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Review Article

Reactive lesions of oral cavity

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ABSTRACT

Oral mucosa is constantly subjected to various external and internal stimuli that leads to various reactive lesions. They are non-neoplastic in nature, but clinically these lesions closely mimic benign neoplastic lesions leading the clinician to misdiagnosis and sub-optimal treatment. Reactive lesions are typically occurring as a response to chronic inflammation caused by various forms of low-grade chronic irritations to the oral mucosa such as dental plaque and calculus, sharp edges of grossly carious teeth, faulty dental restorations, chronic biting habits, ill-fitting dental/oral appliances and food impactions. This review article summarizes the various reactive lesions of oral cavity and their distinctive histopathologic features.

1. Introduction

Oral cavity is repeatedly being exposed to many insults and stimuli that can irritate the oral mucosa leading to a variety of reactive hyperplastic reactions.[1] Reactive lesions represent the most frequent oral mucosal lesions in humans. These continuous irritation causes injuries that may result from mechanical trauma like chronic or frequent irritations like calculus, overhanging dental restorations, ill-fitting dentures; fractured, carious, misaligned, or malformed teeth; as well as chemical, electrical and thermal insults may also be involved.[1] Reactive lesions are most commonly encountered in the gingiva followed by buccal mucosa, lip, tongue, and palate.[2]

To classify any lesion as a reactive lesion, it should showcase;

1. Excessive proliferation of the tissue in response to a stimulus.
2. Some kind of stimulus should be present –trauma, low grade irritation or injury.
3. Remission or partially regress after an appropriate treatment and removal of the stimuli.[3]

All these lesions intermingle with each other in their clinical appearances, but the final diagnosis depends on their histopathological aspect of the lesion. According to Eversole and Rovin, the different histological entities of reactive inflammatory hyperplasia may be due to the response of connective tissue to varying intensities of mucosal irritation, which is very similar to neoplastic proliferation.[3] This paper explains the distinguishing clinical and histopathological features and the management of some of the most stumbled across reactive lesions.

2. Working Classification

2.1. Lesions predominantly involving gingiva

1. Pyogenic granuloma
2. Peripheral giant cell granuloma

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3. Peripheral ossifying fibroma

2.2. *Lesions predominantly involving palate*

1. Nicotine stomatitis
2. Inflammatory papillary hyperplasia

2.3. *Lesions involving alveolar mucosa*

1. Epulis fissuratum

2.4. *Lesions involving buccal mucosa & vestibule*

1. Smokeless tobacco keratosis.

2.5. *Lesions involving tongue*

1. Hairy tongue.

2.6. *Lesions involving salivary gland*

1. Mucocele.
2. Necrotizing sialometaplasia.

2.7. *Lesions involving neural tissue*

1. Traumatic neuroma.

2.8. *Lesions involving any site*

1. Focal fibrous hyperplasia
2. Traumatic ulcerations
3. Giant cell fibroma
4. Frictional keratosis
5. Oral mucosal peeling (OMP)

3. Lesions Predominantly Invovling Gingiva

3.1.

3.1.1. *Pyogenic granuloma (PG)*

PG is a localised proliferative lesion, clinically exhibited as painless sessile or pedunculated growth which profusely bleeds &presents with a normal overlying surface or ulcerated sometimes. It results from the profuse reaction to numerous provocating factors like chronic local irritation because of sharp margins of tooth crown, calculus, poor restorations and trauma, hormonal imbalances (Pregnancy), bone marrow transplants etc, tongue.[4] The name pyogenic granuloma is an inaccurate term, as they rarely produce pus(pyo means pus & genic means producer) and PG are not true granuloma.[2]

PG related with pregnancy are known as pregnancy tumours or granuloma gravidarum. The growth may appear by the 1^{st} trimester along with a gradual growth incidence to the seventh month of pregnancy, following the increasing levels of oestrogen and progesterone. Local irritation because of poor dental hygiene happens to be one of the

