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HISTOPATHOLOGICAL PATTERN OF ORAL CAVITY AND JAW TUMOURS.

Pathology

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ABSTRACT

Background : Oral cavity and jaw lesions, include a wide variety of lesions including both benign and malignant lesions.Tobacco chewing, ill fitting dentures, cigarette smoking are the predisposing factors. These lesions potentially endanger health of the individual. We have undertaken this study to correlate histopathological changes with clinical findings.

Material and methods: This retrospective study was done in Department of Pathology in collaboration with Department of surgery and Dental College. Data from 92 patients with oral cavity and jaw lesions regarding age, site, sex and histopathological diagnosis was retrieved from the request forms of the Departments of Pathology. Slides and blocks were taken from the archives of the Department of Pathology. All the slides were re-evaluated.

Results: A total of 92 cases were included, 58 were male and 34 were female patients.Male pre-dominance was seen with male to female ratio 1.5 to 1. The age range varied from 3 to 74 years. Buccal mucosa was involved in 21 cases (55%) followed by 19 cases in jaw (45%). The commonest tumour was Squamous cell carcinoma with 23 cases followed by 21 cases of capillary hemangioma .

Conclusion: In conclusion; Squamous cell carcinoma is the most common malignant jaw tumour followed by malignant salivary gland tumours.

KEYWORDS

Squamous cell carcinoma, Oral cavity, Jaw, Buccal mucosa.

Introduction

Oral cavity and jaw lesions, though inappreciable in proportion of the pathological conditions, are very important to diagnose as they have potential to jeopardise the health of the patient. They include a wide variety of lesions, of which malignant lesions comprise a significant number of cases. Tobacco chewing, ill fitting dentures, cigarette smoking are the predisposing factors for both benign and malignant lesions of oral cavity.1.2 The prevalence rate of oral cavity lesions varies from 0.2% to 0.5 % in India with higher percentage being found in southern areas of country.3, 4 The jaws are host to a wide variety of cysts and neoplasms.[5]

Aim of the study

The aim of the present study was to analyze the histopathological features of various lesions of the oral cavity and jaw and correlate the histopathological changes with relevant clinical findings.

Material and methods:

This retrospective study was done in the of Department of Pathology GMC Jammu, by using medical records and pathology reports of last three and half years. Data from 92 patients with oral cavity and jaw lesions regarding age, site, sex and histopathological diagnosis was retrieved from the request forms of the Departments of Pathology. Slides and blocks were taken from the archives of the Department of Pathology. All the slides were re-evaluated. Complete clinical details, examination findings, and X-ray of the patients were studied.

All lesions of oral cavity i.e lip, floor of the mouth, oral tongue, buccal mucosa, gingiva, retro molar trigone, hard palate and all lesions of jaw were included in study.

Results:

A total of 92 samples of oral cavity and jaw lesions were received for histopathological examination. All the 92 cases were studied in detail along with clinical features and radiological findings. In this series, male preponderance was observed as compared to females (63.1% vs 36.9 % respectively) [Table 1]. Majority of the benign lesions were observed in age group 21-30 years while malignant lesions were seen in 31-40 years age group [Table 1]. Most common site observed in the present study was tongue n=22 followed by floor of mouth n=20 and jaw n=14 [Table 2]. Patients diagnosed with malignant lesions presented mainly with short progressive history. Among 92 cases, 70.6 % were diagnosed as benign lesions while malignant lesions were seen in 29.4% [Table 3].

BENIGN LESIONS: Among bening lesions, majority of the cases were diagnosed as capillary haemangioma (n=14).The second most common benign lesion was observed as squamous papilloma (n =10). 9 cases were diagnosed as ameloblastoma. 3 cases of radicular cyst were reported. 2 cases each of pleomorphic adenoma of minor salivary gland and giant cell epulis were reported. 7 cases were reported as irritational fibroma. There were 2 cases which were diagnosed as leukoplakia. In one patient who presented with cystic swelling on lower jaw with a history of uninterrupted tooth and panoramic radiograph showed classically expansile "soap-bubble" lesion, with well-demarcated borders. The excised swelling on H&E section was reported adenoamelobastoma. One patient with swelling from left mandibular region showed round radioopaque mass attached to left mandibular region (first molar) and delineated by radiolucent band at the periphery on panoramic radiograph. The excised swelling was reported as benign cementoblastoma.

