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REACTIVE LESIONS OF THE ORAL CAVITY: A REVIEW

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ABSTRACT

Reactive hyperplasia is a group of fibrous connective tissue lesions that occur commonly on the oral mucosa as a result of injury or trauma. They represent the chronic process in which an over exuberant repair follows injury. Various reactive lesions have different patterns and appearances. Though the reactive lesions are common in the oral cavity, many of the clinical practitioners are still unaware of the various reactive lesions and their management. The present article describes the most common reactive lesions of the oral cavity.

Keywords: Focal hyperplasia, Reactive hyperplasia, Traumatic lesions.

INTRODUCTION

In the oral cavity, mucosa is constantly subjected to various external and internal stimuli and therefore manifests a spectrum of lesions ranging from developmental, reactive, and inflammatory to neoplastic. Reactive hyperplastic lesions represent the most frequent oral mucosal lesions in humans. [1]

The injuries may result from mechanical trauma like chronic or recurrent irritations such as calculus, overhanging dental restorations, ill-fitting dentures; fractured, carious, malaligned or malformed teeth; as well as chemical, electrical and thermal insults may also be involved.

Reactive lesions are more common in gingiva followed by buccal mucosa, lip, tongue and palate. [1-3]

The usual appearance of these proliferating lesions is painless pedunculated or sessile masses in different colors, from light pink to red. The surface texture is variable from non-ulcerated smooth to ulcerated mass. Lesion size varies from a few millimeters to several centimeters.4 Reactive lesions histologically represent fibrous hyperplasia with chronic inflammation, granulation tissue and proliferation of endothelial cells and fibroblasts. Other histologic component such as multinucleated giant cells, calcified material, or small vessels hyperplasia may also be eveident. [2,4]

Classification

On the basis of site involved reactive lesions can be classified as[5-7]

A. Lesions predominantly affecting gingiva:
   Peripheral fibroma (Fibrous hyperplasia, fibrous epulis)
   Pyogenic granuloma
   Peripheral giant cell granuloma
   Peripheral ossifying fibroma
B. Lesions affecting tongue:
   Hairy tongue
   Hairy leukoplakia
C. Lesions affecting Palate
   Nicotine stomatitis
D. Lesions affecting Buccal mucosa and vestibule
   White lesions associated with smokeless tobacco
   Dentifrice associated slough
E. Lesions involving any site of oral cavity
   Traumatic ulcerations: acute and chronic

Some important reactive lesions of the oral cavity

Peripheral fibroma: The peripheral fibroma results from trauma or local irritation and is considered as the most common reactive lesion. Clinically it appears as a raised mass that is sessile or pedunculated with a smooth surface. It is usually of the same color as the surrounding gingiva and is commonly found in the interdental papilla of the anterior teeth in

adults. Histologically, the peripheral fibroma consists of hyperplastic fibrous tissue with a varying degree of vascularity. [5,8]

Peripheral giant cell granuloma: Bernier and Cohn coined the term 'peripheral giant cell granuloma' in 1954, and the term 'giant cell reparative granuloma' was first used by Jaffe. [9] It is benign and asymptomatic hyperplastic lesion of the attached gingiva or alveolar mucosa. It is thought to arise from gingival connective tissue or periosteum of alveolar ridge in response to injury. [9] The lesion presents as sessile or pedunculated growth with smooth surface. It can also be seen as dome shaped papule or nodule, which is vascular hemorrhagic and manifest surface ulceration. [10]

Microscopic examination shows keratinized stratified squamous epithelium with sometimes presence of focus of ulceration. In some instances the marginal epithelium shows pseudoepitheliomatous hyperplasia. It consistently showed giant cells, extravasation of red blood cells with deposition of varying amounts of hemosiderin and densely cellular stroma. [11]

Pyogenic granuloma: It is a type of inflammatory hyperplasia seen in oral cavity. The name pyogenic granuloma is a misnomer, as they rarely produce pus (pyo=pus genic=producer) and they are not true granuloma. [12] Hullihens' description in 1844, was most likely the first pyogenic granuloma reported in English literature, but Hartzell in 1904 introduced the term 'pyogenic granuloma' or 'granuloma pyogenicum'. [13] The lesion arises in response to various stimuli such as chronic low grade irritation, traumatic injury, hormonal factors or certain kinds of drugs.[14] Clinically it is exophytic lesion with smooth or lobulated surface, manifesting as red erythematous papule on the sessile or pedunculated which is hemorrhagic, compressible and may develop a dumbell shaped mass. [8, 11,15]

