type 1 transmembrane sialomucin-like glycoproteins. It consists of 162 amino acids, with an extracellular domain rich in serine and threonine residues, a single hydrophobic membrane spanning domain, and a short cytoplasmic endodomain. The extracellular domain is composed of repetitive mucin sequences with extensive O-glycosylated Ser and Thr residues ensuing in an extended rigid structure and a net negative charge. The extremely short cytoplasmic domain composed of only nine amino acids having a functional motif, i.e., Ser (Ser 167), which is a potential cAMP-dependent protein kinase (PKA) and protein kinase C (PKC) phosphorylation site and a cluster of highly conserved basic amino acids, recruiting site for proteins of the ezrin, radixin, and moesin (ERM) family [3, 4].

Apart from being a prominent lymphatic endothelial cell marker, PDPN has been reported to have a wide cellular distribution such as osteocytes, osteoblasts [5], odontoblasts [6], enamel epithelia, mesothelial cells [7], epidermal basal layer cells [8], salivary myoepithelial cells [9], choroid plexus epithelial cells [10], thymus type 1 epithelial cells [11], prostate myofibroblasts [12], follicular dendritic cells [13], immature cells like fetal germ cells, and developing Sertoli cells [2, 14]. Physiologically, PDPN seems to have multifaceted role in embryogenic development and growth of various organs like lymphatic system, lungs, and heart in humans. In vasculogenesis, PDPN has a crucial role in the separation of lymphatic from the blood circulatory system. The interaction of platelet and PDPN on lymphatic endothelial cells induces platelet aggregation and prevents blood from flowing into new lymphatic vessels budding from the cardinal vein. Furthermore, continued expression of PDPN into adulthood reinforces its importance in maintaining proper lymphatic architecture. In embryogenesis of the heart and lungs, the tissue-specific intrinsic role of PDPN in cell differentiation and

N. Swain (✉) · S. V. Kumar · J. Pathak · S. Patel
Department of Oral Pathology, MGM Dental College and Hospital, 304, Siddhi Vinayak Residency Sector 9, Plot no. 40 Kamothe, Navi Mumbai 410209, Maharashtra, India
e-mail: niharikadec30@gmail.com

S. Routray
Department of Oral Pathology, Institute of Dental Sciences, Siksha 'O' Anusandhan University, Bhubaneswar, Odisha, India

fibroblasts as a favorable prognostic marker in patients with colorectal carcinoma. Oncology. 2009;77:53–62.
41. Astarita JL, Acton SE, Turley SJ. Podoplanin: emerging functions in development, the immune system, and cancer. Front Immunol. 2012;3:283. doi:10.3389/fimmu.2012.00283.
42. Kawaguchi H, El-Naggar AK, Papadimitrakopoulou V, Ren H, Fan YH, Feng L, et al. Podoplanin: a novel marker for oral cancer risk in patients with oral premalignancy. J Clin Oncol. 2008;26:354–60.
43. Zhang G, Guo ZL, Gao Y. Podoplanin expression in oral squamous cell carcinoma and leukoplakia and its correlation with lymph vessels density. Zhonghua Kou Qiang Yi Xue Za Zhi. 2009;44:488–91.
44. de Vicente JC, Rodrigo JP, Rodriguez-Santamarta T, Lequerica-Fernández P, Allonca E, García-Pedrero JM. Podoplanin expression in oral leukoplakia: tumorigenic role. Oral Oncol. 2013;49:598–603.
45. Kreppel M, Kreppel B, Drebber U, Wedemayer I, Rothamel D, Zöller JE, et al. Podoplanin expression in oral leukoplakia: prognostic value and clinicopathological implications. Oral Dis. 2012;18:692–9.
46. Feng JQ, Mi JG, Wu L, Ma LW, Shi LJ, Yang X, et al. Expression of podoplanin and ABCG2 in oral erythroplakia correlate with oral cancer development. Oral Oncol. 2012;48:848–52.
47. Funayama A, Cheng J, Maruyama S, Yamazaki M, Kobayashi T, Syafriadi M, et al. Enhanced expression of podoplanin in oral carcinomas in situ and squamous cell carcinomas. Pathobiology. 2011;78:171–80.
48. Shi P, Liu W, Zhou ZT, He QB, Jiang WW. Podoplanin and ABCG2: malignant transformation risk markers for oral lichen planus. Cancer Epidemiol Biomarkers Prev. 2010;19:844–9.
49. Shintamura Y, Abe T, Nakahira M, Yoda T, Murata S, Sugasawa M. Immunohistochemical analysis of oral dysplasia: diagnostic assessment by fascin and podoplanin expression. Acta Histochem Cytochem. 2011;44:239–45.
50. Martin-Villar E, Scholl FG, Gamallo C, Yurrita MM, Muñoz-Guerra M, Cruces J, et al. Characterization of human PA2.26 antigen (T1alpha-2, podoplanin), a small membrane mucin induced in oral squamous cell carcinomas. Int J Cancer. 2005;113(6):899–910.
51. Yuan P, Temam S, El-Naggar A, Zhou X, Liu DD, Lee JJ, et al. Overexpression of podoplanin in oral cancer and its association with poor clinical outcome. Cancer. 2006;107(3):563–9.
52. Kreppel M, Scheer M, Drebber U, Ritter L, Zöller JE. Impact of podoplanin expression in oral squamous cell carcinoma: clinical and histopathologic correlations. Virchows Arch. 2010;456(5):473–82.
53. dos Santos AA, Oliveira DT, Pereira MC, Faustino SE, Nonogaki S, Carvalho AL, et al. Podoplanin and VEGF-C immunoexpression in oral squamous cell carcinomas: prognostic significance. Anticancer Res. 2013;33(9):3969–76.
54. Zhao D, Pan J, Li XQ, Wang XY, Tang C, Xuan M. Intratumoral lymphangiogenesis in oral squamous cell carcinoma and its clinicopathological significance. J Oral Pathol Med. 2008;37(10):616–25.
55. de Sousa SF, Gleber-Netto FO, de Oliveira-Neto HH, Batista AC, Nogueira Guimarães Abreu MH, de Aguiar MC. Lymphangiogenesis and podoplanin expression in oral squamous cell carcinoma and the associated lymph nodes. Appl Immunohistochem Mol Morphol. 2012;20(6):588–94.
56. Muñoz-Guerra MF, Marazuela EG, Martín-Villar E, Quintanilla M, Gamallo C. Prognostic significance of intratumoral lymphangiogenesis in squamous cell carcinoma of the oral cavity. Cancer. 2004;100(3):553–60.
57. Kyzas PA, Geleff S, Batistatou A, Agnantis NJ, Stefanou D. Evidence for lymphangiogenesis and its prognostic implications in head and neck squamous cell carcinoma. J Pathol. 2005;206(2):170–7.
58. Ohno F, Nakanishi H, Abe A, Seki Y, Kinoshita A, Hasegawa Y, et al. Regional difference in intratumoral lymphangiogenesis of oral squamous cell carcinomas evaluated by immunohistochemistry using D2-40 and podoplanin antibody: an analysis in comparison with angiogenesis. J Oral Pathol Med. 2007;36(5):281–9.
59. Ochoa-Alvarez JA, Krishnan H, Shen Y, Achaya NK, Han M, et al. Plant lectin can target receptors containing sialic acid, exemplified by podoplanin, to inhibit transformed cell growth and migration. PLoS ONE. 2012;7(7):e41845. doi:10.1371/journal.pone.0041845.
60. Chandramohan V, Bao X, Kato KM, Kato Y, Keir ST, Szafranski SE, et al. Recombinant anti-podoplanin (NZ-1) immunotoxin for the treatment of malignant brain tumors. Int J Cancer. 2013;132(10):2339–48.
61. Takagi S, Sato S, Oh-hara T, Takami M, Koike S, et al. Platelets promote tumor growth and metastasis via direct interaction between Aggrus/podoplanin and CLEC-2. PLoS ONE. 2013;8(8):e73609. doi:10.1371/journal.pone.0073609.
62. Cueni LN, et al. Podoplanin-Fc reduces lymphatic vessel formation in vitro and in vivo and causes disseminated intravascular coagulation when transgenically expressed in the skin. Blood. 2010;116(20):4376–84.

KNOW YOUR FIELD

Histopathological variants of oral squamous cell carcinoma-institutional case reports

Jigna Pathak, Niharika Swain, Shilpa Patel, LS Poonja
Department of Oral Pathology, Mahatma Gandhi Mission's Dental College and Hospital, Navi Mumbai, Maharashtra, India

INTRODUCTION

Squamous cell carcinoma is by far the most important and the most common malignant mucosal neoplasm of the head and neck accounting for over 90% of all malignancies. Conventional oral squamous cell carcinoma (OSCC) can present as several variants that make up in aggregate about 10-15% of all squamous cell carcinomas (SCC).[1] These variants include verrucous carcinoma (VC), adenoid/acantholytic/pseudoglandular SCC (AdSCC), spindle cell/sarcomatoid carcinoma (SCSC), adenosquamous carcinoma (ASC), basaloid SCC (BSCC) and papillary SCC (PSCC). Each of these variants has a unique histomorphological appearance. This is a short treatise designed to give a brief overview of the different histopathological variants of OSCC observed in our institute, the separation of which helped in achieving appropriate clinical management.

CASE REPORT

A brief overview of the clinico-pathological appearance of variants of OSCC cases reported in the Department of Oral and Maxillofacial Pathology is presented in Table 1.

DISCUSSION

Conventional SCC [Figure 1] and variants of OSCC frequently arise within the oral cavity. Precise histopathological diagnosis can help the clinician to plan accurate treatment, as the prognosis of each of them differs considerably.

VC [Figure 2] is a very well-differentiated SCC that does not metastasize and has an excellent prognosis with 5-year survival rate of approximately 75%.[2] The lesion has a possibility of metastasis only if it is left long enough and allowed to become more invasive. AdSCC [Figure 3] occurs in the oral cavity infrequently as they usually affect sun-exposed areas with vermillion border of the lip being the most commonly affected site. They have a relative poorer prognosis as compared with conventional SCC [Figure 1]. SCSC [Figure 4] metastasizes to the regional lymph nodes in upto 25% cases, but distant metastasis is less

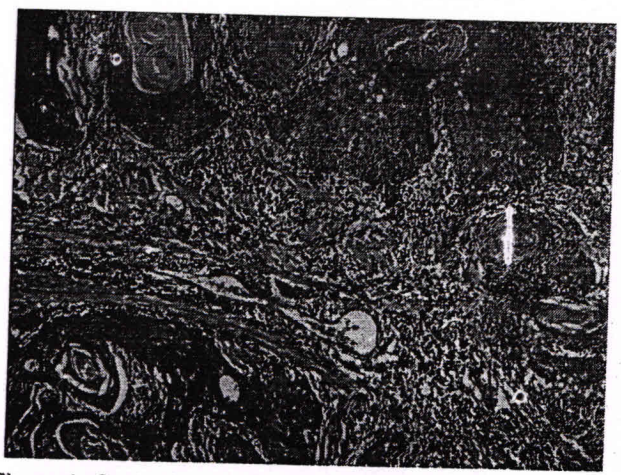


Figure 1: Conventional oral squamous cell carcinoma-malignant epithelial islands showing keratin pearl formation. (H&E stain, ×100)

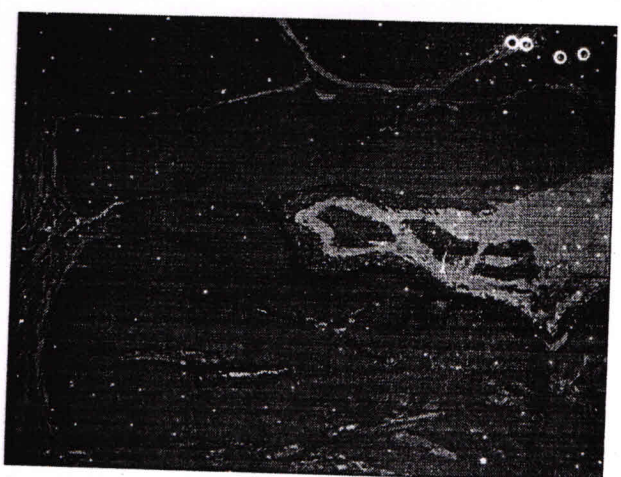


Figure 2: Verrucous carcinoma-broad bulbous pushing rete ridges with parakeratotic plugging (H&E stain, ×100)

Access this article online
Quick Response Code:
Website: www.jomfp.in
DOI: 10.4103/0973-029X.131945

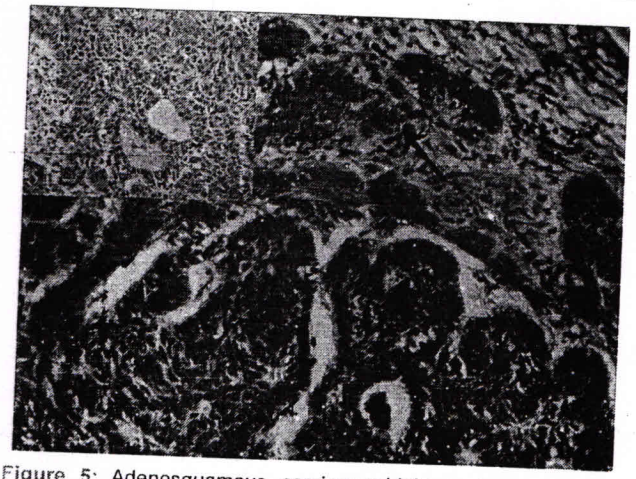


Figure 5: Adenosquamous carcinoma-biphasic tumor showing true glandular differentiation (arrowhead) along with squamous differentiation (arrow) (H&E stain, ×100). Inset depicts alcian blue-positive mucin secretion (×400)

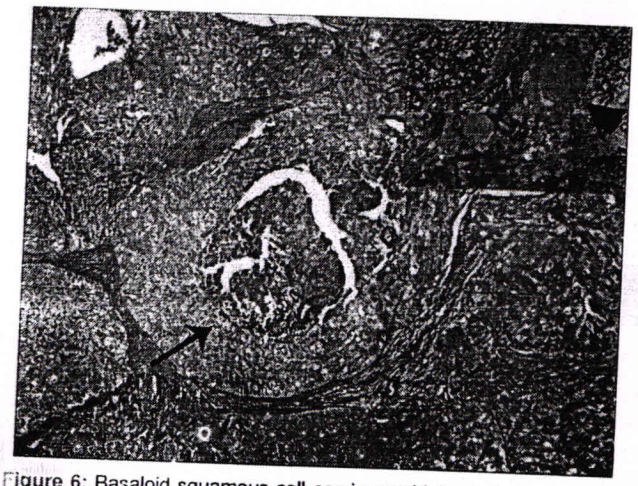


Figure 6: Basaloid squamous cell carcinoma biphasic tumor showing basaloid malignant islands with peripheral palisading and comedonecrosis (arrow) (H&E stain, ×100). Inset depicts squamous differentiation with keratin pearl formation (arrowhead) (H&E stain, ×100)

aggressive multimodality treatment. The 2-year survival rate is 40%.[4] PSCC more frequently affects the larynx. It has a better prognosis when compared with location and stage-matched conventional OSCC.[5]

CONCLUSION

Histopathological variants of OSCC may pose a diagnostic challenge especially the SCSC and ASC, which warrants the use of immunohistochemistry and special stains for an accurate diagnosis. The prognosis, metastatic potential, survival rate and treatment of each of the variants are diverse, thus mandating their distinction.

