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Pyogenic granuloma of tongue – A rare case report

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ABSTRACT

Pyogenic granuloma (PG) is a kind of inflammatory hyperplasia in response to chronic irritation. Here, we report a case of 64 year old male patient with PG on midline of the dorsum of the posterior third of the tongue. Its differential diagnosis, the importance of biopsy findings in establishing definitive diagnosis and about the new approaches for its treatment is discussed.

Key words: Pyogenic granuloma, Tongue, Midline

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INTRODUCTION

Oral Inflammatory hyperplasias include pyogenic granuloma, peripheral giant cell granuloma, epulis fissuratum, irritation fibroma, cemento-ossifying fibroma, inflammatory fibrous hyperplasia, and inflammatory papillary hyperplasia. The term pyogenic granuloma is a misnomer as the cause is traumatic and not infectious. Pyogenic granuloma seen in the oral cavity arises in response to various stimuli such as local irritation, trauma or hormonal factors. It usually occurs on gingiva but uncommonly it can occur on extragingival sites such as lips, tongue and buccal mucosa.[1] Characteristically, pyogenic granuloma of tongue is more common on the lateral side of the tongue which may be related to trauma from adjacent teeth or dentures.[2] It usually presents in second decade of life.

We present a case with lesion in the midline of the dorsum of the posterior third of the tongue in order to enlighten the readers to keep pyogenic granuloma as a differential diagnosis while suspecting tumors of oral cavity especially in older patients.

CASE REPORT

A 64 year old male reported to the outpatient department with a six month history of slowly enlarging growth in the midline of the dorsum of the posterior third of the tongue. He was a non-smoker and was able to eat and drink normally. Past history was unremarkable. Physical examination revealed no abnormalities or cervical lymphadenopathy. There were no constitutional symptoms such as loss of appetite or weight loss. Intraoral examination revealed the oral hygiene to be poor and the patient had severe haliotosis. A sessile, bluish growth (1 cms × 0.5 cms) in the midline of the dorsum of the posterior third of the tongue was noticed (Figure 1). It was firm and non-tender on palpation but it caused the lesion to bleed significantly. A differential diagnosis of haemangioma or pyogenic granuloma was made. After routine laboratory investigations, as the lesion was small an excisional biopsy was done under local anaesthesia as treatment and the defect was closed with vicryl.

Histopathological evaluation revealed the lesion comprising of vascular channels of varying sizes, lined partly by keratinizing stratified squamous epithelium. The stroma was infiltrated by mononuclear cells with interspersed polymorphs. There was no evidence of malignancy. The appearance was consistent with that of a pyogenic granuloma (Figure 2).

Poor oral hygiene may be the precipitating factor in this case. Scaling and root planing of teeth was done and patient was advised to maintain oral hygiene with the help of soft toothbrush and to clean the tongue on regular basis.

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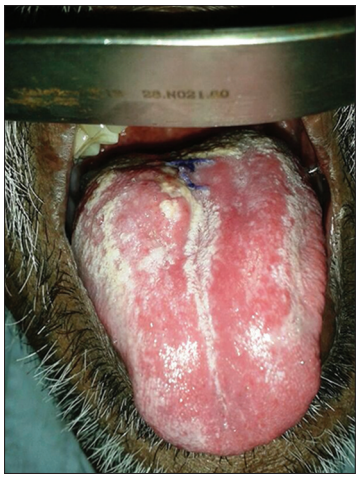

Figure 1: Intraoral photograph showing PG on dorsum of tongue

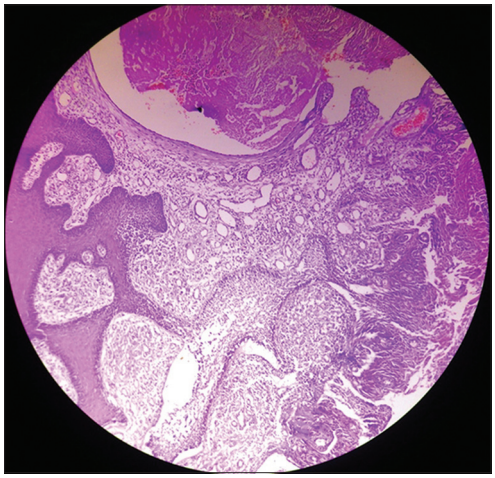

Figure 2: Photomicrograph showing proliferation of capillaries with the overlying squamous epithelium and infiltration by inflammatory cells (H& E, 40x)