MALIGNANT LESIONS: Out of 92 lesions of oral cavity and jaw region, 27 cases (29.4%) were diagnosed as malignant. 23 cases (25%) were reported as squamous cell carcinoma. Verrucous carcinoma was diagnosed was diagnosed in 3 patients (3.2%). One (1.2%) adolescent female patient presented with swelling over mandibular region , underwent incisional biopsy of the swelling and was reported as osteosarcoma of jaw.

Discussion

It is indispensable to establish an accurate diagnosis of oral cavity lesions to initiate optimal therapy. An adequate incision biopsy taken from an area representative of the lesion can provide over 98% diagnostic accuracy as to whether the lesion is malignant or not, when routine pathological techniques are used.[6]In India, most oropharyngeal cancers present in advanced stages of malignancy. The cause of oral cavity lesions can usually be identified by history and physical examination however; it is often determined definitively by histopath ological examination and therapeutic response.[7] In case of jaw lesions, many benign jaw tumors and several cysts, both of odontogenic and nonodontogenic origin, can exhibit a biologically aggressive course and can be diagnostically difficult. Traditional histopathology continues to be the mainstay for the diagnosis of these lesions, as immunohistochemistry and molecular techniques have had, as yet, little impact in this area.[5]

In present study, the age range was 02-82 years. Mehta8 et al and Khateeb et al.[9] found age range 7-80 years and 6 -98 years respectively.

In our study, there was a male preponderance with 63.1% males and 36.9% females. Ramandeep S.G.[10] et al, in their study found 59% males and 41% females. The higher percentage of males in our population may be attributed to the more chewing habits of pan, pan masala, betel nut, and tobacco chewing than the females.

The most common site observed was buccal mucosa followed by jaw. Pudasaini et al found lip as the predominant site while Mehta et al also

showed buccal mucosa as the main site.11,[9]

Majority of the lesions were benign 70.6% while 29.4% were malignant. Shamim et al. in their study showed non-neoplastic in 75.5% and neoplastic in 24.5% while Mehta et al found incidence of both benign and malignant lesions as 75% and 25% respectively.[9,12]

Capillary haemangioma was observed as the most common lesion 15.2% . Lip was the most common site .The microscopic examination showed thin, ulcerated layer of stratified squamous epithelium lining, many vascular spaces lined by endothelial cells, acute on chronic inflammatory infiltrate along with extensive fibroblastic proliferation. The findings were in accordance with the study done by Kashyao et al.[13] Being common in head and neck region, they also occur in lips, cheek and tongue.[14]

The second most common lesion was found as squamous papilloma. The most common site involved was buccal mucosa. This benign mucosal neoplasm is caused by human papilloma virus (HPV), most commonly HPV-6 and HPV-11.15 Mishima et al in their study concluded that though most of these growths are noncancerous in nature, but a growth within the mouth does have a slightly higher likelihood of developing into a malignant mass.16 7.6% cases of irritational fibroma were reported. Microscopic examination showed parakeratosis, stratified squamous epithelium, flat rete-ridge pattern along with dense bundles of collagen fibers in haphazard arrangement and fibroblasts. Irritation fibroma represents a reactive focal fibrous hyperplasia due to trauma or local irritation.17, 18 Clinically, it resembles as pyogenic granuloma, peripheral giant cell granuloma or odontogenic tumors, so radiographic and histopathological examination is essential for accurate diagnosis. [19]