REFERENCES

1. Thompson LD. Squamous cell carcinoma variants of the head and neck. Curr Diagn Pathol 2003;9:384-96.
2. Koch BB, Trask DK, Hoffman HT, Karnell LH, Robinson RA, Zhen W, *et al.* National survey of head and neck verrucous carcinoma: Patterns of presentation, care, and outcome. Cancer 2001;92:110-20.
3. Viswanathan S, Rahman K, Pallavi S, Sachin J, Patil A, Chaturvedi P, *et al.* Sarcomatoid (spindle cell) carcinoma of the head and neck mucosal region: A clinicopathologic review of 103 cases from a tertiary referral cancer centre. Head Neck Pathol 2010;4:265-75.
4. Cardesa A, Zidar N, Ereño C. Basaloid squamous cell carcinoma. In: Barnes L, editor. World Health Organization Classification of Tumors. Pathology & Genetics of Head and Neck Tumours. Lyon: IARC Press; 2005.
5. Thompson LD, Wenig BM, Heffner DK, Gnepp DR. Exophytic and papillary squamous cell carcinomas of the larynx: A clinicopathologic series of 104 cases. Otolaryngol Head Neck Surg 1999;120:718-24.

How to cite this article: Pathak J, Swain N, Patel S, Poonja LS. Histopathological variants of oral squamous cell carcinoma-institutional case reports. J Oral Maxillofac Pathol 2014;18:143-5.

Source of Support: Nil. **Conflict of Interest:** None declared.

10.5005/jp-journals-10031-1068

ASE REPORT

Trabecular Juvenile Ossifying Fibroma of the Craniofacial Skeleton: Etiopathogenesis and a Case Report of the Rare Entity

[1]Rahul Kadam, [2]Shilpa Patel, [3]Jigna Pathak, [4]Niharika Swain, [5]Shwetha Kumar

ABSTRACT

The term, fibro-osseous lesions, is used for a group of pathological disturbances encompassing developmental, reactive or dysplastic lesions and neoplasms characterized by placement of normal bone architecture by tissue composed collagen fibers and fibroblasts containing various amount of calcified tissue. The groups of the fibro-osseous lesions are best considered as a spectrum of processes arising from cells in the periodontal ligament. Juvenile ossifying fibroma (JOF) is a benign, but potentially aggressive, fibro-osseous tumor of the craniofacial bones. This uncommon neoplasm is distinguished from other fibro-osseous lesions primarily by its age of onset, clinical presentation, potential behavior and the high tendency to recur. Clinically presenting as an actively growing lesion. Histopathologically consists cell rich fibrous stroma containing bands of cellular osteoid without osteoblastic lining together with trabeculae of more typical woven bone. Pathogenesis of JOF may be related to mutations of HRPT2 gene which may arises due to haploinsufficiency of the HRPT2 gene.

Here, we reported a case of trabecular JOF (TJOF) which had variations in clinical, radiographic features and histopathological characteristics and it's etiopathogenesis in detail.

Keywords: Fibro-osseous lesions, Juvenile ossifying fibroma, Trabecular, Maxilla, Mandible.

How to cite this article: Kadam R, Patel S, Pathak J, Swain N, Kumar S. Trabecular Juvenile Ossifying Fibroma of the Craniofacial Skeleton: Etiopathogenesis and a Case Report of the Rare Entity. J Contemp Dent 2014;4(1):51-55.

Source of support: Nil

Conflict of interest: None

INTRODUCTION

Juvenile ossifying fibroma (JOF) is a benign, but potentially aggressive, fibro-osseous tumor of the craniofacial bones. This uncommon bone forming neoplasm is distinguished from other fibro-osseous lesions primarily by its age of onset, clinical presentation, potential behavior and the high tendency to recur.[1,2] The second edition of the WHO classification of odontogenic tumor defines JOF as 'an actively growing lesion consisting of a cell-rich fibrous stroma, containing bands of cellular osteoid without osteoblastic lining, together with trabeculae of more typical woven bone. Small foci of giant cells may also be present; the lesion is non-encapsulated but well-demarcated from surrounding bone'.[3] This terminology is given to some rarely reported ossifying fibromas, mainly found in maxilla at an early age and in 79% of the patients are diagnosed before the age of 15 or younger which histologically showed a highly cellular but loose fibroblastic stroma containing strands of osteoid with entrapped osteoblast.[4] The JOF occurs predominantly but not exclusively in children. It affects males and females equally without any gender predilection.[5,6] The JOF is located mainly (85%) in facial bones, in some cases (12%) in calvarium and very rarely (3%) extracranially. Ninety percent of the lesions located in the facial region.[4] Mandibular lesions are seen in 10% of the cases. Clinically, the lesion seems to be expands in the facial bone leading to facial asymmetry. The clinical symptoms, such as pain, paraesthesia, malocclusion, sinusitis and proptosis depend upon the site of tumor involvement. Root displacement is common but root resorption is rare. The lesion can cause expansion as well as perforation.[7]

Radiographically, they present as a well-circumscribed radiolucencies that in some cases contain central radiopacities or they appear radiolucent, radiopaque or mixed radiolucent-radiopaque with a well-defined sclerotic border, lesions that are more aggressive may show cortical thinning and perforation.[8] Being on the basis of different histopathological and clinical features in the WHO classification of odontogenic tumors 2005, juvenile ossifying fibroma (JOF) is further subdivided into juvenile psammomatoid ossifying fibroma (JPOF) and juvenile trabecular ossifying fibroma (JTOF).[9,10]

Here, we reported a case of trabecular type of JOF in 18-year-old male patient with a chief complaint of painless swelling in the lower right anterior region with its variation in clinical behavior, radiographic features and histopathological characteristics.

[1]Postgraduate Student, [2]Professor and Head, [3]Professor [4,5]Senior Lecturer

[1-5]Department of Oral Pathology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India

Corresponding Author: Rahul Kadam, Postgraduate Student, Department of Oral Pathology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India, Phone: 02227426604, e-mail: rahul33kadam@gmail.com

Multiple cementomas (periapical cemental dysplasia): ... of a case. Oral Surg Oral Med Oral Pathol 1976;42: ...06.

...ovitz BKB, Holland GR, Moxham BJ. Color atlas and ...tbook of oral anatomy, histology and embryology. 2nd ed. ...t Louis: Mosby-Year Book 1992;191-192.

... Di Fiore PM, Cemento-osseous dysplasia in African-American men: a report of two clinical cases. J Tennessee Dent Assoc 2010; 90(4):24-28.

22. Bencharit S, Schardt-Sacco D, Zuniga JR, Minsley GE. Surgical and prosthodontic rehabilitation for a patient with aggressive florid cemento-osseous dysplasia: a clinical report. J Prosthet Dent 2003;90:220-224.
23. Dumas M, Ohanian H, Forest D. A case report of fluoride cemento-osseous dysplasia. J Dent Que 2000;37:97-101.
24. Melrose RJ. The clinico-pathologic spectrum of cemento-osseous dysplasia. Oral Maxillofac Clin Nor Am 1997;9:643-653.
25. Yazicioglu D, et al. Focal cemento-osseous dysplasia: a case report and literature review. Health 2010;941-944.
26. Radojica, et al. Focal cemento-osseous dysplasia in the maxilla mimicking periapical granuloma. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999;88:87-89.

Case Report

An insight into diagnostically challenging salivary gland malignancy with case report: Polymorphous low grade adenocarcinoma

Amit Shah, Shilpa Patel, Jigna Pathak, Niharika Swain, Swenil Shah[2]

Department of Oral Pathology and Microbiology, MGM Dental College and Hospital, Kamothe, Navi Mumbai, [2]Department of Radiology, Government Medical College and Hospital, Nagpur, Maharashtra, India

Access this article online

Website: www.ladrsd.org

Quick response code

Abstract

Polymorphous low-grade adenocarcinoma (PLGA) is difficult to diagnose due to its indolent clinical presentation and due to its morphological diversity that includes several microscopic patterns. Distinguishing it from high-grade tumors of salivary gland is important, as the management and prognosis of this tumor differ. We report a considerably rare case of PLGA in retromolar area highlighting various diagnostic challenges caused by the overlap of clinical and microscopic features between PLGA and other salivary gland neoplasms and discuss current management strategies.

Key words: High-grade tumors, polymorphous low-grade adenocarcinoma, retromolar area, salivary gland neoplasms

Introduction

The present term, polymorphous low-grade adenocarcinoma (PLGA) was first described by Evans and Batsakis in 1984 and has replaced various previously described terms like lobular carcinoma described by Freedman and Lumerman in 1983 and terminal duct carcinoma described by Batsakis *et al.* in 1983.[1-3] World Health Organization in their 1991 classification of salivary gland tumors considers PLGA as separate entity and included tumors which were previously misdiagnosed as pleomorphic adenomas (PAs), monomorphic adenomas, malignant PAs, adenoid cystic carcinomas (AdCCs) or adenocarcinoma not otherwise specified.[4,5] Almost exclusive to minor salivary glands, PLGA affects palate most frequently and accounts for about 60% of all PLGAs. Buccal and labial mucosa, alveolar mucosa, retromolar trigone, tongue, floor of mouth and parotid gland are other sites of occurrence in that order.[6,7] Uncommon locations include lacrimal glands, nasopharynx, nasal cavity, tonsillar region and paranasal sinuses.[5-7] We present a rare case of PLGA of retromolar area highlighting various diagnostic challenges caused by the overlap of clinical and microscopic features between PLGA and other salivary gland neoplasms and discuss current management strategies.

Case Report

The present case report is about a 20-year-old male patient who reported with an asymptomatic swelling in his right retromolar region of about 4 months duration. On examination, a soft, smooth mass of about 2 cm × 1 cm in size was present in the right retromolar area. The soft-tissue mass was non-ulcerated, non-tender and was not fixed to underlying structures. There was no associated lymphadenopathy or sensory deficit. Hard tissue involvement was not present and was confirmed radiographically [Figure 1].

Considering the innocuous clinical presentation, excision of the lesion was performed. On histopathological examination, a well-circumscribed but unencapsulated lesion with infiltrative growth pattern was observed. Tumor consisted of polymorphous growth pattern in the center while characteristic Indian file pattern was present at the periphery. The individual cells were round to oval and spindled at places, with moderate cytoplasm and vesicular nuclei with prominent nucleoli and occasional mitosis. A small population of mucous like cells and clear cells were also evident. Connective tissue stroma was predominantly myxoid with delicate fibrous tissue. Lymphoid aggregates were observed in association with the tumor.

Correspondence to: Dr. Amit Shah, Department of Oral Pathology, MGM Dental College and Hospital, At the Junction of NH4 and Sion Panvel Expressway, Kamothe, Navi Mumbai - 410 209, Maharashtra, India. E-mail: amitshah@dr.com

14. Zarbo RJ. Salivary gland neoplasia: A review for the practicing pathologist. Mod Pathol 2002;15:298-323
15. Gnepp DR, Chen JC, Warren C. Polymorphous low-grade adenocarcinoma of minor salivary gland. An immunohistochemical and clinicopathologic study. Am J Surg Pathol 1988;12:461-8.
16. Caselitz J, Schulze I, Seifert G. Adenoid cystic carcinoma of the salivary glands: An immunohistochemical study. J Oral Pathol 1986;15:308-18.
17. Darling MR, Schneider JW, Phillips VM. Polymorphous low-grade adenocarcinoma and adenoid cystic carcinoma: A review and comparison of immunohistochemical markers. Oral Oncol 2002;38:641-5.
18. Skálová A, Simpson RH, Lehtonen H, Leivo I. Assessment of proliferative activity using the MIB1 antibody help to distinguish polymorphous low grade adenocarcinoma from adenoid cystic carcinoma of salivary glands. Pathol Res Pract 1997;193:695-703.
19. Curran AE, White DK, Damm DD, Murrah VA. Polymorphous low-grade adenocarcinoma versus pleomorphic adenoma of minor salivary glands: Resolution of a diagnostic dilemma by immunohistochemical analysis with glial fibrillary acidic protein. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;91:194-9.
20. Simpson RH, Pereira EM, Ribeiro AC, Abdulkadir A, Reis-Filho JS. Polymorphous low-grade adenocarcinoma of the salivary glands with transformation to high-grade carcinoma. Histopathology 2002;41:250-9.
21. Olusanya AA, Akadiri OA, Akinmoladun VI, Adeyemi BF. Polymorphous low grade adenocarcinoma: Literature review and report of lower lip lesion with suspected lung metastasis. J Maxillofac Oral Surg 2011;10:60-3.
22. Takubo K, Doi R, Kidani K, Nakabayashi M, Sonoda M, Ohtake F, *et al.* Polymorphous low-grade adenocarcinoma arising at the retromolar region: A rare case of high-grade malignancy. Yonago Acta Med 2007;50:17-22.
23. Seethala RR, Johnson JT, Barnes EL, Myers EN. Polymorphous low-grade adenocarcinoma: The University of Pittsburgh experience. Arch Otolaryngol Head Neck Surg 2010;136:385-92.

How to cite this article: Shah A, Patel S, Pathak J, Swain N, Shah S. An insight into diagnostically challenging salivary gland malignancy with case report: Polymorphous low grade adenocarcinoma. JDRSD 2014;1:20-3

Source of Support: Nil. **Conflict of Interest:** No conflict of interest.

Medknow Publications and Media Pvt. Ltd.

B 9-12, Kanara Business Centre, Off Link Road, Ghatkopar (E), Mumbai – 400075, India. Phone: 91-22-66491818 Website: www.medknow.com

Hindawi Publishing Corporation
The Scientific World Journal
Volume 2014, Article ID 842491, 8 pages
http://dx.doi.org/10.1155/2014/842491

57

Dr. Shilpa Patel.
Category - I
Author - II
Point - 7.5

4 copies

Hindawi

Review Article

The Evolving Concepts of Cancer Stem Cells in Head and Neck Squamous Cell Carcinoma

Amit Shah, Shilpa Patel, Jigna Pathak, Niharika Swain, and Shwetha Kumar

Department of Oral Pathology & Microbiology, M.G.M. Dental College & Hospital, Kamothe, Navi Mumbai 410209, India

Correspondence should be addressed to Amit Shah; amitshah@dr.com

Received 31 August 2013; Accepted 24 October 2013; Published 21 January 2014

Academic Editors: L. Vermeulen and Z. Wang

There is increasing evidence that the growth and spread of cancers is driven by a small subpopulation of cancer stem cells (CSCs)—the only cells that are capable of long-term self-renewal and generation of the phenotypically diverse tumor cell population. CSCs have been identified and isolated in a variety of human cancers including head and neck squamous cell carcinoma (HNSCC). The concept of cancer stem cells may have profound implications for our understanding of tumor biology and for the design of novel treatments targeted toward these cells. The present review is an attempt to conceptualize the role of CSCs in HNSCC—its implication in tumorigenesis and the possible additional approach in current treatment strategies.