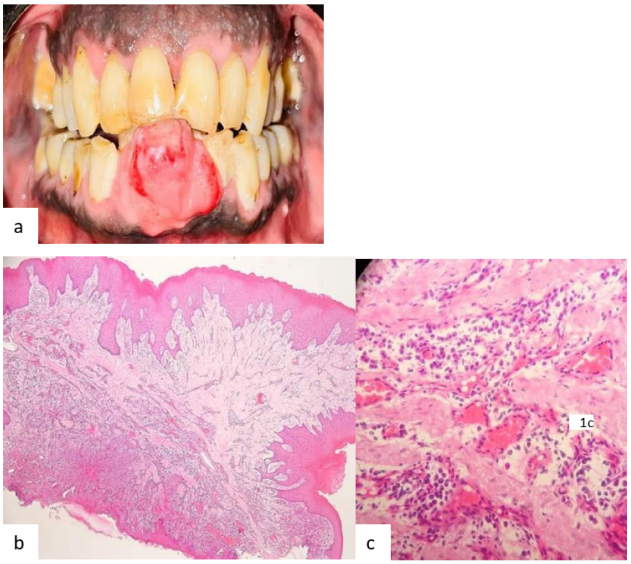

Figure 1: a): Nodular sessille mass present in lower anterior gingiva; **b**): Haematoxylin and eosin-stained section, 10x magnification showing hyperplastic parakeratinized epithelium with surface ulceration and underlying cellular stroma showing proliferating capillaries and inflammatory infiltrate; **c**): Higher power showing endothelial lined capillaries distended with RBCs

etiologic cause. Women with pregnancy tumours shows significant rise in Fibroblast growth factor (FGF) and vascular endothelial growth factor (VEGF) and less TNF-α. Some of these lesions resolve on its own following delivery. Surgery is often not necessary. It can be removed during the second trimester if it affects occlusion or is painful and bleeds excessively or is very large in size.[5]

Histopathologically, it shows distinguishing feature. The lesion has characteristic appearances like (i) the cellular phase, (ii) capillary phase/vascular phase, and (iii) involutionary phase.PG are of two types, they are lobular capillary haemangioma (LCH) & nonlobular capillary haemangioma (non-LCH).[3] The overlying epithelium, if present, is generally thin and atrophic, but may be hyperplastic. If ulceration is present, it is accompanied with fibrinous exudate of varying thickness over the surface.[5]

Numerous small and large endothelium lined capillaries are present which are distended with RBCs. These blood vessels are arranged in lobular aggregates. Lobular mass of proliferating endothelium lined capillaries is prominent feature of LCH while the non-LCH group has high vascular proliferation simulate granulation tissue.[6]

3.2. *Peripheral giant cell granuloma (PGCG)*

PGCG usually presents as asymptomatic soft nodular growth of the attached gingiva. The typical bluish red hue is the characteristic of the PGCG. A secondarily infected lesion has a ‘yellow zone’ caused due to the aggregation of

a fibrin clot at the ulcer site.[3]

Clinically, it looks like pyogenic granuloma but can be carefully distinguished based on the colour of the lesion. PG looks bright red in colour whereas PGCG is bluish red in colour. It is believed to originate from gingival connective tissue or periosteum of alveolar ridge in response to injury.[7]

Microscopic examination shows keratinized stratified squamous epithelium along with presence of areas of ulceration seen and sometimes, the marginal epithelium shows pseudoepitheliomatous hyperplasia.[6] Numerous multinucleated giant cells along with proliferating fibroblasts in fibro cellular stroma are the hallmark of the lesion.[3]

Giant cells are of 2 types: the first type comprises of cells with slightly basophilic cytoplasm and many ovoid vesicular nuclei with prominent nucleoli (Type l). The second type consists of smaller cells with eosinophilic cytoplasm and small pyknotic nuclei.[8]

3.3. *Peripheral ossifying fibroma (POF)*

POF occurs mainly due to the injury or local irritation and it present as a slow-growing mass predominantly in inter-proximal area of gingiva as a pedunculated or sessile mass with female predilection. The term peripheral ossifying fibroma is used because of presence of bone forming areas as well as high degree of cellularity.[5] Even though it is a separate entity, several studies show pyogenic granulomas in which fibrous areas undergo maturation followed by calcification are termed as POF. However, not all the POF develop in this manner.[5,6]