2 cases of giant cell epulis were reported . Giant cell epulis is one of the most frequent giant cell lesions of the jaws and originates from the connective tissue of the periosteum or the periodontal membrane.20 H&E stained section showed numerous multinucleated osteoclast-like giant cells lying in a very cellular and vascular stroma. According to Fanourakis et al. radiological examination is essential to determine if the lesion arises from the gingiva (peripheral) or bone (central) growing towards the surface. [21]

Three cases of radicular cyst were reported. Histopathologically, radicular cyst consists of a cavity lined by stratified squamous epithelium exhibiting spongiosis and/or exocytosis and are delimited by a fibrous wall containing a predominantly chronic inflammatory infiltrate.[22]

One case of inflammatory myofibroblastic tumor (IMT) of jaw region was reported. Most common sites of involvement include the lung, liver and orbit, but it has been reported to occur in nearly every site of the body, including the major salivary glands and the oral cavity.23, 24, 25 Histopathologically, IMT of the oral cavity presents with a variable admixture of fascicles of myofibroblastic spindle cells mixed with acute and chronic inflammatory cells.[26]

One case of benign fibrous histiocytoma(BFH) of mandible was reported. In the rest of the head and neck area, non-cutaneous BFH may arise in the buccal spaces, but cases of BFH of the tongue , gingival or alveolar ridge mandible has also been reported.27,28 The main differential diagnosis of oral BFH includes nodular fasciitis, solitary fibrous tumour, neurofibroma, and dermatofibroma. E. Calonje et al found in their study that BFH typically shows a biphasic cell population of histiocytes and fibroblasts.[29,30]

One case of tobacco keratosis was reported. Microscopically, tobacco keratoses show hyperkeratosis and acanthosis of the mucosal epithelium. True epithelial dysplasia is uncommon; when dysplasia is found, it is usually mild in degree. Such lesions typically occur in the buccal or labial vestibule where the tobacco is held, but they can also extend onto the adjacent gingiva and buccal mucosa. [31]

One case was reported as true giant cell tumor of mandible. The tumor comprised of sheets of spindle cells arranged in fascicular and trabecular pattern along with many multinucleated osteoclasic type of giant cells .They most frequently occur around the knee joint and can occur in mandible and wrist and the hip bones.[32]

Two cases of oral mucocele was reported. Most of the oral mucocele are devoid of the epithelial lining or are covered by granulation tissue.33 The surface of long-standing lesions may show fibrosis. It is the most common minor (accessory) salivary gland lesion affecting the general population.[34]

Two cases of benign pleomorphic adenoma were reported. Gnepp et al found that pleomorphic adenomas of minor salivary glands, like those on the palate, buccal mucosa or lip, occasionally lack encapsulations and may mix into normal host tissue as tumor growth hence, a wide excision is necessary even if previous biopsies report a benign nature[35]

Two cases were reported as proliferative verrucous leukoplakia (PVL). Microscopic examination showed lymphocytic infiltrate along with pronounced lichenoid pattern characterized by basal vacuolar degeneration containing apoptotic cells and eosinophilic bodies.PVL shows almost 100% rate of malignant transformation, mainly over an extended follow-up period. [36]

Two cases were reported as pseudoepitheliomatous hyperplasia. It is characterized by hyperplasia of epidermis and adnexal epithelium.This lesion is also referred as pseudocarcinomatous hyperplasia as they mimic squamous cell carcinoma.37 Invasion of epithelial proliferation by leucocytes and disintegration of some of the epidermal cells is typically seen in PEH, a finding that is absent in SCC. In case of verrucous carcinoma, it shows a verrucous upward and downward proliferation with more pronounced keartinisation in downward extension, which appears bulbous rather than sharply pointed as in PEH.[38]

Two cases of leukoplakia were reported and predominantly found in males in the age group of 41-60 years. The findings were in accordance with the study done by Walls Vander I et al.39 Moderate hyperkeratosis and epithelial hyperplasia without dysplasia are the most common histological findings reported for leukoplakia.[40]