1. Introduction

Global increase in incidence and mortality associated with head and neck squamous cell carcinomas (HNSCC) have intensified efforts in the field of research pertaining to tumor biology and therapeutics. HNSCC is one of the most prevalent types of malignancy worldwide. The mortality due to HNSCC is mainly caused by local recurrence and cervical lymph node metastasis and occasionally by distant organ metastasis. Research in cancer therapeutics has helped in targeting pathways that appear to contribute in tumourigenesis and metastasis with greater efficacy and fewer unwanted side effects. An important premise guiding this work is the cancer stem cell hypothesis. The cancer stem cell (CSC) theory of tumourigenesis was originally proposed in the late 1970s and was first described in hematologic malignancies in 1994 [1]. Since then, CSCs have been identified in multiple other solid organ malignancies, including Central Nervous System (CNS), pancreatic, lung, colon, and recently HNSCC [2–6].

The consensus definition of a cancer stem cell that arrived at an "American Association of Cancer Research Workshop on cancer stem cell" is a cell within a tumor that possesses the capacity to self-renew and to cause the heterogeneous lineages of cancer cells that comprise the tumor [7]. Various alternative terms have been used in the literature, such as "tumor-initiating cell" and "tumorigenic cell" to describe putative cancer stem cells. The origin of these cells, their role in cancer progression and metastasis, and possible therapeutic approaches with special implications on HNSCC are highlighted here.

2. Origin of Cancer Stem Cells

Various types of stem cells give rise to progenitor cell which have the ability to further divide into specialized or differentiated cells that carry out the specific functions of the body. It is controversial as to whether CSCs arise from stem cells, progenitor cells, or differentiated cells present in adult tissue. The issue is currently under debate and the theories in origin of stem cells are presented here (Figure 1).

2.1. Hypothesis Number 1: Cancer Cells Arise from Stem Cells. In this scenario, cancer cells could simply utilize the existing stem cell regulatory pathways to promote their self-renewal. The ability to self-renew gives stem cells long lifespans relative to those of mature, differentiated cells [8]. It has therefore been hypothesized that the limited lifespan of a mature cell makes it less likely to live long enough to undergo the multiple mutations necessary for tumor formation and metastasis [9].

Dean
M. G. M. Dental College & Hospital
Kamothe, Navi Mumbai - 410 209.

[58] ...moto, K. Chikamatsu, K. Sakakura, K. Hatsushika, G. ...shiu, and K. Masuyama, "Expansion and characterization ...cancer stem-like cells in squamous cell carcinoma of the head ...and neck," *Oral Oncology*, vol. 45, no. 7, pp. 633–639, 2009.

[59] S. H. Chiou, C. C. Yu, C. Y. Huang et al., "Positive correlations of Oct-4 and Nanog in oral cancer stem-like cells and high-grade oral squamous cell carcinoma," *Clinical Cancer Research*, vol. 14, no. 13, pp. 4085–4095, 2008.

[59] Q. Zhang, S. Shi, Y. Yen, J. Brown, J. Q. Ta, and A. D. Le, "A subpopulation of $CD133^+$ cancer stem-like cells characterized in human oral squamous cell carcinoma confer resistance to chemotherapy," *Cancer Letters*, vol. 289, no. 2, pp. 151–160, 2010.

[60] A. Pannuti, K. Foreman, P. Rizzo et al., "Targeting Notch to target cancer stem cells," *Clinical Cancer Research*, vol. 16, no. 12, pp. 3141–3152, 2010.

[61] F. Takahashi-Yanaga and M. Kahn, "Targeting Wnt signaling: can we safely eradicate cancer stem cells?" *Clinical Cancer Research*, vol. 16, no. 12, pp. 3153–3162, 2010.

[62] T. Takezaki, T. Hide, H. Takanaga, H. Nakamura, J. Kuratsu, and T. Kondo, "Essential role of the Hedgehog signaling pathway in human glioma-initiating cells," *Cancer Science*, vol. 102, no. 7, pp. 1306–1312, 2011.

[63] J. Smith, E. Ladi, M. Mayer-Pröschel, and M. Noble, "Redox state is a central modulator of the balance between self-renewal and differentiation in a dividing glial precursor cell," *Proceedings of the National Academy of Sciences of the United States of America*, vol. 97, no. 18, pp. 10032–10037, 2000.

[64] J. A. Thomson, J. I. skovitz-Eldor, S. S. Shapiro et al., "Embryonic stem cell lines derived from human blastocysts," *Science*, vol. 282, no. 5391, pp. 1145–1147, 1998.

[65] Y. Zhao, Q. Bao, A. Renner et al., "Cancer stem cells and angiogenesis," *International Journal of Developmental Biology*, vol. 55, no. 4-5, pp. 477–482, 2011.

[66] C. Calabrese, H. Poppleton, M. Kocak et al., "A perivascular Niche for brain tumor stem cells," *Cancer Cell*, vol. 11, no. 1, pp. 69–82, 2007.

[67] S. Krishnamurthy, Z. Dong, D. Vodopyanov et al., "Endothelial cell-initiated signaling promotes the survival and self-renewal of cancer stem cells," *Cancer Research*, vol. 70, no. 23, pp. 9969–9978, 2010.

[68] M. Pàez-Ribes, E. Allen, J. Hudock et al., "Antiangiogenic therapy elicits malignant progression of tumors to increased local invasion and distant metastasis," *Cancer Cell*, vol. 15, no. 3, pp. 220–231, 2009.

[69] C. Tang, B. T. Ang, and S. Pervaiz, "Cancer stem cell: target for anti-cancer therapy," *The FASEB Journal*, vol. 21, no. 14, pp. 3777–3785, 2007.

Journal of Oral and Maxillofacial Surgery, Medicine, and Pathology xxx (2013) xxx–xxx

Contents lists available at ScienceDirect

Journal of Oral and Maxillofacial Surgery, Medicine, and Pathology

journal homepage: www.elsevier.com/locate/jomsmp

Case report

Soft tissue myoepithelial carcinoma of neck: A rare case report with review of literature☆

Niharika Swain*, Shwetha V. Kumar, Jigna Pathak, Shilpa Patel

Department of Oral Pathology, MGM Dental College and Hospital, Junction of NH-4 and Sion Panvel Expressway, Sector-18, Kamothe, Navi Mumbai 410 209, Maharashtra, India

ARTICLE INFO

Article history:
Received 18 March 2013
Received in revised form 21 June 2013
Accepted 11 July 2013

Keywords:
Soft tissue tumor
Myoepithelial neoplasms
p63
Imunoexpression

ABSTRACT

Primary soft tissue myoepithelial tumor, an uncommon variant of myoepithelial neoplasms, has been recently described in reviewed literature. We report a rare case of soft tissue myoepithelial carcinoma in a 60 year old male patient presented as a unilateral neck mass. MRI showed a large lobulated infiltrating heterointense mass with central necrotic area involving left parapharyngeal space. Histopathological examination revealed multilobular growth pattern of epitheloid cells with marked atypia and frequent mitosis. Immunohistochemistry was reactive for p63, calponin, CD10, pancytokeratin, EMA and podoplanin with high (40%) proliferative labeling index. Except for the presence of increased malignant cell population, soft tissue myoepithelial carcinoma mimics their salivary gland counterpart in clinical and biologic behavior. Rarity in head and neck region (only 16%) and heterogeneity in cellular morphology and architectural patterns, may lead to misdiagnosis of extraglandular myoepithelial carcinoma. So, careful and meticulous observation of both histopathologic and immunophenotypic features are essential for correct diagnosis of such entities.

1. Introduction

Myoepithelial neoplasms, initially recognized to be of salivary gland origin, in recent years are known to arise from other primary sites like skin, nasal cavity, paranasal sinuses, breast, lacrimal glands, bronchus, lungs or kidneys [1]. In 1995, the first case of soft tissue myoepithelioma in retroperitonium was reported by Burke et al. Two years later, a case series of 19 cases of soft tissue myoepitheliomas was published by Kilpatrick et al. [2]. This uncommon soft tissue counterpart was included as a separate entity in World Health Organization Classification of soft tissue tumors in 2002 [3]. In 2003, Hornick et al. published the largest case series of 101 cases of this entity with proposal of criteria for malignancy and prognostic parameters [4]. In 2007, 29 cases of soft tissue myoepithelial carcinoma in pediatric population were reported by Gleason et al. The histogenesis of these extraglandular neoplasms is poorly understood [5]. Due to wide variation in morphologic and immunophenotypic expression of myoepithelial cells, it is challenging to distinguish these neoplasms from other soft tissue tumors, when arising in unusual sites. Furthermore, as these neoplasms are extremely rare in head and neck region, soft tissue myoepithelial tumors are more liable to misdiagnosis.

2. Case report

A 60-year-old male patient reported with a large rapidly growing unilateral neck swelling since 2 months (Fig. 1 A). The swelling was non-tender with no associated symptoms. The magnetic resonance imaging (MRI) showed a large lobulated infiltrating heterointense mass extending medially anterior to the carotid space in left parapharyngeal space, posteriorly into the posterior triangle of neck, inferiorly along the lateral aspect of carotid space in submandibular region and superiorly upto the parotid space. The lesion did not show any parotid gland involvement. On post contrast evaluation there was moderate heterogenous enhancement of the lesion with large non-enhancing well defined necrotic area in the center (Fig. 1B and C). An incisional biopsy revealed poorly differentiated carcinoma. The patient further underwent wide surgical tumor resection including radical neck dissection. Intra-operatively, no continuity with regional major salivary glands was observed (Fig. 2 A and B).

Histopathologically, multilobular growth pattern with nests of predominantly epitheloid cells with areas of necrosis and hemorrhage was noted. In some areas spindle and clear cell

☆ AsianAOMS: Asian Association of Oral and Maxillofacial Surgeons; ASOMP: Asian Society of Oral and Maxillofacial Pathology; JSOP: Japanese Society of Oral Pathology; JSOMS: Japanese Society of Oral and Maxillofacial Surgeons; JSOM: Japanese Society of Oral Medicine; JAMI: Japanese Academy of Maxillofacial Implants.

* Corresponding author. Tel.: +91 93 24701036; fax: +91 22 27423185.
E-mail address: niharikadec30@gmail.com (N. Swain).

2212-5558/$ – see front matter © 2013 Asian AOMS, ASOMP, JSOP, JSOMS, JSOM, and JAMI. Published by Elsevier Ltd. All rights reserved.
http://dx.doi.org/10.1016/j.ajoms.2013.07.014

Please cite this article in press as: Swain N, et al. Soft tissue myoepithelial carcinoma of neck: A rare case report with review of literature. J Oral Maxillofac Surg Med Pathol (2013), http://dx.doi.org/10.1016/j.ajoms.2013.07.014

Gleason BC, Fletcher CDM. Myoepithelial carcinoma of soft tissue in children: an aggressive neoplasm analyzed in a series of 29 cases. Am J Surg Pathol 2007;31:1813–24.
[6] Gleason BC, Hornick JL. Minisymphosium; soft tissue tumor pathology. Diagn Histopathol 2008;14:552–62.
[7] Skalova A, Jakel KT. Myoepithelial carcinoma. In: Barnes L, Eveson JW, Reichart PA, et al, editors. Pathology and genetics of head and neck tumours. World Health Organization Classification of Tumours. Lyon: IARC Press; 2005. p. 240–1.
[8] Kane SV, Bagwan IN. Myoepithelial carcinoma of the salivary glands: a clinicopathologic study of 51 cases in a tertiary cancer center. Arch Otolaryngol Head Neck Surg 2010;136:702–12.
[9] Kundu S, Chatterjee S, Mondal D, Dashidar AG, Roy A. Myoepithelial carcinoma in infratemporal fossa. Indian J Med Pediatr Oncol 2008;29:23–5.
[10] Go JH. A case of soft tissue myoepithelial tumor arising in masticator space. Yonsei Med J 2005;46:710–4.
[11] Minoda R, Masako M, Masuyama K, Yumoto E. Malignant myoepithelioma arising within the masticator space ectopically. Otolaryngol Head Neck Surg 2001;124:342–3.
[12] Erdogan S, Rodriguez FJ, Scheithauer BW, Abell-Aleff PC, Rabin M. Malignant myoepithelioma of cranial dura. Am J Surg Pathol 2007;31:807–11.
[13] Tsuneki M, Maruyama S, Yamazaki M, Essa A, Abé T, Babkair HA, et al. Podoplanin is a novel myoepithelial cell marker in pleomorphic adenoma and other salivary gland tumors with myoepithelial differentiation. Virchows Arch 2013;462:297–305.
[14] Joseph LD, Kumar AR, Kannan S, Prathiba D. Malignant myoepithelioma of the mastoid region—a case report. Indian J Otolaryngol Head Neck Surg 2007;59:194–6.
[15] Ghosh A, Saha S, Saha VP, Sadhu A, Chattopadhyay S. Infratemporal fossa myoepithelial carcinoma—a rare case report. Oral Maxillofac Surg 2009;13:59–62.

Please cite this article in press as: Swain N, et al. Soft tissue myoepithelial carcinoma of neck: A rare case report with review of literature. J Oral Maxillofac Surg Med Pathol (2013), http://dx.doi.org/10.1016/j.ajoms.2013.07.014

10.5005/jp-journals-10031-1048

CASE REPORT

Focal Cemento-osseous Dysplasia

Rahul Kadam, Shilpa Patel, Jigna Pathak, Niharika Swain, Shwetha Kumar

ABSTRACT

Focal cemento-osseous dysplasia (FCOD) is a benign fibroosseous condition that can be seen in dentulous and edentulous patients. It is an asymptomatic lesion and needs no treatment; however, follow-up is essential due to the possibility that it can progress to a condition called florid cemento-osseous dysplasia. Clinically, the lesion resembles periapical pathosis of odontogenic origin. FCOD is an asymptomatic lesion and occurs in the periapical area of teeth with vital pulps or in regions of extractions. The lesion is detected only on radiographic examination varying from completely radiolucent to densely radiopaque. The histopathologic appearance consists of trabeculae of bone and cementum like material present within a vascular fibrous stroma. Presented here is a case of FCOD in the mandible that occurred in the periapical region of a vital tooth.

Keywords: Fibroosseous lesions, Cemento-osseous dysplasia, Jaws.

How to cite this article: Kadam R, Patel S, Pathak J, Swain N, Kumar S. Focal Cemento-osseous Dysplasia. J Contemp Dent 2013;3(2):112-115.

Source of support: Nil

Conflict of interest: None declared

INTRODUCTION

Benign fibroosseous lesions are rare diseases which are characterized by replacement of healthy bone and connective tissue that transforms to cemento-osseous tissue.[1] Fibroosseous lesions can be classified in three categories as fibrous dysplasia, benign fibroosseous neoplasms and reactive lesions.[1] The term cemento-osseous dysplasia is a non-neoplastic lesion related to teeth bearing area. The term cemento-osseous dysplasia was used first time for the World Health Organization (WHO) classification in 1992.[2] It is used because of its difficulty in discrimination of cementum and bone tissue in lesions which produce cementum, bone and connective tissue. Cemento-osseous dysplasias are non-neoplastic lesions which include periapical osseous dysplasia, focal cemento-osseous dysplasia (FCOD) and florid cemental dysplasia.[3]

Focal cemento-osseous dysplasia (FCOD) is seen predominantly in African-American females, with a peak incidence in the fourth and fifth decades. FCOD affects edentulous jaws and tooth extraction sockets attaining 1 to 2 cm size in dimensions. FCOD can cause expansion of the surrounding bone and can be secondarily infected.[4-6]

It is mostly a well-defined radiolucency with a sclerotic border or a mixed radiolucent and radiopaque lesion.[4] Histopathologically, FCOD is formed by spindle cells, bone-cementum like trabeculation and connective tissue stroma. Histopathological view varies due to the stage of the lesion. In the early osteolytic stage, cellular and vascular structures are predominant and surrounded by a proliferated fibrous connective tissue stroma.[4,5] Cementum-like structures cannot be seen.[4,5] In the late osteosclerotic stage, the lesion reveals poor cellularity, bony trabeculae and irregular cementum-like structures making anastomosis with each other. There is no evidence of cementoblastic and osteoblastic rimming.[4-6] Mid stage consists of both early and late stage characteristics. FCOD does not require treatment and certainly not routine biopsy unless it is infected and symptomatic.[4,7]

In this report, we present a case of a 13-year-old female patient with the lesion on the right mandibular molar region diagnosed as FCOD by histopathological and radiological examination.