Microscopically, there is presence of fibro cellular proliferation, along with areas of calcification in the connective tissue, surfaced with stratified squamous epithelium. The mineralized component may vary from bone or scattered basophilic, cementum like material or dystrophic calcification. The bone is woven and trabecular type.[9,10]

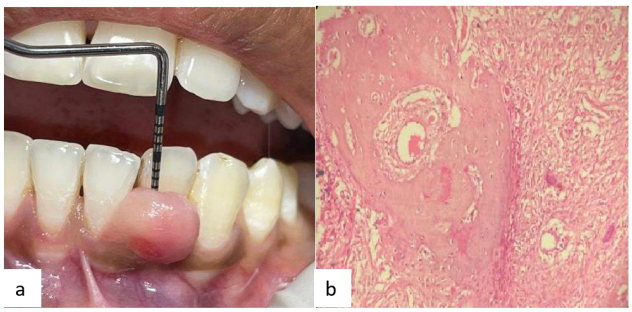

Figure 2: a): Nodular lesion present in anterior lower gingiva; **b**): Haematoxylin and eosin-stained section, 60x magnification showing presence of fibrocellular proliferation with areas of calcification and bone formation.

Treatment for these lesions involving gingiva is mainly by eliminating the local irritating factors along with complete surgical excision. Recurrence of these lesions are rare.[5,9]

4. Lesions Predominantly Involving Palate

4.1. *Nicotine stomatatis*

Nicotine stomatitis occurs as a result of reactive response to heat produced due to cigarette, pipe and cigar smoking or with chronic ingestion of hot liquids. It was termed as "stomatitis nicotina" by Thoma because it is usually seen in smokers.[11]

Clinically, the lesion begins as an erythematous area, which gradually becomes thick fissured and white or whitish grey. It is exclusively present on the palate especially posterior to rugae and adjacent to the soft palate. And it usually manifests as a flat-topped nodule that have central red depressed dot.[5,6] Additionally, there may be fissures running across the palate. The nodules are formed by the cystic dilatation of salivary ducts. The red dots are due

to inflamed orifices of palatal salivary glands and are attenuated by leukoplakia which represents hyper orthokeratosis.[11]

Microscopic examination shows acanthotic and hyperkeratotic stratified squamous epithelium with underlying connective tissue stroma showing some mild to moderate chronic inflammation.[5] The epithelium of the minor salivary gland ducts often shows squamous metaplasia. The degree of epithelial hyperplasia and hyperkeratosis depends upon the duration and the level of heat exposure. Rarely, epithelial dysplasia is seen.[11]

Nicotine stomatitis is completely reversible upon cessation of smoking habit usually within 1-2 weeks.

4.2. *Inflammatory papillary hyperplasia*

Inflammatory papillary hyperplasia (IPH) is a reactive hyperplasia exclusively affecting hard palate and presents as growth of one or more nodular lesions, measuring about 2mm or less. Most commonly noticed in poor-fitting dentures, continuous night use of denture, poor oral hygiene, sensitivity to denture liners, tobacco. Synonyms for IPH are "denture stomatitis," "granular stomatitis" and "Newton's type III."[12]

Microscopic examination shows parakeratotic stratified squamous epithelium and numerous papillary growths, with or without chronic inflammation. The deeper aspects of the epithelium show pseudo epitheliomatous hyperplasia or the formation of keratin pearls and microcysts. The keratin pearls may also undergo calcification. The epithelium is supported by hyperplastic central cores of well vascularized connective tissue which shows oedema, myxomatous degeneration, plasma cells and lymphocytic infiltration.[13]

Management of IPH depends upon severity of lesion. Denture removal allows erythema and edema to subside and for severe lesions, surgical excision of hyperplastic tissue followed by denture lined with tissue conditioners provides relief.

5. Lesions Involving Alveolar Mucosa

5.1. Epulis fissuratum

Epulis fissuratum means reactive response from tissue because of too much mechanical pressure from an ill-fitting prosthesis.[11] Clinically, it appears as single or multiple folds of hyperplastic tissue on the alveolar mucosa which is usually firm and fibrous on palpation. Most commonly seen in the facial aspects of alveolar ridge compared to lingual side. If it is secondarily infected, it shows erythema and ulceration.