Two patients presented with gingival swelling. H&E stained sections of the excisional biopsy of the swellings were reported as with periapical ossifying fibroma. It is a relatively common growth of gingiva and is reactive in nature rather than neoplastic. Microscopic examination shows a high degree of cellularity along with bone formation, and occasionally, dystrophic calcification or cementum-like material.[41]

Two cases were reported as dentigerous cyst of left lower jaw. Cyst was unilocular. Microscopic examination showed thin layer of cuboidal epithelium along with acute on chronic inflammation. They are usually derived from the epithelial remnants of tooth forming organs. Most commonly dentigerous cyst involves lower 3rd molar (mandibular). 42 Due to their specific structure and location of Odontogenic tumours, they have been identified and classified by pathologists into a separate group, differing in histogenesis, biology, clinical manifestations and radiological signs from other tumors developing in the oral cavity and facial bones.[43]

Nine cases of ameloblastoma were reported. They were further categorized. 5 cases were of follicular type, 2 cases were of plexiform type and 2 cases were of acanthotic type. Ameloblastomas, histologically are classified into follicular, plexiform, acanthomatous, granular cell, desmoplastic and basal cell types, with the follicular and plexiform patterns being the most common.44 The radiographic appearance of ameloblastomas varies from unilocular to multilocular, with well-defined sclerotic margins which may appear scalloped or may expand the cortical plate. [45]

One case was reported as benign cementoblastoma. Microscopic examination showed irregular lacune and uncalcified matrix within fibrovascular connective tissue and new trabeculae rimmed by atypical osteoblasts like cells. Prominent cementoblasts, irregular lacunae ,increased activity of cementoblasts and cementoclasts and trabeculae of uncalcified matrix perpendicular to the surface are typical characteristics of this lesion.46,47 The most difficult challenge in the differential diagnosis of cementoblastoma is osteoblastoma which may exhibit the same histomorphology but they differ in their origin.48 One case of adenomatoid odentogenic tumor was reported. This tumour constitutes only 0.1% of tumors and cysts of the jaw and 3% of all odontogenic tumors. Microscopic examination showed tumor cells arranged in trabeculae along with duct-like structures, foci of calcification and eosinophilic fibrillary material. The lesion sometimes exhibits aggressive behavior such as becoming unusually large, or spreading into the intracranial space.[49]

Three cases of verrucous carcinoma were reported .The lesser aggressiveness of verrucous carcinoma (VC) reinforces its classification as a well-differentiated variant of oral SCC with an excellent prognosis and indolent clinical behavior. Microscopically, VC is a predominantly exophytic growth of well-differentiated stratified squamous epithelium with deep bulbous rete ridges that exhibit little or no cytologic atypia and deep surface invaginations filled with parakeratin or orthokeratin . The lesion's margins show a compressive growth pattern and despite exaggerated rete pegs, the associated basement membrane appears intact. [50,51]

23 cases of Squamous Cell Carcinoma (SCC) was reported. The most common site was floor of mouth. Histopathological characteristics like keratinization, nuclear pleomorphism, number of mitoses and invasion pattern, were assessed, and accordingly well or moderately differentiated tumors were reported. No poorly differentiated SCC was detected in our patients. Histopathological examination revealed most of the tumors as well differentiated (16 cases). A wide range of tumour features, including size and site, histologic malignant grade, perineural spread at the invasive front, lymphovascular invasion and tumour thickness, have been described as major risk factors that adversely affect the prognosis for patients with oral SCC.52 The ventral surface of the tongue and the floor of the mouth are the sites most commonly affected by SCC because they are lined by thin non-keratinised epithelium.[53]

One case of osteosarcoma of mandible was reported. H&E stained sections showed round to oval spindle shaped highly pleomorphic cells, hyperchromatic nuclei along with tumor giant cells and mitotic figures. The osteoid in the form of thin tabeculae was laid down by tumor cells.The tumor cells can be spindled, clear , epithelioid, plasmacytoid or anaplastic giant cells.54 Approximately 7% of all primary osteosarcoma, arise in the jaw bones and mandible. Possibility of oral metastases should be considered in patients with known primary malignant disease and biopsy is essential for establishing the diagnosis.[55]

Conclusion :

In our study, we concluded that majority of oral cavity and jaw lesions were benign. However, the origin and nature of the oral cavity and jaw lesions cannot be confirmed by clinical examination alone. Hence, it is must to have a histopathological examination to confirm the histogenesis and malignant potential of the oral lesions.