CASE REPORT

A 13-year-old female patient reported to Department of Oral Pathology, MGM Dental College and Hospital, with the complaint of pain and swelling on right side of lower jaw since 9 to 10 months.

Patient apparently had no lesion 9 months back. Since last 7 to 8 months, patient complains of a small painful swelling in relation to 45, 46 and 47 which has gradually increased to the present size. The pain associated with the swelling was intermittent in nature. There were no aggravating or relieving factors in association with the present growth. Extraoral examination revealed a diffuse small swelling on lower right posterior side of mandible (Fig. 1).

Intraoral examination revealed a diffuse swelling on mandibular right posterior region approx. 2 × 3 cm in size and irregular in shape with ulcerated surface. Extent of the

Fig. 1: Extraoral appearance of swelling

3. Mupparapu M, Singer SR, Miles M, Rinaggio J. Simultaneous presentation of focal cemento-osseous dysplasia and simple bone cyst of mandible masquerading as a multilocular radioluscency. Dentomaxillofacial Radiology 2005;34(1):39-43.
4. Summerlin DJ, Tomich CE. Focal cemento-osseous dysplasia: a clinicopathological study of 221 cases. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology and Endodontology 1994;78(5):611-620.
5. Su L,Weathers DR, Waldron CA. Distinguishing features of focal cemento-osseous dysplasias and cemento-ossifying fibromas: I. A pathologic spectrum of 316 cases. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology and Endodontology 1997;84(3):301-309.
6. Pindborg JJ, Kramer IRH, Torloni H. Histological typing of odontogenic tumours, jaw cysts and allied lesions. World Health Organization, Geneva 1971.
7. Gunhan O. Oral ve maxillofasiyal patoloji. birinci baský. Atlas Kitapçýlýk Tic. Ltd. 2001;148-149.
8. Jerjes W, Banu B, Hopper C. British Den J 2005;198: 477-478.
9. Marcelo G, Ronaldo P. Carlos Eduardo B Braz Dent J 2005; 16(3):124-250.
10. Ohkura K. Clinicopathological studies on localized cemento-osseous dysplasia of the jaws. Kokubyo Gakkai Zasshi 2001; 68(1):99-110.
11. Melrose RJ. The clinico-pathologic spectrum of cementoosseous dysplasia. Oral Maxillofac Clin Nor Am 1997;9:643-653.
12. Smith S, Patel K, Hoskinson AE. Periapical cemental dysplasia: A case of misdiagnosis. British Dent J 1998;185(3):122-123.
13. Forman GH. Periapical cemental dysplasia resembling apical granulomata and radicular cyst. Br Dent J 1975;138:22-40.
14. Drazzic R, MiniC AJ. Focal cemento-osseous dysplasia in the maxilla mimicking periapical granuloma. Oral Surg, Oral Med, Oral Pathol, Oral Radiol Endod 1999;88:87-90.

ABOUT THE AUTHORS

Rahul Kadam (Corresponding Author)

Postgraduate Student, Department of Oral Pathology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India Phone: 02227426604, e-mail: rahul33kadam@gmail.com

Shilpa Patel

Professor and Head, Department of Oral Pathology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India

Jigna Pathak

Professor, Department of Oral Pathology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India

Niharika Swain

Senior Lecturer, Department of Oral Pathology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India

Shwetha Kumar

Senior Lecturer, Department of Oral Pathology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India

10.5005/jp-journals-10031-1019
REVIEW ARTICLE

Etiological Factors of Recurrent Aphthous Stomatitis: A Common Perplexity

Niharika Swain, Jigna Pathak, Leela S Poonja, Yogita Penkar

ABSTRACT

Recurrent aphthous stomatitis (RAS) is one of the most common oral mucosal disorders. Nevertheless, while the clinical characteristics of RAS are well-defined, the precise etiology and pathogenesis of RAS remain unclear. The present article provides a detailed review of the current knowledge of various etiological factors of RAS.

Keywords: Mouth ulcers, Recurrent aphthous stomatitis, Etiology.

How to cite this article: Swain N, Pathak J, Poonja LS, Penkar Y. Etiological Factors of Recurrent Aphthous Stomatitis: A Common Perplexity. J Contemp Dent 2012;2(3):96-100.

Source of support: Nil

Conflict of interest: None declared

INTRODUCTION

Recurrent aphthous stomatitis (RAS; Aphthae; Canker sores), a common oral mucosal disorder that is characterized by multiple, recurrent, small, round or ovoid ulcers with circumscribed margins, erythematous haloes, and yellow or gray floors that present first in childhood or adolescence. Although it is one of the most common recurrent oral ulcerative conditions of adults and children recognized throughout the world, RAS is also one of the least understood oral diseases and is among the most vexing problems faced by affected patients and clinicians alike.[1,2] The triggering factors that precipitate recurrent episodes in RAS patients seem to be as diverse and unique as the affected individuals themselves, which has posed a challenge for researchers in their attempts to identify a specific causation for this disease. Although the exact etiology of RAS remains obscure, there is growing lucidity with regard to its pathogenesis which has significantly influenced contemporary approaches toward its management. This article reviews the clinical features and various etiological factors of RAS.

CLINICAL FEATURES

'Aphthous' comes from the Greek word 'aphtha', which means ulcer. Despite the redundancy, the medical literature continues to refer to these oral lesions as aphthous ulcers. 'Aphthous stomatitis' has been used interchangeably with 'aphthous ulcers' and may be a more accurate terminology. Aphthous ulcers are round or oval, with a grayish yellow, crateriform base surrounded by an erythematous halo of inflamed mucosa. For 24 to 48 hours preceding the appearance of an ulcer, most patients have a pricking or burning sensation in the affected area. The ulcer usually occurs on the nonkeratinized oral mucosa, including the lips, the buccal mucosa, floor of the mouth, soft palate and the ventral surface of the tongue.

RAS is seen worldwide and may affect up to 25% of the population.[1-3] Recurrent aphthous ulceration has three different variants—minor aphthous ulcers, major aphthous ulcers and herpetiform ulcers, according to the classification described by Stanley[4] in 1972. Minor RAU (MiRAU) is the common variety, affecting about 80% of RAU patients. It is characterized by painful round or oval shallow ulcers, regular in outline, less than 10 mm in diameter, with a gray-white pseudomembrane surrounded by a thin erythematous halo. MiRAU usually occurs on nonkeratinized mucosa such as labial mucosa, buccal mucosa and floor of the mouth. It is uncommon on the keratinized mucosa. Minor RAU is the most common form of childhood RAU. The lesions recur at varying frequencies (from every few years to almost constantly) and heal within 7 to 10 days without scarring. Major RAU (MaRAU), also known as periadenitis mucosa necrotica recurrens, occurs in approximately 10% of RAU patients. The lesions are similar in appearance to those of minor RAU, but they are larger than 10 mm in diameter, single or multiple and very painful. MaRAU has a predilection for the lips, soft palate, and fauces, but can affect any site. The ulcers of MaRAU persist for up to 6 weeks or longer and often heal with scarring. Herpetiform aphthae accounts for 7 to 10% of all RAU cases. In herpetiform RAU there are 10 to 100 ulcers at a time, ulcer size is usually 1 to 3 cm, and the ulcers form clusters that coalesce into widespread areas of ulceration lasting 7 to 10 days. These ulcers are only herpes-like in appearance; herpes simplex virus has not been cultured from them.

ETIOLOGY

To date, the precise etiology of RAS has not been disclosed, despite years of collective effort on the part of many researchers. Historically, conjecture about the origin of RAS focused on a wide spectrum of potential local and systemic factors that encompassed microbial agents, hematologic and hormonal disturbances, physical injury, emotional stress and other influences. Also confounding the search for a singular

28. MacPhail LA, Greenspan JS. Oral ulceration in HIV infection: Investigation and pathogenesis. Oral Dis 1997;3(Suppl 1): S190-93.
29. Phelan JA, Eisig S, Freedman PD, et al. Major aphthous- like ulcers in patients with AIDS. Oral Surg Oral Med Oral Pathol 1991;71:68-72.
30. Cohen L. Etiology, pathogenesis and classification of aphthous stomatitis and Behçet's syndrome. J Oral Pathol 1978;7:347-52.
31. Lehner T. Immunologic aspects of recurrent oral ulcers. Oral Surg Oral Med Oral Pathol 1972;33:80-85.
32. Ben Aryeh H, Malberger E, Gutman D, et al. Salivary IgA and serum IgG and IgA in recurrent aphthous stomatitis. Oral Surg Oral Med Oral Pathol 1976;42:746-52.
33. Wormser GP, Mack L, Lenox T, et al. Lack of effect of oral acyclovir on prevention of aphthous stomatitis. Otolaryngol Head Neck Surg 1988;98:14-17.
34. Kameyama T, Sujaku C, Yamamoto S, et al. Shedding of herpes simplex virus type I into saliva. J Oral Pathol 1988;17:478-81.
35. Neville BW, Damm DD, Allen CM, Bouquot JE. Viral infections: HSV, VZV, CMV. In: Oral and maxillofacial pathology (2nd ed). Philadelphia: WB Saunders 2002:213-24.
36. Veloso FT, Saleiro JV. Small-bowel changes in recurrent ulceration of the mouth. Hepatogastroenterology 1987;34:36-37.
37. Wray D. Gluten-sensitive recurrent aphthous stomatitis. Dig Dis Sci 1981;26:737-40.
38. Olson JA, Feinberg I, Silverman S Jr, et al. Serum vitamin B_{12}, folate and iron levels in recurrent aphthous stomatitis. Oral Surg Oral Med Oral Pathol 1982;54:517-20.
39. Merchant HW, Gangarosa LP, Glassman AB, Sobel RE. Zinc sulphate supplementation for the treatment of recurring oral ulcers. South Med J 1977;70:559-61.
40. Endre L. Recurrent aphthous ulceration with zinc deficiency and cellular immune deficiency. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1991;72:559-61.
41. Merchant HW, Gangarosa LP, Morse PK, Strain WH, Baisden CR. Zinc sulphate as a preventive of recurrent aphthous ulcers. J Dent Res 1981;60A:609.
42. Wray D. A double-blind trial of systemic zinc sulfate in recurrent aphthous stomatitis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1982;53:469-72.
43. Pang JF. Relation between treatment with traditional Chinese medicine for recurrent aphthous ulcer and human zinc and copper. Zhongguo Zhong Xi Yi Jie He Za Zhi 1992;12:280-82.
44. Andrews VH, Hall HR. The effect of relaxation/imagery training on recurrent aphthous stomatitis: A preliminary study. Psychosom Med 1990;52:526-35.
45. Wray D, Graywoski EA, Notkins AL. Role of mucosal injury in initiating recurrent aphthous ulceration. Br Med J 1981;283:1569-70.
46. Shapiro S, Olsson DL, Chellemi SJ. The association between smoking and aphthous ulcers. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1970;30:624-30.
47. Grady D, Ernster VL, Stillman L, Greenspan J. Smokeless tobacco use prevents aphthous stomatitis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1992;74:463-65.
48. Tuzun B, Wolf R, Tuzun Y, Serdaroglu S. Recurrent aphthous stomatitis and smoking. Int J Dermatol 2000;39:358-60.
49. Eversole LR, Shopper TP, Chambers DW. Effects of suspected foodstuff challenging agents in the etiology of recurrent aphthous stomatitis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1982;54:33-38.
50. Wray D, Vlagopoulus TP, Siraganian RP. Food allergens and basophil histamine release in recurrent aphthous stomatitis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1982;54:388-95.
51. Seigel MA, Balciunas BA. Medication can induce severe ulceration. J Am Dent Assoc 1991;122:75-77.
52. Natah SS, Konttinen YT, Enattah NS, Ashammakhi N, Sharkey KA, Häyrinen-Immonenb R. Recurrent aphthous ulcers today: A review of the growing knowledge. Int J Oral Maxillofac Surg 2004;33:221-34.
53. Eisenberg E. Diagnosis and treatment of recurrent aphthous stomatitis. Oral Maxillofacial Surg Clin N Am 2003;15:111-22.
54. Preeti L, Magesh KT, Rajkumar K, Karthik R. Recurrent aphthous stomatitis. J Oral Maxillofac Pathol 2011 Sep-Dec;15(3):252-56.

ABOUT THE AUTHORS

Niharika Swain (Corresponding Author)

Lecturer, Department of Oral Pathology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India
e-mail: niharikadec30@gmail.com

Jigna Pathak

Professor, Department of Oral Pathology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India

Leela S Poonja

Professor, Department of Oral Pathology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India

Yogita Penkar

Lecturer, Department of Oral Pathology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India

10.5005/jp-journals-10031-1006
CASE REPORT

5 Copies

Orthokeratinized Odontogenic Cyst

Niharika Swain, Shilpa Patel, LS Poonja, Jigna Pathak, Kamlesh Dekate

ABSTRACT

Orthokeratinized odontogenic cyst (OOC) is a developmental cyst of odontogenic origin and was initially defined as the uncommon orthokeratinized variant of odontogenic keratocyst (OKC). However, recently World Health Organization has designated OOC as a distinct clinicopathologic entity as it has peculiar clinicopathologic aspects when compared to other developmental odontogenic cysts, especially OKCs. The orthokeratinized odontogenic cyst is histologically characterized by a thin, uniform, epithelial lining with orthokeratinization and a subjacent prominent granular cell layer. The purpose of the article is to present a case of OOC arising in the anterior mandible, an unusual site for the lesion and also highlights the importance of distinguishing it from the more commonly occurring keratocystic odontogenic tumor (KCOT).

Keywords: Odontogenic cyst, Orthokeratinization, Parakeratinization.

How to cite this article: Swain N, Patel S, Poonja LS, Pathak J, Dekate K. Orthokeratinized Odontogenic Cyst. J Contemp Dent 2012;2(2):31-33.

Source of support: Nil

Conflict of interest: None declared

INTRODUCTION

Orthokeratinized odontogenic cyst (OOC), a developmental cyst was first described as a dermoid cyst by Schultz in 1927. In 1945, Philipsen considered this entity as a variant of odontogenic keratocyst (OKC). OOC gained individuality in 1981 as Wright described its clinicopathological features after observing for 30-year period.[1] As recent World Health Organization classification (2005) has considered the odontogenic keratocyst as a neoplasm and designated it as keratocystic odontogenic tumor (KCOT), it is necessary that both oral surgeons and pathologists should possess a thorough knowledge of the clinicopathologic differences between the more aggressive KCOT and the less aggressive OOC so that patients receive the most appropriate treatment.[2]

Here, we report a case of orthokeratinized odontogenic cyst in 42-year-old male patient.