Microscopic examination of pyogenic granuloma shows highly vascular proliferation that resembles granulation tissue. Numerous small and large endothelium lined channels are found that are engorged with red blood cells. These blood vessels are organized in lobular aggregates. [11,15]

Peripheral ossifying fibroma: Peripheral ossifying fibroma has also been called as calcifying fibroblastic granuloma, peripheral odontogenic fibroma, and peripheral cementifying fibroma. It is a reactive focal overgrowth usually occurring on gingiva. The

lesion is usually limited to the interdental papilla and arises from periosteum, periodontal membrane or from gingival corium. [5,15,16] Clinically, the lesion appears as nodular sessile or pedunculated mass and is usually of red to pink in color. [17]

Histopathologically it shows connective tissue with varying contents of fibroblasts, myofibroblasts and collagen. Sparse to profuse endothelial cell proliferation with mineralized material in the form of mature lamellar, osteoid, cementum like material or dystrophic calcification is seen. This mineralized material is usually in form of tiny or large globules or irregular masses of multiple basophilic color. [18]

Traumatic ulcerations: As the oral cavity is lined by a thin mucosa as compared to epithelium of skin, it is more vulnerable to trauma. The traumatic insults to the oral mucosa can break the integrity of the oral epithelium which is referred to as ulcer. Oral ulcer can be superficial erosion or may extend deep in the underlying connective tissue, which leads to painful ulceration. [11] It can result from biting, contact with sharp foodstuffs, denture irritation, toothbrush injury, irritation from sharp tooth or caused by electrical, thermal and chemical burns. [11,19]

Microscopically, a simple traumatic ulceration shows an adjacent normal surface epithelium, which may or may not show hyperkeratosis. The underlying connective tissue of the affected side or an ulcer bed shows plenty of granulation tissue intermixed with infiltrate of lymphocytes, histiocytes and plasma cells. [20]

Nicotine stomatitis: It was also named as “stomatitis nicotina” by Thoma because it is usually seen in smokers.[21] It arises as a reactive response to the heat produced due to pipe, cigarette and cigar smoking or with chronic ingestion of hot liquids. [20] The lesion begins as an erythematous area, which gradually becomes thick fissured and white or whitish grey. The lesions are exclusively present on the palate especially posterior to rugae and adjacent to the soft palate. [20]

It usually manifests as a flat topped nodules that have central red depressed dot. Additionally there may be fissures running across the palate. The nodules are produced by the cystic dilatation of salivary ducts. The red dots represent the inflamed orifices of palatal salivary glands and are attenuated by leukoplakia which represents hyperorthokeratosis. [20]

Histologically the lesions exhibit the acanthotic and hyperkeratotic stratified squamous epithelium with underlying connective tissue stroma showing some mild to moderate chronic inflammation. The epithelium of the minor salivary gland ducts often shows squamous metaplasia. [20]

Dentifrice- associated slough: It is associated with the use of different brands of toothpaste. The etiology is believed to be superficial chemical burn or reaction to content of the dentifrice. It appears as a superficial whitish slough of the buccal mucosa. It is painless and resolves after shifting to another blander toothpaste. [7,11]

White lesions associated with smokeless tobacco: White lesions develop at the immediate area of tobacco placement. Most common area of involvement is the mucobuccal fold of the mandible. It appears as granular or wrinkled lesions. Histologically it shows hyperkeratinization with edematous superficial epithelium and chronic inflammatory cell infiltration. Epithelial dysplasia can develop in these lesions. [7,15]

Hairy tongue: It is condition of the filiform papillae overgrowth on the dorsal surface of the tongue. Broad spectrum antibiotics, oxygenating mouthrinses containing hydrogen peroxide, smokers, and patient's undergone radiotherapy are considered as the possible predisposing factors. The basic pathogenesis is believed to be related to the change of the microbial flora, with proliferation of the fungi and chromogenic bacteria. [7,11,15] Microscopic examination shows elongated filiform papillae with surface contamination by microorganisms. [7]

Hairy leukoplakia: It represents the opportunistic infection due to Epstein-Barr virus (EBV) and is found only in patients with HIV infection. Clinically it appears as a flat, plaquelike, or papillary to filiform lesion on the lateral margins of the tongue. Histologically characteristic viral inclusions or peripheral displacement of chromatin evident in the superficial layer epithelial cells; resulting in smudgy nucleus appearance. The surface shows marked hyperparakeratosis. [7,11,22]

CONCLUSION

Though the reactive lesions appear similar, they show differences in some clinical characteristics, duration or histologic features. Although benign in nature, they have tendency to recur with incomplete removal of the lesion. Therefore it is advised to do complete surgical excision.