Table 1 Gender wise distribution of lesions.

Age (years)	Sex		No of lesions	Benign	Malignant
	Male	Female			
0-10	1	3	4	4	nil
11-20	8	2	10	10	nil
21-30	10	9	19	18	1
31-40	14	6	20	8	12
41-50	12	3	15	10	5
51-60	8	9	17	11	6
61-70	3	2	5	3	2
71-80	2	nil	2	1	1
Total	58(63.1%)	34(36.9%)	92(100%)	65(70.6%)	27(29.4%)

Table 2 :Distribution of various pathological lesions in various sites of oral cavity

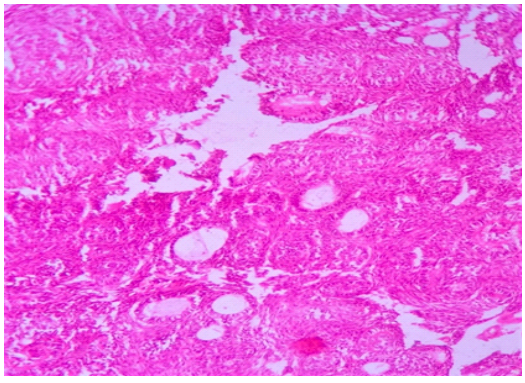
Lesion	Buccal mucosa	Lip	Floor of mouth	Tongue	Palate	Gingiva	Jaw	Total
Squamous cell carcinoma	5	3	5	4		2	4	23
Capillary haemengioma	4	7		3				14
Squamous papilloma	4	2		2	2			10
Ameloblastoma							9	9
Irritational fibroma	2			3	2			7
Radicular cyst/ Periapical cyst						3		3
Giant cell epulis/ peripheral giant cell granuloma						2		2
Verrucous carcinoma	3							3
Pleomorphic adenoma					2			2
Leukoplakia	2							2
Adenoameloblastoma							1	1
Benign Cementoblastoma							1	1
Giant cell tumour							1	1
Benign fibrous tumor	1							1
Myofibroblastic tumor							1	1
Pseudoepitheliomatous hyperplasia						2		2
Periapical ossifying fibroma						2		2
Proliferative verrucous leukoplakia		1				1		2
Dentigerous cyst							2	2
Mucocele				2				2
Tobacco keratosis						1		1
Osteosarcoma							1	1
Total	21	13	5	12	5	17	19	92

Table:3 Various benign and malignant tumors

Benign Lesions	Total	Percentage
Capillary haemengioma	14	15.4
Squamous papilloma	10	10.8
Ameloblastoma	9	9.7
Irritational fibroma	7	7.6
Radicular cyst/ Periapical cyst	3	3.2
Giant cell epulis/ Peripheral giant cell granuloma	2	2.1
Pleomorphic adenoma	2	2.1
Leukoplakia	2	2.1
Adenoameloblastoma	1	1.2
Benign Cementoblastoma	1	1.2
Giant cell tumour	1	1.2
Benign fibrous histocytoma	1	1.2
Myofibroblastic tumor	1	1.2
Pseudoepitheliomatous hyperplasia	2	2.1
Proliferative verrucous leukoplakia	2	2.1
Periapical ossifying fibroma	2	2.1
Dentigerous cyst	2	2.1
Mucocele	2	2.1
Tobacco keratosis	1	1.2
Malignant lesions		
Squamous cell carcinoma	23	25
Osteosarcoma	1	1.2
Verrucous carcinoma	3	3.2
Total	92	100