CASE REPORT

A 42-year-old man presented with a 6-month history of discomfort in the anterior region of mandible following extraction of regional teeth. On intraoral examination, extraction wound of 41 was observed (Fig. 1). No obvious swelling was observed in the area of concern. On palpation, slight obliteration of the buccal vestibule extending from 35 to 45 with areas of fluctuation was assessed. Orthopantomograph view showed well-defined unilocular radiolucency with sclerotic margins extending from 37 to 47 and an impacted 33 within (Fig. 2). After clinical and radiographic evaluation, a preliminary diagnosis of odontogenic keratocyst was made. An incisional biopsy was performed under local anesthesia. Tissue sample was submitted for histopathological examination. Microscopic examinations showed a thick cyst wall, lined by the orthokeratinized squamous epithelium (Fig. 3). The flattened basal cell layer lacked the palisading and the prominent granular cell layer was apparent (Fig. 4). The lesion was finally diagnosed as OOC. Patient has undergone segmental mandibulectomy followed by reconstructive surgery.

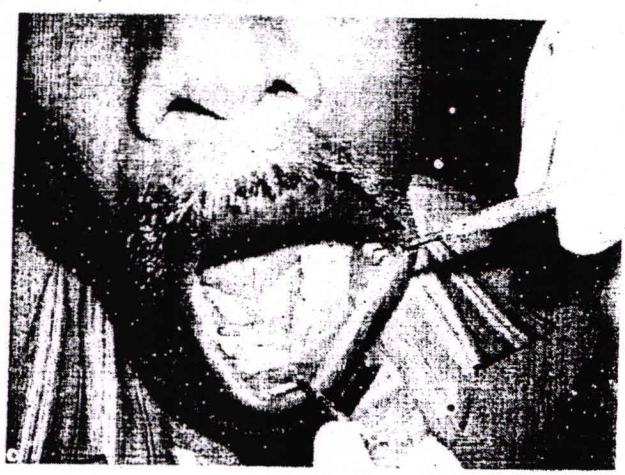


Fig. 1: Intraoral view

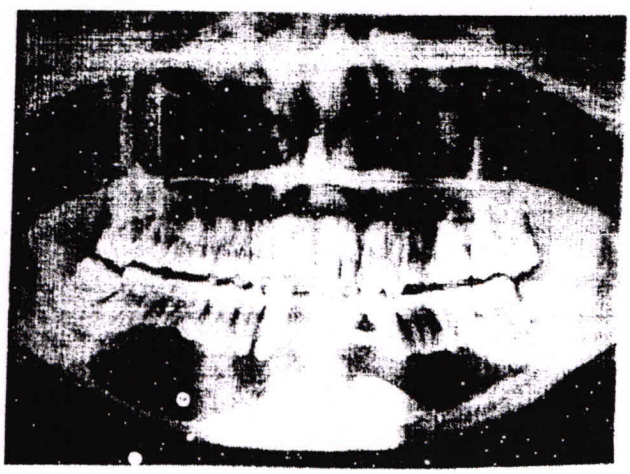


Fig. 2: Orthopantomograph view showed well-defined unilocular radiolucency with impacted 33

4. Li TJ, Kitano M, Chen XM, Itoh T, Kawashima K, Sugihara K, et al. Orthokeratinized odontogenic cyst: A clinicopathological and immunocytochemical study of 15 cases. Histopathology 1998;32(3):242-51.
5. Siar CH, Ng KH. Orthokeratinized odontogenic keratocysts in Malaysians. Br J Oral Maxillofac Surg 1988;26:215-20.
6. MacDonald-Jankowski DS, Li TK. Orthokeratinized odontogenic cyst in a Hong Kong community: The clinical and radiological features. Dentomaxillofacial Radiology 2010;39: 240-45.
7. Jordan RCK. Histology and ultrastructural features of the odontogenic keratocyst. Oral Maxillofacial Surg Clin N Am 2003;15:325-33.
8. Da Silva MJ, de Sousa SO, Corrêa L, Carvalhosa AA, De Araújo VC. Immunohistochemical study of the orthokeratinized odontogenic cyst: A comparison with the odontogenic keratocyst. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002;94(6): 732-37.
9. Rangiani A, Motahhary P. Evaluation of bax and bcl-2 expression in odontogenic keratocysts and orthokeratinized odontogenic cysts: A comparison of two cysts. Oral Oncol 2009;45:e41-44.
10. Simarpreet Virk Sandhu, Sudesh K Rao, Ramandeep Singh Brar, Tushar Kakkar. Orthokeratinized odontogenic cyst of the mandible: A case report. Int J Oral Maxillofac Pathol 2012;3(1):69-73.

ABOUT THE AUTHORS

Niharika Swain (Corresponding Author)

Senior Lecturer, Department of Oral Pathology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India Phone: +919324701036, e-mail: niharikadec30@gmail.com

Shilpa Patel

Professor and Head, Department of Oral Pathology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India

LS Poonja

Professor, Department of Oral Pathology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India

Jigna Pathak

Professor, Department of Oral Pathology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India

Kamlesh Dekate

Reader, Department of Oral Pathology, MGM Dental College and Hospital, Navi Mumbai, Maharashtra, India

Desmoplastic Ameloblastoma

Kamlesh Dekate[1], Niharika Swain[2], Jigna Pathak[3], L.S Poonja[4]

Abstract

According to the WHO (2005) classification of odontogenic tumors, Desmoplastic Ameloblastoma is recognized as a variant of ameloblastoma. This rare entity differs from the other forms of ameloblastoma in its anatomical location, morphology, and radiographic appearance. Due to its unusual clinic-pathological presentation, this tumor mimics various odontogenic as well as non odontogenic neoplasms. We are presenting a rare case of desmoplastic ameloblastoma in the maxilla in a 53 year old male with regards to its clinical and radiographical and histological viewpoints.

Keywords: Ameloblastoma, Desmoplastic ameloblastoma

Introduction

Ameloblastoma is a most common odontogenic tumor that usually exhibits aggressive behavior. It causes severe expansions of the cortical bones and may have high recurrence rate[1]. It may cause mobility and displacement of teeth as well as root resorption[2]. Follicular, plexiform, acanthomatous, desmoplastic are histological variants of ameloblastoma.[3] Desmoplastic ameloblastoma was first described by Eversole et al in 1984.[4] As compared the classical type of ameloblastoma ,this tumor exhibits differences in anatomical distribution, histological appearance and radiographic findings. Maxillary anterior region is a common site of tumor location. Radiographically, it appears as a mixed radiopaque/radiolucent lesion with soap bubble or honeycomb appearance. Histologically it shows pronounced desmoplasia containing epithelial islands, nests and cords.[5]

In this case report we have an unique opportunity to discuss a rare case of desmoplastic ameloblastoma along with its clinical, radiological, histological features and differential diagnoses.

Case Report

A 53 year old male patient reported to MGM Dental College and Hospital Kamothe with the chief complaint swelling in upper left anterior region of since last six month. Initially it was smaller in size and gradually increases to present size. On examination extra oral (Fig-1) diffuse swelling was present on left midface region. The borders of swelling were indistinct, overlying surface was normal skin and on palpation it is firm in consistency. Intra orally (Fig-2) swelling was present on the buccal and palatal aspects of maxillary left anterior region measuring approximately 2×1 cm and 3×2 cmrespectively. Radiographically (Fig-3) the orthopantomogram showed mixed radio-opaque and radio-lucent lesion with ill-defined borders extending from 22 to 26 with the roots of 24 and 25 is deflected distally.

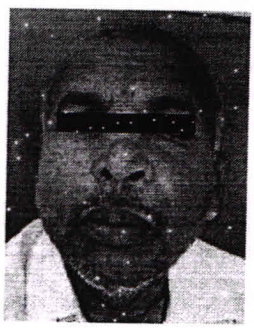


Fig1. Diffuse extra oral swelling on left maxilla.

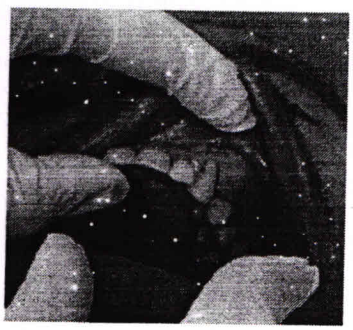


Fig 2. Intra orally tumor involving buccal and lingual side

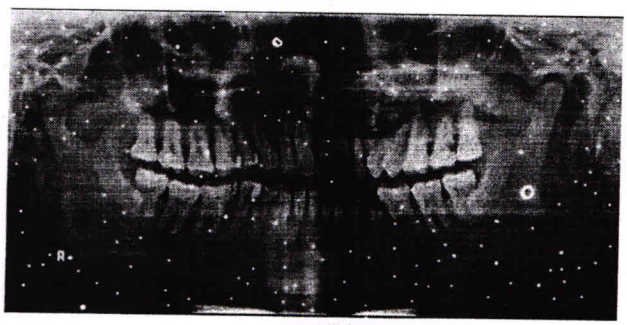


Fig3. mixed radioopaque/ radiolucent areas having ill defined border with root displacement of 24,25.

[1] Reader
[2] Lecturer
[3] Professor
[4] Professor

Department of Oral Pathology,
MGM Dental College and Hospital, Navi Mumbai.

Address for Correspondence :
Dr.Kamlesh Dekate
Dept. of Oral Pathology
MGM Dental College and Hospital, Navi Mumbai
Mobile-09223290372
Email- kamleshdekate@indiatimes.com

It is almost impossible to find the exact interface of the lesion with normal bone, making it especially difficult to be treated surgically.[19]

References

1. Neville BW, Damm DD, Alen Cm, Bouquot Je. Textbook of oral medicine and maxillofacial pathology(2nd ed) 2004;296.
2. Adebiyi K, Ugboko V, Esan G, Ndukwe K, Oginni F. clinicopathological analysis of histological varients of ameloblastoma in a suburban Nigerian population. Head and Face medicine 1006;2;42.
3. Santos J, Souza V, Azevedo R, Sarmento V, Souza L. case report. Hybrid lesion of desmoplastic and conventional ameloblastoma, immunohistochemical aspect. Rev Bras otorrinolaringology 2006;72(5):709-13
4. Thompson IOC, Van Rensburg LJ, Philips VMJ. Desmoplastic ameloblastoma. Correlative histopathology, radiology and CT-MR imaging. Oral Pathol Med 1996;25:405-10
5. Pillai R, Ongole R, Ahsan A, Radhakrishnan R, Pai K. recurrent Desmoplastic ameloblastomaof maxilla. A rare case report. J Can Dent Assoc 2004;70(2):100-04
6. Reichart PA, Philipsen HP. Odontogenic tumors and allied lesions. Quintessesnce Publishing Co. London p69-p77, 2004, 1 st edition.
7. Kawai T, Kishino M, Hiranuma H, Tadashi S, Ishida T. A unique case of desmoplastic ameloblastoma of the mandible; report of a case and brief review of English language literature. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999; 87: 258-263.
8. Mintz SH, Velez I. Desmoplastic variant of ameloblastoma: report of two cases and review of the literature. *JADA*. 2002; 133: 1072 – 1075.
9. Kaffe I, Buchner A, Taicher S. Radiologic features of desmoplastic variant of ameloblastoma. Oral Surg OralMed Oral Pathol. 1993; 76: 525 – 529.
10. Tanimoto K, Takata T, Suei Y, Wada T. A case of desmoplastic variant of mandibular ameloblastoma. *J Oral Maxillofac Surg*. 1991; 49: 94 – 97.
11. Waldron CA, El Mofty SK. A histopathologic study of 116 ameloblastomas with special reference to the desmoplastic variant. *Oral Surg Oral Med Oral Pathol*. 1987; 63: 441 – 451.
12. Saap JP, Eversole LR, Wysocki GP. *Contemporary Oral and Maxillofacial Pathology*. St. Louis: Mosby; 1997:131 – 132.
13. Beckley ML, Farhood V, Helfend LK, Alijanian A. Desmoplastic ameloblastoma of the mandible: a case report and review of the literature. *J Oral Maxillofac Surg* 2002; 60(2):194–8.
14. Philipsen HP, Ormistom IW, Reichart PA. The desmo- and osteoplastic amelobiastoma. Histologic variant or clinicopathologic entity? Case reports. *Int J Oral Maxillofac Surg* 1992; 21(6):352–7.
15. Lam KY, Chan AC, Wu PC, Chau KY, Tideman H, Wei W. Desmoplastic variant of ameloblastoma in Chinese patients. *Br J Oral Maxillofac Surg* 1998; 36(2):129–34.
16. Ng KH, Siar CH. Desmoplastic variant of ameloblastoma in Malaysians. *Br J Oral Maxillofac Surg* 1993; 31(5):299–303.
17. Waldron CA, el-Mofty SK. A histopathological study of 116 ameloblastomas with special reference to the desmoplastic variant. *Oral Surg Oral Med OralPathol* 1987; 63(4):441–51.
18. Nakamura N, Higuchi Y, Mitsuyasu T, Sandra F, Ohishi M. Comparison of long-term results from different approaches to ameloblastoma. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2002; 93(1):13–20.
19. Gardner DG. Some current concepts on the pathology of ameloblastomas. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1996;82(6):660–9.

Oral Surgery


Dr. Jigna Pathak
MDS, Professor


Dr. L. S. Poonja
MDS, Professor


Dr. Shwetha V. Kumar
MDS, Senior Lecturer


Dr. Niharika Swain
MDS, Senior Lecturer


Dr. Kamlesh Dekate
Reader

Correspondence Address
Dr. Jigna Pathak
Department Oral Pathology and Micro Biology
Mgm Dental College and Hospital,
Kamothe
Navi Mumbai

CONCOMITTANT OCCURRENCE OF AMELOBLASTIC FIBRO-ODONTOME AND COMPOUND ODONTOMA

Abstract

|| Brief Background
A case of concomitant occurrence of Ameloblastic fibro-Odontoma (AFO) and Compound Odontoma in a 29 year old male patient is presented here.

|| Materials and Methods
A male patient reported with a chief complaint of painless swelling on the right side of the lower jaw since 5-6 years. Radiographically a radio-opacity surrounded by a radiolucent area was seen in the mandibular right posterior region and also presence of small radio-opaque denticle like masses in relation to the impacted left maxillary canine. A provisional diagnosis of multiple odontomas was given. The histopathological examination revealed the lesion in the right mandibular posterior region to be Ameloblastic fibro-Odontoma and the denticles in the left maxillary canine region represented the Compound Odontoma.

|| Discussion
The discussion relates to the different theories of development and progression, clinical, radiographic and histopathologic presentation of Ameloblastic Fibro-Odontoma and Compound Odontoma.

|| Summary and Conclusions
This is probably the first report of a large AFO with concomitant occurrence of a Compound Odontoma. We hypothesize that the majority of AFOs may be neoplastic but some may also be hamartomatous.

|| Key Words
Odontogenic tumour, Ameloblastic Fibro-Odontoma, Compound Odontoma, Hamartoma.

9. Reichart P A, Philipsen O H, Odontogenic Tumours and allied lesions,1st edition. Quintessence Publication,USA,2004

10. Sivapathasundaram B, Manikandan R, Siva Kumar G, Goerge T. Ameloblastic Fibro-Odontoma. Indian J Dent Res 2005; 16:19-21.