Histopathologically, the overlying epithelium is hyper parakeratotic with hyperplasia of the rete ridges and hyperplasia of fibrous connective tissue. Sometimes, the epithelium shows pseudoepitheliomatous hyperplasia. Chronic inflammatory cells are seen.[5,6] Focal areas of ulceration are seen, especially at the base of the grooves between the folds. Another histologic finding seen in surface epithelium is mucopolysaccharide keratin dystrophy ("plasma pooling.") It appears as homogeneous, eosinophilic pools of material in the superficial spinous layer of epithelium, where it appears to have replaced individual cells. They are called as Toto's bodies.[11]

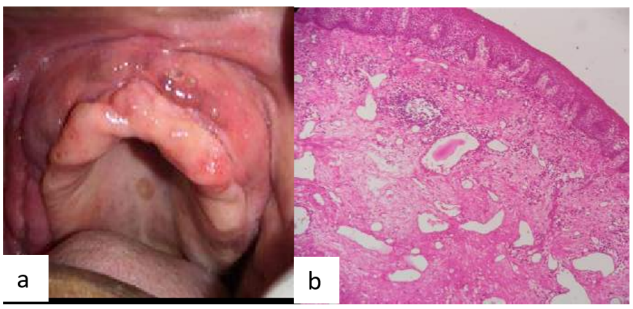

Figure 3: a): Hyperplastic tissue present on alveolar mucosa due to ill-fitting prosthesis; **b**): Haematoxylin and eosin-stained section 10x magnification shows hyperparakeratotic epithelium with hyperplasia of rete ridges and hyperplasia of connection tissue.

Treatment of epulis fissuratum includes surgical excision of hyperplastic tissue, removal of denture flanges and after healing, relining & rebasing of denture should be done.[5,11]

6. Lesions Involving Buccal Mucosa & Vestibule

6.1. Smokeless tobacco keratosis

Smokeless tobacco keratosis (STK) occurs mainly because of constant irritation caused by tobacco and it causes keratotic changes in the oral mucosa where smokeless tobacco quid is placed. Also known as snuff dipper's lesion, snuff pouch. The principle changes seen in the oral cavity related to smokeless tobacco (SLT) include (i) SLT-induced keratosis (STKs); (ii) gingival inflammation, periodontal inflammation, and alveolar bone damage; (iii) dental caries, tooth abrasion, and staining of tooth structure; and (iv) dysplasia and oral cancer.[14]

Clinically, early lesions show filmy white to gray opalescent appearance with a wrinkled surface and minimal mucosal thickening. With progression the lesions become more keratotic with furrowing of the epithelium and thickening.[15]

Histopathology presents with a non-specific appearance, with hyperkeratotic and/or acanthotic squamous epithelium. Increased sub-epithelial vascularity and intracellular edema may also be present. Para keratin chevrons above or within the superficial epithelial layers can generally be visualized. So, these lesions should be evaluated for epithelial dysplasia.[15]

Management of STK usually involves cessation of habit which leads to normal appearance of mucosa usually within 2 weeks.[15]

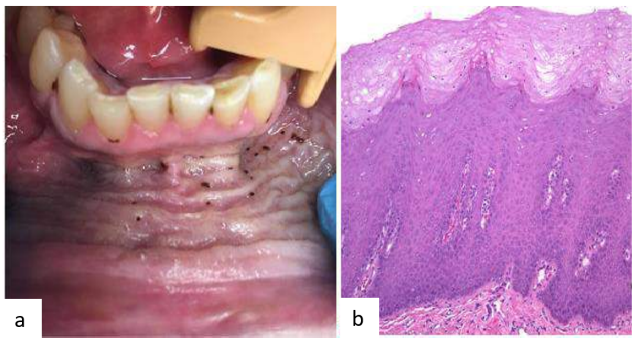

Figure 4: a): Lesion shows gray opalescent appearance with a wrinkled surface and flecks of smokeless tobacco within the lesion; **b**): Haematoxylin and eosin stained section(100x magnification) shows parakeratotic surface and epithelial acanthosis, prominent chevron keratinization are seen. The basal layer shows hyperchromatic nuclei[11]