Figure1: Panoramic view x ray was labeled as dentigerous cyst

Figure2 : Adenoamelobalstoma (H&E stained section)



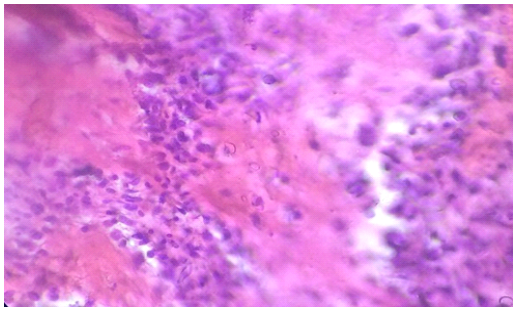


Figure 3 : Osteosarcoma of mandible (H&E stained section)

References

1. Mujica V, Rivera H ,Carrero M.Prevelance of oral soft tissue lesions in an elderly Venezuelan population . Med Oral Pathol Oral Cir Buccal. 2008;13:270-4.
2. Shamim T, Varghese VI, Shamim PM ,et al. A retrospective study of gingival biopsied lesions in south Indian population. Med Oral Pathol Oral Cir Buccal .2008;13:414-8.
3. Wahi PN, Luthra UK, Kapur VL. Submucous fibrosis of the oral cavity. Histomorphological studies. Br J Cancer. 1966 ; 20:676-87.
4. Mehta F.S. Pindborg J.J. Hammer J.E. et al Report on Investigations of Oral Cancer and Precancerous Conditions in Indian rural population 1966-69 Copenhagen Munks-Gaord 1971.
5. Joseph A. Regezi, D.D.S., M.S. Odontogenic Cysts, Odontogenic Tumors, Fibroosseous, and Giant Cell Lesions of the JawsMod Pathol 2002;15:331–341.
6. Warren S. Biopsy in relation to tumour diagnosis. 4th ed. Boston: American Cancer Society, Mass Division; 1968.
7. Shah JP, Patel SG: Head & Neck Surgery and Oncology, 3rd Ed. Mosby, London; 203.
8. Nikunj V. Mehta, Kalpana K. Dave, R.N.Gonsai, H.M.Goswami, Purvi S. Patel, Tarang B. Kadam Histopathological Study Of Oral Cavity Lesions: A Study On 100 Cases. Int J Cur ResRev. 2013; 05:110-116
9. Al-Khateeb TH. Benign oral masses in a Northern Jordanian population- a retrospective study. Open Dent J. 2009; 3:147-53.
10. Ramandeep SG, FL Veersha, Raman S, Heena K, Amit A, Deepak G.J. Clin Exp Dent. 2011 : 3(1) :7.
11. Pudasaini S, Baral R, Oral cavity lesions: A study of 21 cases, Journal of Pathology of Nepal. 2011:49-51.
12. Shamim T, Varghese VI, Shameena PM,et al. A retrospective analysis of gingival biopsied lesions in south Indian population: 2001-2006.Med Oral Pathol Oral Cir Buccal. 2008; 13: 414-18.
13. Bina Kashyap, P. Sridhar Reddy, P. Nalini Reactive lesions of oral cavity: A survey of 100 cases in Eluru, West Godavari district .Contemp Clin Dent. 2012 ; 3: 294–297.
14. Mehrortra MC.capillary haemengioma of hard palate. A case report .J all india dental association. 1965;37:11-12.
15. StinaSyrjanen. Human papillomavirus infections and oral tumors. Med MicrobiolImmunol. 2003; 192:123–128.
16. Koichi Mishima, Yoshiki Nariai, Seiji Obara, Yasuro Yoshimura. Human Papilloma virus in Oral Squamous Papilloma and Verrucous Carcinoma: Evaluation Using in Situ Polymerase Chain Reaction Detection. Asian J Oral Maxillofac Surg. 2002; 14:95-99.
17. Kolte AP, Kolte RA, Shrirao TS. Focal fibrous overgrowths: A case series and review of literature. Contemporary clinical dentistry. 2010;1:271.
18. Mathur LK, Bhalodi AP, Manohar B, Bhatia A, Rai N, Mathur A. Focal fibrous hyperplasia: a case report. International Journal of Dental Clinics. 2010;2:4-6