11. Veda Hegde, S Hemavathy.A Massive Ameloblastic Fibro-Odontoma of the maxilla. Indian J Dent Res 2008; 19(2):162-64.

12. Morning P. Impacted teeth in relation to odontomas. International Journal of Oral Surgery 1980; 9:81-91.

13. Chen Y, Li T, Gao Y, Yu S.Ameloblastic Fibroma and related lesions: a clinicopathological study with reference to their nature and inter-relationship. J Oral Pathol Med 2005; 34:588-95.

14. Slootweg P J,. An analysis of the inter-relationship of the mixed odontogenic tumours-ameloblastic Fibroma, Ameloblastic fibro-Odontoma and the odontomas. Oral Surg Oral Med Oral Pathol 1981; 51:266-76.

15. Takeda Y. Ameloblastic Fibroma and related lesions. Current Pathologic Concept. Oral Oncol 1999; 35:535-40.

16. G F Favia, L di Alberti, A Scarano, A Piattelli. Ameloblastic Fibro-odontoma: Report of 2 cases. Oral Oncol 1997; 33(6) :444-446

Infiltrative Type of Bone Invasion in Oral Squamous Cell Carcinoma – Case Report

Jigna Pathak[1], Niharika Swain[2], Shwetha Kumar[3]

Abstract

Oral squamous cell carcinoma (OSCC) is a well known malignancy which accounts for more than 90% of all oral cancers. OSCC are malignant tumors that frequently invade bone and bone invasion is a common clinical problem. Bone invasion by oral squamous cell carcinoma may progress by either an infiltrative or an erosive histological pattern. The pattern of bone invasion co-relates with the clinical behavior of OSCC thus having a potential prognostic value. The present case report is of a 35-year- old female patient presenting with a lesion in the lower right buccal vestibule which was histopathologically confirmed as OSCC.The type of bony invasion was also assessed microscopically. The objective of this paper was to define the characteristics associated with each histological pattern of invasion and its significance when reviewing oral squamous cell carcinoma with mandibular invasion.

Key Words: Oral cancers, Osteoclastogenesis, Osteoprotegerin

Introduction

OSCC is the sixth most common cancer and more than 3, 00,000 new cases are diagnosed each year world wide.[1] Oral carcinoma of the mandibular region has been defined as carcinoma of the mandibular alveolar ridge, lower buccal sulcus, sublingual sulcus and mandibular retro molar trigone.[2] Carcinoma at this site may eventually progress to directly invade the mandible, a feature associated with a worse prognosis. Mandibular invasion is one criterion of the American Joint Committee on Cancer classification for the most advanced primary stage (T4) and overall stage (IV) for these tumors. The 5-year determinate survival of patients with stage IV oral lesions has been demonstrated to be 39%, as compared with 53%, 68%, and 70% for stages III, II, and I disease, respectively.[3]

OSCC invades the mandibular bone through an erosive, infiltrative or mixed pattern that correlates with clinical behavior. The erosive pattern is characterized by a broad, expansive tumor front with a sharp interface between tumor and bone. In contrast, the infiltrative pattern is composed of nests of tumor cells with fingerlike projections along an irregular tumor front. The recent distinction between these two histological patterns challenges the previously held assumption that mandible invasion universally presents a poor prognosis. The erosive pattern of bone invasion has been hypothesized to extend in a more predictable fashion than the infiltrative pattern. Infiltrative pattern of bone invasion is associated with a higher recurrence rate of about 53% compared with the erosive pattern which is about 17%.[4] The present case report describes about the infiltrative pattern of bony invasion by squamous cell carcinoma originating from the buccal vestibule in a middle aged woman.

Professor[1]

Senior Lecturer[2]

Senior Lecturer[3]

Department of Oral Pathology,
MGM Dental College and Hospital, Navi Mumbai

Address for Correspondence:
Dr. Jigna Pathak
501, Pleasant View Society, Plot 56/57
Sector-14, Vashi, Navi Mumbai-400703
Mobile: +919819175805
E mail: drjignapathak@gmail.com.

Case report

A 35-year-old female patient reported in MGM dental college and hospital with a chief complaint of a non healing cut in lower right cheek since the past 3-4 months. Past medical history was non contributory. The patient had habit of chewing tobacco since the past 20 years. She also had history of Misheri application on teeth and gums since the past 20-25 years. There was no history of trauma, sinus or pus discharge. Extraoral examination revealed a very mild facial deformity with a diffuse swelling in the right side of the face. Ipsilateral cervical lymphadenopathy (level IB) was also noticed. Intraorally there was presence of a linear endophytic lesion extending from lower right first premolar to lower right third molar region in the gingivo- buccal sulcus region.(Fig. 1) Additional feature i.e. Grade II mobility of teeth from mandibular right third molar to mandibular left canine was seen. On radiological examination, Orthopantamogram (OPG) revealed an ill-defined radiolucency extending from mandibular right third molar (48) to mandibular right canine (43). Computed tomography (C.T scan) showed an osteolytic lesion involving the right side of the mandible crossing the midline. (Fig.2) A provisional diagnosis of Squamous Cell Carcinoma involving the bone was given. Incisional biopsy was taken. The histopathological report of well differentiated SCC was

to be crossing the midline. This was initially suspected clinically as the teeth showed mobility from 48 to 33.

As tumor cells grow and mitosis increases, they invade the basement membrane, destroy the surrounding tissue regionally, resist the immune system, and secrete certain proteins and angiogenic factors that will facilitate lymphovascular invasion and metastasize regionally or distantly. OSCC tends to invade the adjacent bone due to its close anatomical proximity, so higher bone invasion will occur in the OSCC that lies in direct contact with the bone. The size and proximity of the primary tumor to the jaw bone will determine the degree of bone invasion. Prognosis is affected by the pattern of bone invasion, which could be either an erosive, infiltrative or mixed[7]. The erosive pattern of bone invasion is marked by a broad pushing front, a sharp interface between tumour and bone, osteoclastic bone resorption and fibrosis along the tumour front and an absence of bone islands within the tumour mass. In contrast, the infiltrative pattern is characterized by nests and projections of tumour cells along an irregular front, residual bone islands within the tumour and Haversian system penetration. The histological pattern of mandibular invasion seems to correlate with the clinical behavior. Infact the infiltrative lesions are more likely to have primary, regional and distant recurrence[8].The 3 year disease free survival in the infiltrative pattern is reported as 30% as against that in erosive pattern is 73%4.The present case showed an infiltrative pattern of bone invasion suggesting that it was an aggressive lesion. It is seen that cellular and molecular mechanisms regulate osteoclast differentiation.

Thus, it can be deduced that stromal cells regulate osteoclast formation induced by OSCC. Also IL-6 and PTHrP released from oral cancer cells induce Osteoclastogenesis through RANKL expression in stromal cells.[3]

Patients with mandibular invasion should be treated surgically but the extent of mandibular resection required remains controversial[5].Histological pattern of mandibular invasion has prognostic significance. Poor clinical outcome is highly correlated with the infiltrative histological pattern of invasion. Infiltrative pattern has a 4-fold increased risk of death as compared to erosive pattern. In the present case hemimandibulectomy was performed and considering a 1 cm safe margin, the mandible was resected up to Lower left first premolar.(34) However, on histopathology of the resected mandible, the margin considered to be safe showed infiltrative pattern of bone invasion, thus being positive for tumor. Therefore the patient was informed about the prognosis, advised radiotherapy and explained about the need of another surgical intervention.

A recent study has demonstrated that tumour invasion of the mandible is not significantly correlated with the survival of the patient with OSCC and if bone invasion is identified histologically, the prognosis is not worsened and additional surgery need not be undertaken.[9]

However, more studies are required with more number of cases to prove the prognostic value of the pattern of bony invasion in OSCC.

Conclusion

The infiltrative pattern intuitively appears to be an aggressive tumor that is difficult to resect surgically. The intraoperative and preoperative determination of invasion pattern remains problematic. If the preoperative imaging studies do show radiographic characteristics suggesting an infiltrative pattern such as an irregular front or bone spicules, a wide surgical margin should be taken around the grossly apparent tumor. Pattern of invasion provides important prognostic information and therefore should be routinely commented on by pathologists reviewing cases with mandibular bone invasion.

In addition, new approaches have been developed to examine cellular and molecular mechanisms of bone invasion by OSCC. Inhibition of osteoclast differentiation and function by blocking RANKL and RANK by inhibitor antibody constitutes a novel approach to development of target therapy.

References

1. Choi S, Myers J N .Molecular pathogenesis of oral squamous cell carcinoma: Implications for therapy. J Dent Res 2008:87;14-32.
2. Pandey M, Rao LP, Das S R , Mathews A, Chacko EM, Naik BR . Patterns of mandibular invasion in oral squamous cell carcinoma of the mandibular region. World J Surg Oncol. 2007; 5: 12.
3. Shah J, Lydiatt WM. Buccal mucosa, alveolus, retromolar trigone, floor of mouth, hard palate, and tongue tumors. In: Thawley SE, ed. Comprehensive Management of Head and Neck Tumors.II Ed. Philadelphia: WB Saunders;1999: 686-693.
4. Wong R J,. Keel SB, Glynn RJ ,. Varvares MA. Histological Pattern of Mandibular Invasion by Oral Squamous Cell Carcinoma .The Laryngoscope. January 2000;110(1):65-72.
5. Warnakulasurya S. Living with oral cancer: epidemiology with particular reference to prevalence and life style changes that influences survival.Oral Oncology 2010;45:407-10
6. Vidiri et al. MDCT and MRI in the evaluation of mandibular invasion by OSCC. Correlation with pathological data. Journal of Experimental & Clinical Cancer Research 2010, 29:73
7. Alkindi Mohammed.Effects of soluble factors released by OSCC on osteoclasts. M.Sc thesis February 2011.McGill University,Montreal,Canada.
8. Jimi, H.Furuta, K.Matsuo, K.Tominaga, T.Takahashi, O.Nakanishi. The cellular and molecular mechanisms of bone invasion by Oral Squamous Cell Carcinoma. Review article. Oral Diseases 2011;17:462-468.
9. Mücke T , Hölzle F , Wagenpfeil S , Wolff K , Kesting M. The role of tumor invasion into the mandible of oral squamous cell carcinoma.J Cancer Res Clin Oncol 2011;137:165-171.

3 copies

International Journal of Oral & Maxillofacial Pathology, 2011;2(2):31-34
Available online at [illegible]
ISSN 2229 – 2241

Case Report

Plasmablastic Lymphoma of Gingiva as Primary Oral Manifestation in Previously Undiagnosed HIV Patient - A Case Report

Kamlesh N. Dekate, Vineet Kini, Shwetha V. Kumar, Jigna Pathak, Leela Poonja

Abstract

Non-Hodgkin's lymphomas are the third most common group of malignant lesions in the oral cavity and maxillofacial region. Most such lymphomas have been shown to be predominantly of B- cell lineage. Plasmablastic lymphoma of the oral cavity is an aggressive B-cell lymphoma associated with Human Immunodeficiency Virus infection and is classified as an individual nosological entity by the World Health Organization Classification of Tumours of Hematopoietic and Lymphoid Tissues. It clinically presents with a rapid growth and histologically shows a diffuse pattern with a high mitotic index. Based solely on clinical and microscopic features, separation of Plasmablastic lymphoma from other categories of Non-Hodgkin's lymphoma is very difficult. Therefore demonstration of distinguishing pattern of expression of immunohistochemical markers is an essential component of the diagnostic protocol. Hence we report a case of Plasmablastic Lymphoma in a healthy person with previously undiagnosed Human Immunodeficiency Virus.

Key words: Malignant Lymphoma; Immunoblastic; AIDS-Related; Non-Hodgkin's lymphoma; HIV; Immunohistochemistry; CD138.

Kamlesh N. Dekate, Vineet Kini, Shwetha V. Kumar, Jigna Pathak, Leela Poonja. Plasmablastic Lymphoma of Gingiva as Primary Oral Manifestation in Previously Undiagnosed HIV Patient – A Case Report. International Journal of Oral & Maxillofacial Pathology; 2011;2(2):31-34.

Received on: 14/03/2011 Accepted on: 11/06/2011

Introduction

The development of lymphoma in patients with immune dysregulation, induced by the Human Immunodeficiency Virus (HIV) is fully consistent with the known occurrence of lymphoma in other setting of immunologic compromise[1]. Non-Hodgkin's lymphoma (NHL) represents the most common HIV-associated malignancy, occurring in HIV-infected individuals at 60 times the frequency experienced in an otherwise healthy population.[2]

With the constellation of Acquired Immunodeficiency Syndrome (AIDS) related Non Hodgkin's -Lymphoma, four distinct types are AIDS Burkitt's Lymphoma, Diffuse Large Cell Lymphoma, Immunoplasmacytoid Lymphoma and Primary Effusion Lymphoma.[3,4] Plasmablastic Lymphoma (PBL) represent a recently categorized sub type of HIV-related Diffuse Large B Cell Lymphoma (DLBCL) based on its blastic morphology, with a marked predilection for oral cavity.[5]

The present case report describes an interesting case of solitary intraoral Plasmablastic lymphoma in an apparently healthy individual who was later diagnosed seropositive for HIV infection. Lymphoma is considered to be a relatively late manifestation of HIV infection[1] but in the present case Plasmablastic lymphoma was the only clinical oral manifestation.

Case Report

A 38 year old male patient reported to the Department of Oral Pathology, MGM Dental College and Hospital, with a chief complaint of painless swelling of gingiva on mandibular anterior region since one and half months. He had no other signs and symptoms before the onset of swelling that gradually grown up to present size.

Intra-orally an exophytic lobulated mass (Fig 1) measuring approximately 3x2 cm in size was present on the anterior region of mandibular gingiva. On palpation the swelling was soft in consistency, and not fixed to the underlying bone. Extra orally, neither facial asymmetry nor cervical lymphadenopathy was observed. Radiographic examination revealed vertical bone loss till the apex in between mandibular left central incisor and mandibular left lateral incisor. The differential diagnosis of pyogenic granuloma, peripheral giant cell granuloma and squamous cell carcinoma were considered.

Histopathologic examination of an excisional biopsy (Fig 2) revealed Parakeratinized stratified squamous epithelium at one place. The underlying connective tissue showed a

ISSN 2231 - 2250

Acknowledgement
We sincerely thanks Dr. Mukta Ramdwar, Tata Memorial Hospital, for her kind support in immunohistochemical analysis and Dr. Sheetal Awachar for her valuable guidance.