7. Lesions Involving Tongue

7.1. Hairy tongue

Hairy tongue is usually seen on the dorsal tongue surface and shows hairlike projections, because of the presence of elongated and hypertrophied filiform papillae. Thus, the

normal filiform papillae length which is 1 mm and, in the hairy tongue, it exceeds 3 mm and may go up to 15mm.[16]

Etiologic factors are smoking, poor oral hygiene**.** Other risk factors includes alcohol and IV (intravenous) drug use, excessive coffee consumption, general debilitation, and recent head & neck radiation therapy. Prolonged use of oxidizing mouthwashes containing sodium perborate, sodium peroxide, and hydrogen peroxide are also predisposing factors. Antipsychotics, methyldopa, lansoprazole, antidepressants, antineoplastics, and antibiotics (e.g., cephalosporins, penicillin, tetracyclines, clarithromycin, and linezolid) are also been reported as cause for black hairy tongue.[16]

Clinically, these lesions can be scraped off. Rarely presence of gagging, nausea, dysgeusia, xerostomia, burning sensation and halitosis in some patients.[17]

Histopathologically, it is characterized by marked elongation and hyper parakeratosis of the filiform papillae. Usually, numerous bacteria can be seen growing on the epithelial surface.[6]

There is no treatment required for hairy tongue for most cases except for aesthetic concern and bad breath. Certain predisposing factors like tobacco, antibiotics should be eliminated. Periodic scrapping of tongue is advised for desquamation of hyperkeratotic papillae.[6,17]

8. Lesions Involving Salivary Gland

8.1. Mucocele

Mucocele occurs mainly due to obstruction or trauma to salivary glands and mostly seen in lower lip (90%) and other areas such as floor of mouth (6%) and ventral surface of tongue (4%). Trauma causes splitting of the salivary duct and mucin spillage into the adjoining connective tissue. Clinically, soft tissue swelling which are vesicular, translucent, bluish colour, and fluctuant on palpation are present.[18,19]

Histologically, it is categorized into two types: Mucus extravasations cyst and mucus retention cyst. Mucus extravasation is more common. Mucus extravasation cyst usually undergo three evolutionary phases. In the first phase, mucus discharges diffusely from the excretory duct into the connective tissues. Followed by resorption phase, in this granuloma formation occurs because of foreign body reaction. In the final phase, there is formation of pseudo capsule (without epithelial lining) around the mucous, so it is called pseudocyst. Salivary duct blockage causing decreased or absence of secretion causes mucus retention cyst.[20]

Histologically, the presence of a well-demarcated interstitial mucin surrounded by a granulation tissue containing neutrophils and multinucleated giant cells in the submucosa serves as a peculiar feature to diagnose mucus extravasation phenomenon.[21]

The cyst-like cavity ("pseudocyst") of a mucus retention cyst is lined by normal ductal/ stratified squamous epithelium to pseudostratified epithelium. The cyst-like lumen contains mucin obstructed by a sialolith. The connective tissue around the lesion and adjacent minor salivary glands shows inflammatory changes.[20]

Management of mucocele involves local surgical excision. Sometimes, it will rupture and heal by itslef.[21]

8.2. Necrotizing sialometaplasia

It is a reactive and necrotizing lesion affecting minor salivary glands present in hard palate. Etiology is reduced or lack of blood supply causing infarct of salivary gland tissue. Local anaesthesia, poorly fitting dentures, recent tooth extraction, surgery along with radiation therapy are precipitating factors for this lesion.[22]

Clinically, it presents as a deep ulcer. It appears suddenly and some patients may experience fever, chills, restlessness initially. The posterior region of hard palate is commonly affected followed by junction of hard and soft palate. About 90% of lesions are present unilaterally.[23]

Microscopically, it shows chronic inflammatory infiltration, proliferated vascular areas and partial necrosed areas of the glands, with squamous metaplasia of ductal and acinar areas and presence of mucin pools, lined by ulcerated epithelium. Mucous cells are seen within the salivary gland lobules, simulate a low grade mucoepidermoid carcinoma cytologically.