19. Hiroj Bagde, Alka Waghmare, B Savitha, Priyanka Vhanmane. Irritation fibroma – a case report. Int J dent clin.2013;1:39-40.
20. Chaparro-Avendano AV, Berini-Aytes L, Gay- Escoda C. Peripheral giant cell granuloma.A report of five cases and review of the literature. Med Oral Patol Oral Cir Bucal. 2005; 10:53–57.
21. FANOURAKIS, G. et al. Expression of receptor activator of NF-κB ligand and osteoprotegerin in peripheral giant cell granulomas of the jaws. J Oral Pathol Med. 2010: 39:. 687-689.
22. Nair PN. New perspectives on radicular cysts: do they heal? Int Endod J 1998; 31:155-160.
23. Narla LD, Newman B, Spottswood SS, Narla S, Koll IR. Inflammatorypseudotumor. Radiographics.2003; 23:719–29.
24. 2. Shek AW, Wu PC, Samman N. Inflammatory pseudotumour of the mouth and maxilla. J Clin Pathol.1996; 49:164–167.
25. 3. Van Weert S, Manni JJ, Driessen A. Inflammatory myofibroblastic tumor of the parotid gland: case report and review of the literature. Acta Otolaryngol. 2005; 125:433–437.
26. 2. Shek AW, Wu PC, Samman N. Inflammatory pseudotumour of the mouth and maxilla. J Clin Pathol.1996;49:164–167.
27. A. G. Triantafyllou, A. D. Sklavounou, and G. G. Laskaris, "Benign fibrous histiocytoma of the oral mucosa. J oral Med1985; 1:36–38.
28. P. A. Vargas, F. A. Alves, M. A. Lopes et al., "Solitary fibrous tumour of the mouth: report of two cases involving the tongue and cheek. Oral Diseases, 2002. 8: 111–115.
29. F. A. Alves, P. A. Vargas, S. A. Coelho Siqueira, R. D. Coletta, and O. P. De Almeida, "Benign fibrous histiocytoma of the buccal mucosa: case report with immunohistochemical features," Journal of Oral and Maxillofacial Surgery. 2003; 61: 269–271.
30. E. Calonje, T. Mentzel, and C. D. M. Fletcher, "Cellular benign fibrous histiocytoma: clinicopathologic analysis of 74 cases of a distinctive variant of cutaneous fibrous histiocytoma with frequent recurrence,"American Journal of Surgical Pathology. 1994;18:668–676,.
31. Neville BW, Day TA. Oral cancer nd precancerous lesions. CA Cancer J Clin 2002; 52:195-215.
32. Neville BW, Damm DD, Allen CM, Bouquot JE. Oral and maxillofacial pathology. 2nded. Philadelphia (PA): WB Saunders; 2002. 439-440.
33. Re Cecconi D, Achilli A, Tarozzi M, Lodi G, Demarosi F, Sardella A, Carrassi A Med Oral Patol Oral Cir Bucal. 2010 ;15:551-556.
34. Gupta B, Anegundi R, Sudha P, Gupta M. Mucocele: Two case reports. J Oral Health Community Dent. 2007; 1:56–58.
35. Gnepp DR (2009). Salivary gland (major and minor) and lacrimal gland. In: Gnepp DR (ed) Diagnostic surgical pathology of the head and neck, 2nd edn, WB Saunders, Philadelphia, pp. 434–449.
36. Onofre MA, Sposto MR, Navarro CM, Motta ME, Turatti E, Almeida RT. Potentially malignant epithelial oral lesions: Discrepancies between clinical and histological diagnosis. Oral Dis 1997; 3:148-152.
37. Pseudoepitheliomatous hyperplasia: a review. Zayour M, Lazova RAm J Dermatopathol. 2011; 33: 123-126.