References

1. Levine AM. Acquired immunodeficiency Syndrome related lymphoma. Blood 1992;80(11):8-20.
2. Scheper MA, Nikitakis NG, Fernades R. Oral Plasmablastic Lymphoma in an HIV- negative patient: A report and review of literature. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;100(2):198-206.
3. Radhakrishnan R, Suhas S, Kumar RV. Plasmablastic Lymphoma of the oral cavity in an HIV-positive child. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;100(6):725-31.
4. Delecluse HJ, Anagnostopoulos I, Dallenbach F. Plasmablastic Lymphoma of the oral cavity: a new entity associated with the Human Immunodeficiency Virus infection. Blood 1997;89(4):1413-20.
5. Chang CC, Zhou X, Taylor JJ, Huang WT, Ren X, Monzon F, et al. Genomic proliferation of plasmablastic lymphoma using array comparative genomic hybridization (aCGH): revealing significant overlapping genomic lesions with diffuse large B cell lymphoma. J Hemat Oncol 2009;2:47-52.
6. Desai RS, Vanaki SS, Puranik RS. Plasmablastic Lymphoma presenting as a gingival growth in a previously undiagnosed HIV-positive patient: A case report. J Oral Maxillofac Surg 2007;65(7):1358-61.
7. Sarode SC, Zarkar GA, Desai RS. Plasmablastic lymphoma of the oral cavity in an HIV- positive patient: A case report and review of literature. Int J Oral Maxillofac Surg 2009;38(9):993-9.
8. Dupin N, Diss TL, Kellam P. HIV-8 is associated with plasmablastic variant of Castleman disease that is linked to HIV - 8 positive plasmablastic lymphoma. Blood 2000;95(4):1406-12.
9. Teruya-Feldstain J, Chiao E. CD20-negative large-cell lymphoma with plasmablastic features: A clinically heterogenous spectrum in both HIV-positive and negative patients: Med Ann Oncol 2004;15:1673-9.
10. Armstrong R, Bradrick J. Spontaneous regression of an HIV-associated Plasmablastic lymphoma in the oral cavity: A case report J Oral Maxillofac Surg 2007;65:1361-64.
11. Barkhuysen R, Merlx AW, Weijs WL. Plasmablastic Lymphoma mimicking orbital cellulitis. J Oral Maxillofac Surg 2008;12(3):125-128.

Author Correspondence
Dr. Kamlesh N. Dekate
Department of Oral Pathology,
Mahatma Gandhi Mission's Dental College,
Sector- 16, Kamothe, Navi Mumbai, India.
Ph: +91 9223290372
E-mail: kamleshdekate@indiatimes.com

Source of Support: Nil, Conflict of Interest: None Declared.

Diagnostic Aids in Screening of Oral Cancer

Jigna Pathak*, LS Poonja**

Abstract

Historically, the screening of patients for signs of oral cancer and precancerous lesions has relied upon the conventional oral examination. A variety of commercial diagnostic aids and adjunctive techniques are available to potentially assist in the screening of healthy patients for evidence of otherwise occult cancerous change or to access the biologic potential of clinically abnormal mucosal lesions.

With early detection and timely treatment, deaths from oral cancer could be dramatically reduced. The 5 year survival rate for those with localized disease at diagnosis is 82% compared with only 28% for those cancers which have spread to other parts of the body. Because the 5 year survival rate is directly related to the stage of malignancy at the time of diagnosis, prevention and early detection are vital to decrease the incidence and improve the survival odds of individuals who develop the disease. This manuscript thus examines the literature associated with current oral cancer screening.

Key Words : Screening, Prevention, Early detection, Survival rate.

Introduction

A dentist who examines the mouth of patients, on occasion detects a change in the oral mucosa. The question is to decide if this abnormality requires further investigation. If the answer is yes, there should be a systematic approach to the evaluation of the lesion which includes a methodical gathering of background information and a step by step clinical examination.[1]

A methodical approach is important given that many mucosal conditions have a similar appearance. Early detection of oral cancer is often possible. The WHO has identified prevention and early detection as major objectives in the control of the oral cancer burden worldwide.

Simple visual examination, however, is well known to be limited by subjective interpretation. As a consequence, Adjunctive Techniques have been suggested to increase our ability to differentiate between benign and malignant and also to identify areas of dysplasias and early oral squamous cell carcinomas that are not visible to the naked eye.[2]

Adjunctive Diagnostic Aids[2]

1. Toulidine Blue staining - Vital Tissue Staining.
2. Chemiluminescence - Visualization Adjuncts
3. Tissue Fluorescence Imaging - Visualization Adjuncts
4. Tissue Fluorescence spectroscopy - Visualization Adjuncts
5. ORAL CDX - Cytopathology
6. Biopsy - Cytopathology

*Reader; **Dean, Professor and Head; Department of Oral Pathology, MGM Dental College and Hospital, Kamothe, Navi Mumbai.

Toulidine Blue Staining

Tolonium Chloride, more commonly referred to as Toulidine Blue [TB], has been used for more than 40 years to aid in detection of mucosal abnormalities of the cervix and the oral cavity. It is a metachromatic vital dye that may bind preferrentially to tissues undergoing rapid cell division.[4] It is a dye of the thiazine group that has been effectively used as a nuclear stain because of its binding to DNA. The most probable explanation of binding in vivo by precancerous and cancerous tissue is the immediate binding by sulphated mucopolysaccharides, which are found in higher quantites in tissues that are actively growing, such as tumours and tissues that are healing (Figs. 1-5).[5]

Limitations

1. Absence of randomized control trials.
2. Absence of histological diagnosis as a gold standard.
3. Variability in methods of application.
4. Although good at detecting carcinomas, its sensitivity in detecting dysplasias is significantly lower.
5. High percentage of false positive stains.

Chemiluminescence[3]

Clinical inspection of oral mucosa can be done with the aid of Chemiluminescent Blue/White Light. The relevant technology [Vizilite System, Zila Pharma] involves the use of an oral rinse with a 1% acetic acid solution for 1 minute followed by the examination of the

Tissue fluorescence in the oral cavity is variable and is affected by structural changes, metabolic activity, presence of hemoglobin in the tissue, vessel dilation and possibly inflammation.[4]

It is highly sensitive and specific in assessing the lesion margins but is expensive.

Tissue Fluorescence Spectroscopy[3]

It consists of small optical fibre that produces various excitation wavelengths and a spectrograph that receives and records on a computer and analyzes via a dedicated software, the spectra of reflected fluorescence from the tissue.

Advantages

1. Eliminates subjective interpretation.
2. High sensitivity and specificity.

Limitations

1. Cannot distinguish different types of lesions.
2. Optical fibre can sample only small mucosal areas.
3. Expensive

Brush Biopsy[3]

Oral Brush Biopsy/Oral CDX Brush Test System, consists of a method of collecting Trans-epithelial Sample of cells from a mucosal lesion with representation of the Superficial, Intermediate and Parabasal/basal layers of the epithelium (Fig. 8).

This was specially designed to investigate mucosal abnormalities that would otherwise not be subjected to biopsy because of low risk clinical features. Given the difficulty in clinically differentiating premalignant and malignant lesions from benign lesions with a similar appearance, Oral CDx appears to determine the significance of oral lesions definitely and detect innocuous appearing oral cancers at early, curable stages.

Oral CDx kit consists of: oral brush biopsy instrument, a precoded glass slide, matching coded test requisition form, an alcohol/polyeythelene glycol fixative pouch and a pre-addressed container.

Method

Depending on the lesion's intraoral location and accessibility, either the flat surface or circular border of the brush was placed against the surface of the lesion and while firm pressure is maintained, the brush is rotated 5-10times. Pinkness of tissue or pinpoint bleeding at the brush biopsy site is evidence of proper technique. Neither topical nor local anaesthetic is used. The cellular material collected is then transferred to the bar coded glass slide an rapidly flooded with the fixative to avoid drying. After 15 minutes, the dry slide is placed in the plastic container and sent with bar coded requisition form in the pre-addressed container. All oral CDx specimens are analyzed at Orascan Laboratories in Suffern, New York. The slides are stained with modified PAP (Fig. 9).[3]

The Specimens are categorized in one of the four categories:

Negative = no epithelial abnormality.

Atypical = abnormal epithelial changes of uncertain diagnostic significance.

Positive = definitive cellular evidence of epithelial dysplasia or carcinoma.

Inadequate = incomplete transepithelial biopsy specimens.

Advantages

1. High Sensitivity and Specificity with no subjective interpretation.
2. Easy to perform
3. Simple armamentarium
4. Relatively inexpensive
5. No local anesthesia required

Conclusion

Oral cancer, the sixth most common cancer, accounts for about 3.6% of all cancers diagnosed. Early evaluation of oral precancerous and early stage cancerous lesions can have a dramatic impact on oral cancer mortality rates. Precancers and early stage oral cancers cannot be adequately identified by visual inspection alone and easily may be overlooked and neglected. As a consequence, adjunctive techniques have been suggested to increase our ability to differentiate between benign abnormalities and dysplastic/malignant changes as well as identify areas of dysplasia/early oral squamous cell carcinoma that are not visible to naked eye.

Oral CDx could provide invaluable assistance to clinicians in determining the significance of an oral lesion while examining the oral cavity. Although it doesnot substitute for a scalpel biopsy, it identifies oral lesions that require histologic evaluation since scalpel biopsy is an invasive procedure associated with potential morbidity. Tissue Biopsy and Histological examination remains the gold standard diagnostic test for oral mucosal lesions.

References

1. Michele Williams P, *et al*. Evaluation of a suspicious oral mucosal lesion. *JCDA* 2008; 74(3) : 275.
2. Stefano Fedele. Diagnostic aids in the screening of oral cancer. *Head and Neck Oncology* 2009; 1(5).
3. Scuibba James J. Improving detection of precancerous and cancerous oral lesions. Computer – Assisted Analysis of the Oral Brush Biopsy. *JADA* 1999; 130 : 1445-58.
4. Patton Lauren L, *et al*. Adjunctive techniques for Oral Cancer examination and lesion diagnosis.A systematic Review of literature. *JADA* 2008; 139 (7) : 896-905.
5. Silverman Sol Jr. Oral Cancer. American Cancer Society. 1998 : 51-55.

ORAL SUBMUCOUS FIBROSIS REVISITED

About the author

Dr. Jigna Pathak, M.D.S, Oral Pathology, Professor, M.G.M Dental College and Hospital, Kamothe, Navi Mumbai

INTRODUCTION

Oral Sub mucous Fibrosis (OSMF) is a chronic debilitating disease and a potentially malignant disorder of the oral cavity. In ancient medicine,Shushruta's MOUTH AND THROAT DISEASES mentioned about a condition called VIDARI the features of which simulate with that of OSMF.Schwartz in 1952 was the first person to describe OSMF as a fibrosing condition in 5 Indian women from East Africa and called it ATROPHICA IDIOPATHICA MUCOSAE ORIS.In 1953, JOSHI suggested the name ORAL SUBMUCOUS FIROSIS which is most widely accepted today.This disease shows classic clinical features and despite the availability of the current treatment modalities, unfortunately ,none of them produce satisfactory results.

DEFINITION

Pindborg and Sirsat (1966) defined OSMF as an insidious chronic disease affecting any part of the oral cavity and sometimes the pharynx. Although occasionally preceded by and / or associated with vesicle formation , it is always associated with juxtaepithelial inflammatory reaction followed by fibro elastic change of the lamina propria with epithelial atrophy leading to stiffness of the oral mucosa and causing trismus and inability to eat.[1]

WHO in 1978 defined OSMF as a slowly progressive disease in which fibrous bands form a blanched oral mucosa, resulting in severe restriction of movement of the jaw.

CLINICAL MANIFESTATIONS

It is a chronic disease of insidious onset featuring deposition of fibrous tissue in the sub mucosal layers. OSMF commonly affects the buccal mucosa, labial mucosa ,soft palate ,palatal fauces ,uvula and tongue. The underlying muscles of mastication maybe affected .The earliest clinical sign of OSMF is blanching of the oral mucosa. This blanching imparts a marble-like appearance to the oral mucosa which can be localized, diffuse, or in a lace-like network. Patients frequently present with a history of intolerance to spicy food together with a progressive reduction in oral opening.

OSMF is characterized by excessive collagen production by mucosal fibroblasts and is associated with the habitual chewing of betel nut / areca nut.The primary presenting feature is the limitation of the oral opening which often results in difficulty in eating. Attempts to improve the oral opening by medical means or surgical means, may exacerbate the disease by increased scarring.[3]

SIGNS:[1,6]

1. Palate and Faucial Pillars:

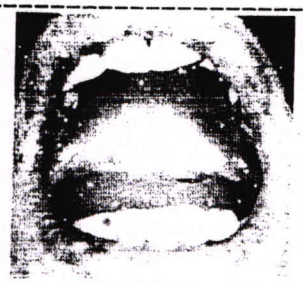


fig 1. vesicle on the soft palate

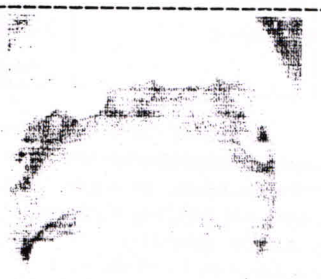


fig 2. Blanching of soft palate

It begins with VESICLES on the palate Fig.1. The vesicles usually form after the consumption of spicy food suggesting possibility of allergic reaction to Capsaicin. Mucosa is blanched; slightly opaque white Fig.2.

2. Buccal Mucosa:

Cheeks become thick, rigid, firm and whitish. Blanching is caused by impairment of local vascularity because of increased fibrosis causing MARBLE-LIKE appearance Fig.3. Firm vertical bands can be felt in the regions opposite the premolars.

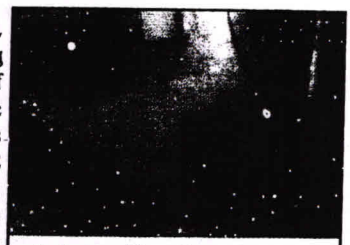


fig 3 . blanching of buccal mucosa

3. Tongue:

ATROPHY of the PAPILLAE Fig.4. Difficulty in PROTRUDING the tongue Fig.5.

fig 4. depapillation and fibrosis of the tongue

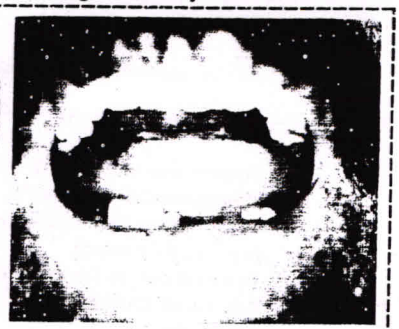


fig 5 . difficulty in protruding the tongue.

4. Uvula:

If fibrosis extends posteriorly, the uvula appears SHRUNKEN Nd in extreme cases BUD-LIKE Fig.6.

fig 6 . atrophic uvula

5. Lips:

Fibrous bands in the lips make the lip thick, rubbery and difficult to Retract / Evert. Bands around the lips give the mouth an Elliptical Appearance Fig.7.

fig 7 .blanching of labial mucosa

6. Gingiva:

Gingival involvement is UNCOMMON. Maybe characterized by fibrosis, blanching and loss of Stippling.

SYMPTOMS:[1,6]

PRODROMAL :Burning Sensation of the Oral Mucosa.
: Inability to eat spicy food.
: Sometimes Stomatitis.

LATER: Stiffening of certain areas of the oral mucosa.
: Inability to protrude the tongue Fig. 5.
: Inability to whistle or blow Fig. 8.

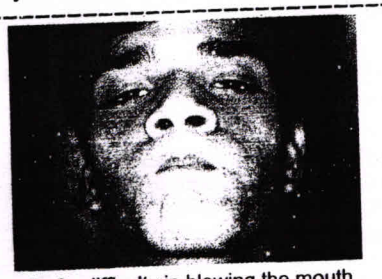


fig 8 . difficulty in blowing the mouth

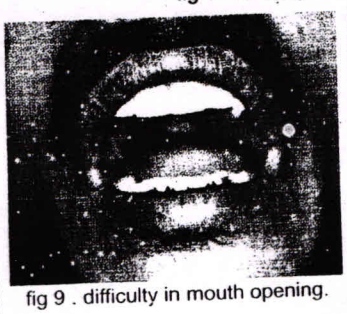


fig 9 . difficulty in mouth opening.