Maintenance of lobular architecture of the glands is characteristic feature of this lesion to distinguish it from neoplasms.[24]

It resolves on its own, mostly within 5-6 weeks and therefore no treatment is required.

9. Lesions Involving Nerves

9.1. Traumatic neuroma

It is reactive response of neural tissue due to trauma or surgery, mostly occurring due to transection or damage to the nerves bundle.[25] In oral cavity, it is rare and affected sites are solitary nodule of the mental foramen, lower lip, or tongue.[26] The most affected location is the posterior mandible, due to damage of the inferior alveolar nerve following tooth extraction or sagittal ramus split during osteotomy. The most common symptom is pain, due to compression of nerves by the tumour.[25,26]

Clinically, presents as a firm nodule lesser than 2cm in size which is slow growing and painful. Some patients may experience paraesthesia over the injured area and dysesthesia. Presence of a specific trigger point for pain in the area of a neuroma may be characteristic feature.[27]

Histopathologically, they are non-encapsulated lesions, containing a large amount of haphazardly arranged nerve fascicles, within a densely collagenous and fibroblastic

stroma. Occasionally, scar tissue may also be seen. Immunohistochemical studies may be needed to achieve the final diagnosis, with S-100 being the single best antibody, whereas antibodies to EMA, CD57, and collagen IV are of secondary value.[5,27]

Management of traumatic neuroma consists of surgical resection of lesion alongwith involved nerve bundle.

10. Lesions Involving any Site

10.1. Irritational fibroma

It is a reactive lesion occurring in any region of oral cavity due to any local irritation or injury, but it is seen most often on the buccal mucosa along the plane of occlusion of the maxillary and mandibular teeth.[28]

Clinically, it presents as an exophytic, firm, yellowish–white or normal mucosa coloured, sessile growth. Due to trauma, ulceration or hyperkeratotic areas are seen on surface. Commonly affected region is buccal mucosa- through the occlusal line due to masticatory trauma – but it also seen in other areas of oral cavity as well.[5,28]

Histopathologically, the surface epithelium is atrophic, but due to repetitive trauma, hyperkeratosis, intracellular edema of the above layers or ulcerated areas may be present. Dense collagen bundles and focal hyperplasia of mature fibroblasts are seen in connective tissue with either little or no inflammatory areas. The collagen bundles arrangement pattern is unique. A radiating and circular pattern of collagen bundles can be seen.[29]

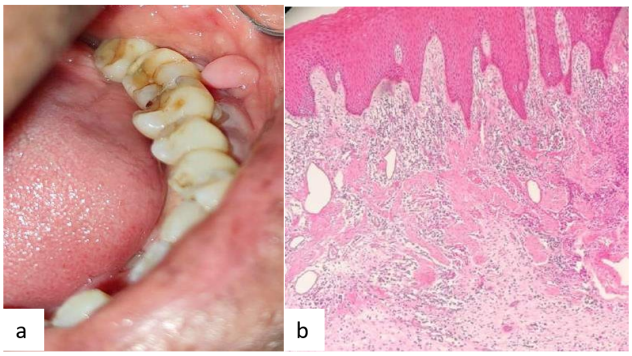

Figure 5: a): An exophytic growth present on buccal mucosa along the line of occlusion of maxillary and mandibular teeth; **b**): Haematoxylin and eosin-stained section 40 x magnification showing stratified squamous epithelium with hyperplasia of rete ridges, connective tissue stroma shows radiating and circular pattern of collagen bundles. Chronic inflammatory infiltrate are seen

Management involves eliminating the source of irritation and local surgical excision of lesion.