REFERENCES

1. Reddy V, Saxena S, Saxena S, Reddy M. Reactive hyperplastic lesions of the oral cavity: A ten year observational study on North Indian Population. J Clin Exp Dent, 2012; 4(3):e136-40.
2. Amirchaghmaghi M, Mohtasham N, Mozafari PM, Dalirsani Z. Survey of Reactive Hyperplastic Lesions of the Oral Cavity in Mashhad, Northeast Iran. J Dent Res Dent Clin Dent Prospect, 2011; 5(4):128-131.
3. Vickers RA. Thomas oral pathology: Vol.2. 6th ed. C.V.Mosby company; 1970.
4. Naderi NJ, Eshghyar N, Esfehanian H. Reactive lesions of the oral cavity: A retrospective study on 2068 cases. Dent Res J, 2012; 9:251-5.
5. Rossmann JA. Reactive Lesions of the Gingiva: Diagnosis and Treatment Options. The Open Pathology Journal, 2011; 5: 23-32.
6. Ramu S, Rodrigues C. Reactive Hyperplastic Lesions of the Gingiva: A Retrospective Study of 260 Cases. World J Dent, 2012; 3(2):126-130.
7. Regezzi JA, Sciubba JJ, Jordan RCK. Oral pathology: Clinical pathologic correlations. 4th ed. USA: Saunders; 2003.
8. Kashyap B, Reddy PS, Nalini P. Reactive lesions of the oral cavity: A survey of 100 cases in Eluru, West Godavary district. Contemp Clin Dent, 2012; 3:294-7.
9. Kfir Y, Buchner A, Hansen L. Reactive lesions of gingiva: A clinicopathological study of 741 cases. J periodontal, 1980; 655-661.
10. Lucas RB. Pathology of tumours of oral tissues. 3rd ed. London: Churchill livingstone; 1976.
11. Neville BR, Damm DD, Allen CM, Bouquot JE. Oral and maxillofacial pathology. 3rd ed. India: Saunders; 2004.
12. Jafarzadeh H, Satankhan M, Mohtasham N. Oral pyogenic granuloma: A review. J Oral Science, 2006; 48(4):167-175.
13. Villmann A, Villmann P, Villmann H. Pyogenic Granuloma; Evaluation of oral condition Brit J Oral Maxillofac Surg, 1986; 24:376-382.
14. Bhasker SN, Jacoway JR. Peripheral Fibroma and peripheral fibroma with calcification : Report of 376 cases. JADA, 1966; 73:1312-1320.
15. Shafer WG, Hine MK, Levy BM. A text book of oral pathology. 5th ed. Bangalore; prism book Pvt Ltd; 2006.
16. Bodner L, Dayan D. Growth potential of peripheral ossifying fibroma. J Clin Periodontal 1987; 14:551-54.

17. Bhaskar SN. Synopsis of Oral Pathology. 7th ed. C.V. Mosby Company; 1986.
18. Kumar SK, Ram S, Jorgensen MG, Charles F, Schuler, Sedghizaden PP. Multicentric peripheral ossifying fibroma. J oral science, 2006; 48(4): 239-243.
19. Nikolaos G. Nikitakis. Oral soft tissue lesion: A guide to differential diagnosis part II; surface alterations. Braz J. Oral Sci, 2005; 4(13): 57-59.
20. Saraf S. Textbook of oral pathology. 1st ed. Noida: Gopson papers Ltd; 2006.
21. Rossie KM; Pittesberg JG. Thermally induced nicotine stomatitis: A case report. Oral Surg Oral Med Oral Pathol, 1990; 70(5):597-599.
22. Cawson RA, Odell EW. Cawson's essentials of oral pathology and oral medicine. 7th ed. Elsevier Science; 2002.