38. Elder DE. Lever's Histopathology of the Skin. 10th ed. Philadelphia: Lippincott Williams & Wilkins; 2008:822–823.
39. Walls Vander I et al. Oral Leukoplakia : A Clinicopathological review. Oral oncology 1997; 33(5):291-301.
40. Reibel J. Prognosis of oral pre-malignant lesions: significance of clinical, histopathological, and molecular biological characteristics. Crit Rev Oral Biol Med. 2003; 14:47-62.
41. Fausto KA. Robbins and Cotran pathologic basis of disease. 7th ed. Philadelphia: WB Saunders; 2008. pp. 775–6.
42. Amin ZA., Amran M., Khairudin. Removal of extensive maxillary dentigerous cyst via a Caldwell¬Luc procedure. Arch Orofac Sci 2008;3(2):48¬51. 2. Neville BW, Dam DD, Allen CM, Bouquet JE. Oral and Maxillofacial Pathology. 2nd ed. Saunders; c2001. 768p
43. Stypulkowska J: Odontogenic tumors and neoplastic-like changes of the jaw bones. Clinical study and evaluation of treatment results. Folia Med Cracov 1998; 39(1-2):35-141.
44. Lee SK, Kim YS. Current concepts and occurrence of epithelial odontogenic tumors: I. Ameloblastoma and adenomatoid odontogenic tumor. Korean J Pathol 2013; 47:191-202.
45. White SC, Pharoah MJ, editors. Oral radiology: Principles and interpretation. 5 th ed. St. Louis, Missouri: Mosby (Elsevier); 2004. p. 419-22.
46. Hubber, A.R. and Folk, G.S.Cementoblastoma. Head and Neck Pathol.2009; 3:133-135.
47. Brannon, R.B., Fowler, C.B., Carpenter, W.M. and Corio, R.L. Cementoblastoma: An innocuous neoplasm? A clinicopathologic study of 44 cases and review of literature with special emphasis on recurrence. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology. 2002;93: 311-320
48. Cementoblastoma and osteoblastoma: A comparison of histologic features. Journal of Oral Pathology & Medicine, 21, 385-389.
49. Raubenheimer EJ, Seeliger JE, van Heerden WF, Dreyer AF. Adenomatoid odontogenic tumour: A report of two large lesions. Dentomaxillofac Radiol 1991; 20:43-45.
50. Kraus F, Perezmesa C. Verrucous carcinoma. Clinical and pathologic study of 105 cases involving oral cavity, larynx and genitalia. Cancer 1966; 19(1):26–38.
51. Jacobson S, Shear M. Verrucous carcinoma of the mouth. J Oral Pathol 1972; 1(2):66–75
52. Johnson N, Franceschi S, Ferlay J, Ramadas K, Schmid S, MacDonald DG, and others. Squamous cell carcinoma. In: Barnes L, Everson JW, Reichart P, Sidransky D, editors. World Health Organization classification of tumours. Pathology and genetics of head and neck tumours. Lyon: International Agency for Research on Cancer Press; 2005. p. 168–75
53. B. W. Neville and T. A. Day, "Oral Cancer and Precancerous Lesions," CA: A Cancer Journal for Clinicians, Vol. 52, No. 4, 2002, pp. 195-215.
54. Kaur H, Singh A. Osteosarcoma of jaw - Case report and review of literature. Int J Med and Dent Sci 2015; 4(1):653-657
55. Suzuki K, Yoshida H, Onizawa K, Onobori M. Metastatic osteosarcoma to the mandibular gingiva: a case report. J Oral Maxillofac Surg 1999; 57:864-868