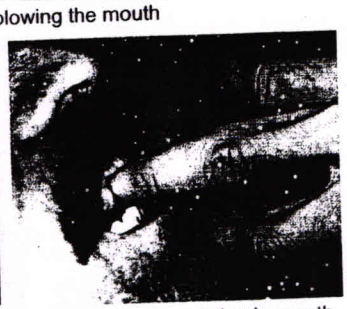


fig 10 . severe restriction in mouth opening.(Only 1 finger space)

: Trismus due to fibrosis of mucosa behind the molars and the pterygomandibular raphe Fig. 9, 10.
: Difficulty in swallowing because of esophageal fibrosis.
: Dryness of the mouth.
: Referred pain in the ears due to blockage of the Eustachian Tube due to fibrosis.
: Nasal Voice.
: Difficulty in maintaining oral hygiene.

CLINICAL STAGING

There are 4 stages described by Gupta et al in 1980 depending upon the increasing intensity of trismus.[5]

1. VERY EARLY STAGE: Burning sensation in the mouth without difficulty in opening the mouth.
2. EARLY STAGE: Along with the symptoms, there is slight difficulty in opening the mouth.
3. MODERATELY ADVANCED STAGE: There is marked trismus, where patient cannot open his mouth more than 2 fingers. There is difficulty in mastication.
4. ADVANCED STAGE: Patient is undernourished, anemic with marked degree of trismus and other associated symptoms of OSMF.

ETIOPATHOGENESIS

The etiology of the disease is still obscure but a multifactorial pathogenesis has been suggested. The strongest risk factor is the chewing of BETEL QUID containing ARECA NUT. QUID has been defined as a substance or mixture of substances, placed in the mouth or chewed and remaining in contact with the mucosa, usually containing one or both of the two basic ingredients, tobacco and / or areca nut, in raw or any manufactured or processed form. In most areas, BETEL QUID (BQ) / PAN , consists of a mixture of areca nut/ betel nut, slaked lime, catechu and several condiments according to taste, wrapped in a betel leaf.[7] Although there are regional variations in the type of areca nut products used in India, the Betel Quid was considered the most popular and prevalent habit in ancient Indian culture. In 1980, several products like PAN MASALA / ARECA QUID = Areca nut, catechu, lime, flavors and spices and GUTKHA = Areca quid +Tobacco were introduced in the Indian market as commercial products. These are a commercial substitute to a local preparation called KHARRA / MAWA. Since then, there is an increased use of PAN MASALA and GUTKHA in the younger age group which leads to the increased incidence of OSMF.[8]

Areca nut is the endosperm of the fruit of the Areca Catechu tree. There are many reasons for chewing Betel: It causes Euphoria, increases salivation, satisfies hunger, relieves tooth pain, etc. The major areca nut alkaloid is arecoline and major flavonoid is tannin. These alkaloids undergo nitrosation and gives rise to N-nitrosamines, which might have a cytotoxic effect on cells. Arecoline is shown to promote collagen synthesis and also there is reduced collagen degradation. Thus OSMF is now considered a collagen metabolic disorder.[6,7]

TREATMENT

There is no definitive treatment of OSMF which results in complete cure.Currently, intralesional steroids are the main treatment modality. These are injected into the fibrotic bands weekly for 6-8 weeks with regular monitoring of mouth opening. Patients are advised to do mouth opening excercises,eg: by placing ice-cream sticks in their mouth and gradually increasing the number or using a Jaw opener. Hyaluronidase, which facilitates the breakdown of connective tissue, can be combined with the steroids for injection. The list of other treatment modalities is enlisted in Table 1.

Table 1:Treatment modalities for OSMF

TREATMENT	TREATMENT DETAILS
Micronutrients & Minerals	Vit A, B, D, E, Iron, copper ,calcium, zinc, magnesium ,selenium
Milk from immunized cows	45g milk powder bid x3 months.
Lycopene	8mg bid x 2 months
Pentoxyfilline	400mg tid x 7 months.
Interferon gamma	Intralesional injection (0.01-10 U/ml) Tid x 6 months.
Steroids	Sub mucosal injections bid at multiple sites x 3 months
Steroids	Topical for 3 months.
Hyalase + Dexamethasone	
Placental Extracts	
Turmeric	Alcoholic extracts of turmeric (3g), turmeric oil (600mg), and turmeric oleoresin (600mg) daily for 3 m.
Chymotrypsin, Hyaluronidase Dexamethasone	Chymotrypsin (5000IU), Hyaluronidase (1500 IU), and Dexamethasone (4mg), twice weekly X 10 weeks

As fibrosis cannot be reversed, when mouth opening is severely limited, surgical interventions like myotomy, coronoidectomy and excision of fibrotic bands, are required. Reconstruction using such techniques as buccal pad flap, superficial temporal flap and forearm flap can also be performed. Alternative procedures, such as insertion of an oral stent, physiotherapy, local heat therapy, mouth exercises using acrylic carrots and ice-cream sticks, have been tried with variable rates of success.

In most cases, depending on the stage of disease and extent of oral involvement, therapy consisting of a combination of the above mentioned drugs and surgery might be useful.

DIFFERENTIAL DIAGNOSIS OF ORAL ULCERS

Dr. Jigna Pathak | Dr. L. S. Poonja

INTRODUCTION:

Although ulcerations of the oral mucosa are commonly seen in dental practice, majority of these heal within a week or two with or without treatment. A small percentage of ulcers however do not heal despite routine treatment or elimination of the cause. These type of ulcerative lesions should be viewed with high degree of suspicion because of the possibility that these may carry the risk of being malignant or could be persistent due to some autoimmune/systemic disorder which maybe potentially fatal.

Thus an attempt is made in the present paper to discuss the various causes, clinical features and important diagnostic criteria of various commonly occurring or recurring, transient or persistent, solitary or multiple ulcerations. Since most oral ulcers have similar clinical appearance and are manifestations of a spectrum of conditions, some common differentiating points are briefly discussed here which may prove to hold value in clinical practice as ulcerations can pose a definite diagnostic challenge for a dental practitioner.

HISTORY TAKING AND CLINICAL EXAMINATION:

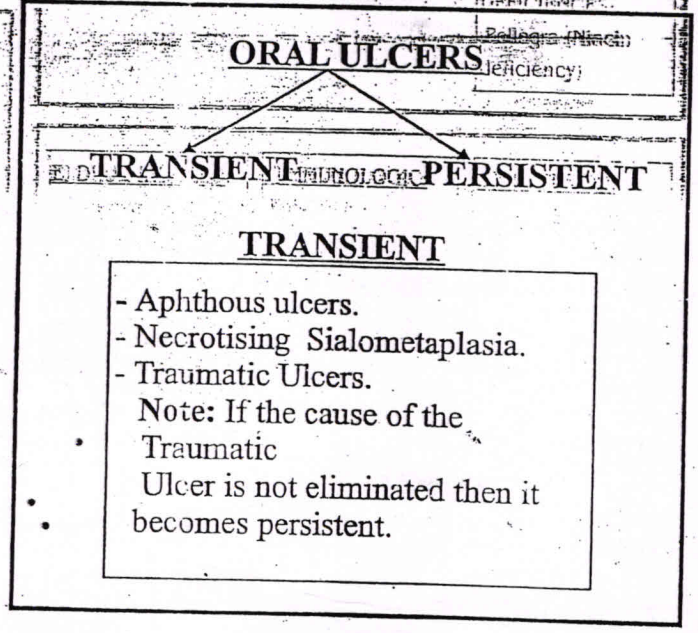
History, thorough clinical examination and appropriate investigations must be carried out to arrive at a diagnosis of ulcerative lesions.

History taking includes:

-Onset, Duration, Etiology, Recurrence, Progression, Developments which precede the ulcers (eg: vesicles/ bullae), Simultaneous occurrence on various other body parts, Associated symptoms like fever, malaise, enlargement of lymph nodes, History of taking drugs and medications.

Important Aspects of Clinical Examination includes:

-Anatomical location, Number, Size, Shape, Colour, Margins, Base, Edges, Texture, Tenderness, Lymph node involvement, involvement of other systems of the body, eg: Skin, Eyes, Genitals.



PERSISTENT:

A] INFECTIONS:

BACTERIAL:	FUNGAL:	VIRAL:
-Syphilis.	-Histoplasmosis.	-Herpes Simplex.
-Tuberculosis.	-Mucormycosis.	-Herpes zoster.
-Actinomycosis.	-Cryptococcosis.	-Chickenpox.
-Noma.	-Blastomycosis.	

B] NON-INFECTIOUS GRANULOMATOSIS:
-Wegner's Granulomatosis.
-Midline lethal granuloma.
-Sarcoidosis.

C] MUCOCUTANEOUS DISORDERS:
-Pemphigus.
-Pemphigoid.
-Erythema Multiforme/Steven-Johnson Syndrome.
-Erosive Lichen Planus.
-Toxic Epidermal Necrolysis.

D] SECONDARY TO SYSTEMIC DISORDERS:
1. HEMATOLOGICAL:
-Leukemia.
-Agranulocytosis.
-Cyclic Neutropenia.
2. METABOLIC:
-Diabetes Mellitus.
-Uremia.
-Histiocytosis X.
-Ulcerative Colitis.
3. NUTRITIONAL DEFICIENCES:
-Pellagra (Niacin deficiency)

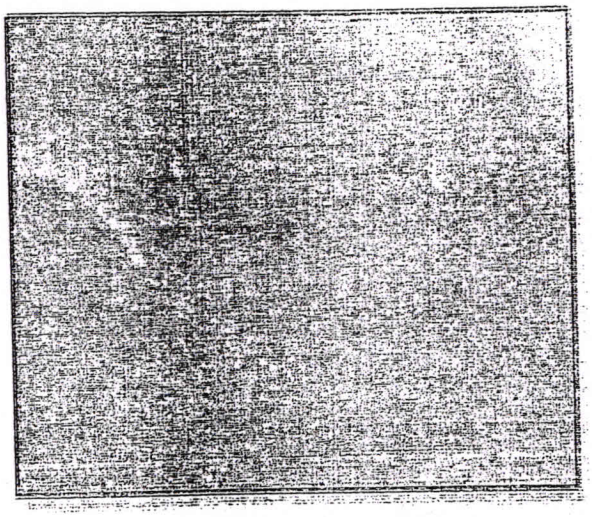
E] DRUG INDUCED:
-Lichenoid Drug reaction.
-Stomatitis Medicamentosa.
-Stomatitis Venenata.
-Midline lethal granuloma.
-Sarcoidosis.

F]IMMUNOLOGIC:
-Reiter's Syndrome.
-Bechet's Syndrome.

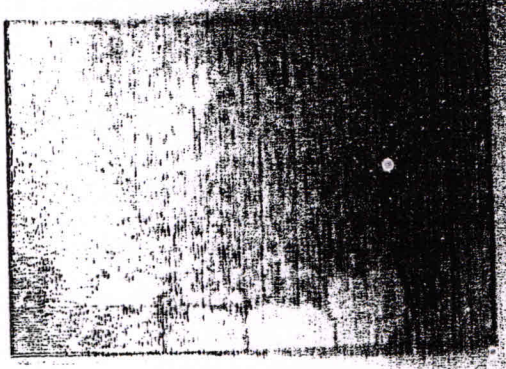
G]NEOPLASMS:
-Squamous cell Carcinoma.
-Verrucous Carcinoma.
-Kaposi's Sarcoma.
-Lymphoma.
-Salivary Gland Neoplasms-
-Mucoepidermoid carcinoma.
-Adenoid Cystic Carcinoma.
-Melanoma.
-Metastatic Tumour.

3. EROSIVE LICHEN PLANUS

-Autoimmune Mucocutaneous disease showing ulcers in the mouth with Wickham's striae at the periphery.
Buccal mucosa mostly affected.



Lateral border of the tongue



SQUAMOUS CELL CARCINOMA

-The most common neoplasm of the oral cavity
-It may present as a painless, persistent, non healing ulcer with indurated rolled out margins and fixed to the underlying tissues.

About the author

DR. JIGNA PATHAK
M.D.S. (ORAL PATHOLOGY AND MICROBIOLOGY),
READER, M.G.M. DENTAL COLLEGE AND HOSPITAL,
KAMOTHE, NAVI MUMBAI.

DR. L.S. POONJA
M.D.S. (ORAL PATHOLOGY AND MICROBIOLOGY),
DEAN, PROFESSOR AND HEAD OF DEPARTMENT,
M.G.M. DENTAL COLLEGE AND HOSPITAL, KAMOTHE,
NAVI MUMBAI.

महाराष्ट्र आरोग्य विज्ञान विद्यापीठ
MAHARASHTRA UNIVERSITY OF HEALTH SCIENCES
वणी रोड, म्हसरुळ, नाशिक - ४२२ ००४
Vani Road, Mhasrul, Nashik – 422 004
Vidya Thakare
Dy. Registrar
Phone: 0253-2539199/239 / EPABX: 0253-2539100 - 300 / Fax: 0253-2539200
E-mail: pgacademic@muhsnashik.com / Web: www.muhsnashik.com

No. MUHS/ PG/E-2/ PGTRC/ 291 /2012 Date: 30/01/2012

To
The Dean / Principal,
MGM's Dental College & Hospital
Sector–18, Kamothe,
Navi Mumbai – 410 20

Sub:- **Recognition as Post-Graduate Teacher's**

1) Your college letter No. MGM/DCH/892/2011 dt. 05/11/2011
2) Your college letter No. MGM/DCH/910/2011 dt. 15/11/2011
3) University letter no. MUHS/PG/E-2/PGTRC/2879/2011 dt. 08/12/11
4) PGTRC meeting dated 16/12/2011
5) Your college letter No. MGM/DCH/1000/2011 dt. 13/12/2011
6) University letter No. MUHS/MET,Pune/150/2012 dt. 27/01/2012

Sir/Madam,

With reference to the above cited subject, I am directed to inform you that in view of the norms prescribed as per provision under the section 29 (2) (l) of the MUHS Act, 1998 Hon'ble Vice-Chancellor is pleased to grant recognition as Post-Graduate Teacher to the following teacher(s) of your College **subject to the terms and conditions of appointment order for imparting instructions** to the Post Graduate Degree in the subject mentioned against their name.

Sr. No.	Name of the Teacher	Subject	Status of PG Recognition
01	Dr. Sabita M. Ram	Prosthodontics & Crown & Bridge	w.e.f. 16/12/2011
02	Dr Nadgere Jyoti B	Prosthodontics & Crown & Bridge	w.e.f. 16/12/2011
03	Dr Sonawane Smita R	Oral & Maxillofacial Surgery	w.e.f. 16/12/2011
04	Dr Pathak Jigna R	Oral Pathology & Microbiology	w.e.f. 16/12/2011

Kindly note that the recognition given by the University is valid till the above said teacher(s) are in services of the private College or attains the age of superannuation whichever is earlier as per the rules made by the University from time to time.

You are requested to handover the copy of letter to the concerned teacher(s).

Yours faithfully,

Dy. Registrar
I/C Academic Section (PG)

Copy to :
1) The Controller of Examinations, MUHS, Nashik
2) The Synopsis Section, MUHS, Nashik

[Note: **In case, if it is found at later stage that information furnished in Post Graduate Recognition form by any teacher is incorrect, PG Recognition granted by the University will stand cancelled.]**

Dean
M. G. M. Dental College & Hospital
Kamothe, Navi Mumbai - 410 209.

Dean
M. G. M. Dental College & Hospital
Kamothe, Navi Mumbai - 410 209.