10.2. Traumatic ulcerations

Traumatic ulcers are most common on the buccal mucosa (28.5%), tongue (16.6%), and lower lips (8.3%). It results from physical, chemical, or thermal injuries. Teeth can alter the soft tissues of the mouth due to improper positioning, sharp or jagged edges due to tooth decay or fractures, or defective restorations.[30]

As the oral cavity has thin mucosal lining as compared to epithelium of skin, it is more prone to trauma. The traumatic injuries to the oral mucosa can break the integrity of the oral epithelium which is referred to as ulcer. Oral ulcer may be present as superficial erosion or extend deep into the connective tissue that causes painful ulceration.[31]

Microscopically, an abrupt ulcer is covered by a very thick fibrinopurulent exudate or membrane. The immediate adjacent epithelium will show pseudoepitheliomatous hyperplasia, while the ulcer bed contains granulation tissue. The granulation tissue shows an endothelial proliferation with infiltration of inflammatory cells, including lymphocytes, histiocytes, neutrophils, eosinophils, and even plasma cells. Mitotic figures are usually easily identified, while necrosis tends to be limited to thermal or electrical injury cases.[32]

Traumatic ulcerations heal fast after incisional biopsy, even with the large eosinophilic ulcerations. Treatment involves removal of source of injury.[31,32]

10.3. Giant cell fibroma

It is a benign sessile or pedunculated growth and it occurs due to chronic irritation and is characterized by functional changes in the fibroblastic cells. The term giant cell fibroma is because of its characteristic large, stellate-shaped, mononuclear, and multinucleated giant cells which are in fact ‘fibroblasts’. Commonly affected region is mandibular gingiva (retrocuspid papilla of the mandible), followed by the maxillary gingiva, the tongue, and the palate with a slight female predilection.[33,34]

Microscopic examination shows dense haphazardly arranged collagen fibres. Stellate shaped giant cells are seen in the connective tissue immediately below the epithelium with short dendritic processes and large hyperchromatic nuclei. Overlying epithelium is keratinized stratified squamous showing mild hyperplasia, with elongated and thick rete ridges.[35]

Treatment involves complete surgical excision of lesion and recurrence is rare.

10.4. Frictional keratosis

It is a reactive keratotic lesion that occurs mainly due to chronic mechanical irritation caused by sharp edges of teeth or overhanging restorations, dental prosthesis, vigorous tooth brushing, and by parafunctional habits such as constant rubbing, chewing, or sucking of the oral

mucosa against the teeth can result in keratoses of the buccal mucosa (morsicatio buccarum), tongue (morsicatio linguarum) and lip. Depending upon degree of trauma, it may vary clinically. Linea alba is the term used to describe the white keratotic line on the buccal mucosa along the occlusal plane and it may be unilateral or bilateral.[14]

Clinically, it presents as an ill-defined area of gray or white papules and plaques and can be associated ulcers if the bite trauma is extensive. In some instances, affected area show a macerated appearance with shredded keratin and peeling.[35]

Microscopic examination shows marked hyperparakeratosis with a shaggy or shredded keratin. The epithelium also shows epithelial hyperplasia and intracellular edema is present in the spinous layer as ballooned cells.[35] If secondary ulceration is present, inflammatory areas are present in superficial connective tissue. In these instances, normal mitotic figures may be present in the basal or parabasal layer, but the features of epithelial dysplasia are absent.[35]

10.5. Oral mucosal peeling

It is mild type of chemical burn which occurs due to use of toothpastes and mouthwashes containing sodium lauryl sulfate (SLS) and mouthwashes containing essential oils and chlorhexidine (CHX).[36] OMP refers to an asymptomatic white mucosal lesion occurs anywhere in oral cavity that can be scraped off.[37]

Histopathologically, the overlying epithelium is typically atrophic and hyperkeratotic and may show epithelial dysplasia.[37]

The problem resolves with a switch to another, blander toothpaste, or mouth rinse.[37]

11. Conclusion

Reactive lesions are the common entities of the oral cavity which occur in response to some kind of irritation or trauma. Clinically some of these lesions may simulate neoplastic processes therefore it is essential in the part of the clinician to be aware of clinicopathological correlations to develop the list of differential diagnosis. The etiopathogenesis, treatment and biologic behaviour of these reactive lesions differ considerably. Hence, clinicians must be familiar with these commonly occurring pathoses for the management.

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None.

13. Conflict of Interest

None.

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