DISCUSSION

The most common sites in the oral cavity for PG are gingiva (61%), lip (14%), tongue (9%) and buccal mucosa (7%).[3] Although PG may occur in all ages, it is predominant in the second decade of life in young adult females, possibly because of the vascular effects of female hormones.[1] It is now generally accepted that the lesion is an exaggerated localized connective tissue reaction to minor injury or irritation.[4] The usual clinical presentation of pyogenic granuloma is a small, deep red to reddish-purple lesion which is either sessile or pedunculated. The surface may be smooth, lobulated or occasionally warty which is commonly ulcerated and shows a tendency for hemorrhage either spontaneously or upon slight trauma. The lesion is painless and soft in consistency; although older lesions tend to become more collagenized and firm. The size of the lesion usually ranges between 0.5 cm-2.5 cm, and they may grow at an alarming rate within weeks or months and remaining indefinitely thereafter.[5]

The pathogenesis of PG at molecular level may be considered as the imbalance of angiogenesis enhancers and inhibitors.[1] There is over production of Vascular Endothelial Growth Factor (VEGF), basic Fibroblast Growth Factor (bFGF) and decreased amounts of angiostatin, thrombospondin-1 and estrogen receptors leading to formation of PG. Although PG can be diagnosed clinically with considerable accuracy, radiographic and histopathological investigations aid in confirming the diagnosis and instituting adequate treatment. Radiographs are advised to rule out bony destructions suggestive of malignancy or to identify a foreign body. A definite diagnosis of PG can only be made by histopathological examination of the biopsied tissue.

Histopathologically, PG is partly or completely covered by parakeratotic or non-keratinized stratified squamous epithelium. Major bulk of the lesion is formed by a lobulated or a non lobulated mass of angiomatous tissue. Usually, lobulated lesions are composed of solid endothelial proliferation or proliferation of capillary sized blood vessels. The amount of collagen in the connective tissue of PG is usually sparse. Surface can be ulcerated and in such ulcerated lesions, edema is a prominent feature and the lesion is infiltrated by plasma cells, lymphocytes and neutrophils.[6]

Immunohistochemically, PG lesions express factor VIII – related antigen positivity in the endothelial cells lining large vessels, but are negative in the cellular areas, whereas Ulex europaeus I lectin binds to endothelial cells in both large vessels and cellular aggregates. There is enhanced expression of the bFGF, Tie-2, anti-CD34 and anti alpha SMA antibodies, and vascular morphogenesis factors such as angiopoietin-1, angiopoietin-2, ephrinB2, and ephrinB4. There is also expression of inducible nitric oxide synthase, increased expression of vascular endothelial growth factor, low apoptotic rate expression of Bax/Bcl-2 proteins and strong expression of phosphorylated mitogen activated protein kinase. Polymerase chain reaction investigations for human papilloma virus and human herpes virus type have yielded negative results.[7]

Differential diagnosis of PG includes peripheral giant cell granuloma, peripheral ossifying fibroma, hemangioma, peripheral fibroma, leiomyoma, hemangioendothelioma, hemangiopericytoma, bacillary angiomatosis, kaposi's sarcoma, angiosarcoma, Non– hodgkin's lymphoma, metastatic tumor, post extraction granuloma and pregnancy tumor.[8]

Therapeutically, several options are available for PG. Firstly, the causative agent must be removed, which often results in spontaneous resolution. Secondly, adequate excision may be carried out.[9] Other surgical modalities for the treatment of pyogenic granuloma reported are cryosurgery in the form of either liquid nitrogen spray or a cryoprobe, excision by Nd: YAG, CO2 and flash lamp pulsed dye lasers, injection of ethanol or corticosteroid and sodium tetradecyl sulphate sclerotherapy.[1] A newly developed tool, ultrasonic scissors which are used in abdominal and thoracic surgeries can be safely utilized to treat this lesion on the tongue as it reduces blood loss and operative time markedly.[10]

CONCLUSION

Poor oral hygiene, especially an uncleaned tongue can give rise to pyogenic granuloma of the tongue. Proper management including diagnosis, treatment and further prevention is very important. The clinical features of pyogenic granulomas of the oral cavity, although are helpful in making a differential diagnosis but are inadequate for a definite diagnosis in a given case. Further, their clinical resemblance to other inflammatory tumors, as well as to some true neoplasms of the oral cavity, necessitate an appropriate microscopic examination of the biopsy specimens, before the final diagnosis is made and adequate treatment is instituted.

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Authors Contribution:
SS, AS – Conceived and designed the study, made the first draft of the manuscript; **SS, AS, AK –** Concept and design of the study, analysis and interpretation of data; **AS, JK, SK –** Literature searches and manuscript preparation.

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