International Journal of Science and Research (IJSR)
ISSN: 2319-7064
ResearchGate Impact Factor (2018): 0.28 | SJIF (2018): 7.426

Oral Pyogenic Granuloma: A Case Report and a Comprehensive Review

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Abstract: ***Pyogenic granuloma (PG) is considered a reactive tumor like lesion seen in the oral cavity caused due to low-grade local irritation, traumatic injury, or hormonal factors. It occurs most commonly in the gingiva, and also lips, tongue and buccal mucosa are the other common sites. The present case reports a localized inflammatory hyperplasia of the maxillary gingiva of a 29 year-old female patient which was interfering with occlusion. Excisional biopsy of the lesion revealed findings suggestive of PG. The patient showed no sign of recurrence in a 6 months follow-up. This article seeks to give a comprehensive review of oral PG with its etiology, clinical features, treatment, and multiple differential diagnoses.***

Keywords: Gingiva, inflammatory hyperplasia, pyogenic granuloma

1. Introduction

Pyogenic granuloma (PG) is an inflammatory hyperplasia describing a large range of nodular growths of the oral mucosa.[1,2] PG is a common non neoplastic growth of the oral cavity, and the first case was described by Hullihen in 1844.[3] Even though various terms have been proposed earlier, Hartzell in 1904 gave the current term of PG or granuloma pyogenicum.[4] Although it is a common disease in the skin, it is rare in the gastrointestinal tract, except for the oral cavity, [5] and it is mostly found in keratinized mucosa.[6] There are various factors such as chronic low-grade irritation,[7,8] trauma, hormonal factors,[9] and certain kinds of drugs[10] which are proved to be the causative factors in the development of PG. Oral PGs occur in the gingiva in 75% of cases, and precipitating factors include poor oral hygiene, local irritants, and foreign material in gingiva. Although many lesions occurring in the oral cavity have got similar appearance as PG, a detailed history, clinical examination, and a proper treatment plan will be helpful to pinpoint the disease. In this article, I will present a case report of a large pyogenic granuloma of the gingiva in a 29-year-old female patient and also will review the literature in detail.

2. Case Report

A 29-year-old female patient reported to the Dental OPD with the chief complaint of a growth in the gums in the upper right back tooth region since 3 months. The growth initially started as a small one, which progressively increased to the present size. There was no pain until 1-month back, but since the growth increased in size, the patient gave history of pain on mastication, and also due to impinging opposing teeth. The patient also stopped brushing in the area due to excessive bleeding from that region. Her medical and family histories were noncontributory, and general physical examination revealed no other abnormalities. There were no relevant abnormal findings extra orally, and there were no palpable regional lymph nodes. Intraoral examination revealed a poor oral hygiene. Soft tissue examination revealed a well-defined gingival growth in the 14-15 region extending on the buccal aspect. The growth was reddish pink in color around 5 cm × 4 cm in size. The surface of the growth was irregular and nodular. Fig 1. The growth was non tender and firm on palpation and on probing, there was severe bleeding from the site. A provisional diagnosis of PG of the gingiva was made. Differential diagnosis of irritational fibroma and peripheral giant cell granuloma were given.

Hematological and biochemical investigations, which were carried out were all within normal limits.(Table 1). OPG view was also taken, which showed no bone loss, suggestive of no other bony pathology in relation to 14-15 region. Fig 2. Phase 1 therapy was given and patient recalled after one week for checkup. An excisional biopsy was planned, and the excised tissue was sent for histopathological examination. The biopsy report revealed parakeratinized epithelium and connective tissue showing engorged dilated blood vessels, extravasated red blood cells, inflammatory cells, and collagen fibers. A diagnosis of PG was histologically confirmed. Fig 3. The patient was recalled after 1 month, Fig 4. and from then, with regular visits for the past 6 months and there was no recurrence of the lesion so far.

3. Discussion

PG is a kind of inflammatory hyperplasia and also termed as granuloma pyogenicum.[12] PG is a misnomer because the lesion does not contain pus and is not a granuloma also.[13] Histologically, it is described by Angelopoulos as hemangiomatous granuloma due to increase in blood vessels[14] and also termed as granuloma telangietacticum according to Cawson *et al.*[15] It was also named a Crocker and Hartzell's disease. There are 2 forms of PG - the lobular capillary hemangioma (LCH) and the non-LCH.[11] PG is caused by a known stimulant such as calculus or foreign material in the gingival crevice resulting in a proliferation of connective tissue.[8] In addition, one-third of the lesions occur after trauma. Ainamol suggested that routine tooth brushing habit caused repeated trauma to gingiva, resulting in these lesions.[16] Furthermore, release

of variety of endogenous substances and angiogenic factors, trauma to deciduous teeth,[17] aberrant tooth development,[18] occlusal interferences,[19] drugs such as cyclosporine[20] and selection of wrong healing cap for implants are some of the precipitating factors for PG.[21]

Oral PGs occur in all age groups, children to older adults, but frequently seen in females in the second decade due to increased levels of hormones.[22] It appears as an elevated sessile or pedunculated growth covered with red hemorrhagic and erythematous papules and show ulcerations and is covered by a fibrinous membrane.[7,8] The color varies from red, purple pink, depending on the vascularity of the growth.[23] Gingiva is affected primarily, especially the marginal gingiva and common in maxillary than mandibular gingiva. Anterior areas are more frequently affected than posterior areas. All these lesions are more common on the facial than the lingual aspect.[7] Clinically, the lesion can be slow-growing, asymptomatic and painless, but it may also grow rapidly sometimes.[8] Radiographic findings are usually absent, [24] however, some long standing gingival PGs can cause localized alveolar bone resorption.[14]

Differential diagnosis includes peripheral giant cell granuloma, peripheral ossifying fibroma, metastatic cancer, hemangioma, pregnancy tumor, hyperplastic gingival inflammation, Kaposi's sarcoma, bacillary angiomatosis, angiosarcoma, and non-Hodgkin's kymphoma.[25] Peripheral giant cell granuloma is clinically similar to PG, but bone resorption in radiograph and appearance of the multinucleated giant cell are differentiating features.[8] Also, fibroma can be distinguished by the consistency, texture, and the lighter color.[7] Metastatic tumors, even though clinically resembles PG, the microscopic appearance resemble as the tumor of origin.[7] Hemangioma is a developmental disorder and is most commonly seen on the tongue. It can be multinodular, bluish red and can be diagnosed by a chairside procedure called diascopy.[1] Kaposi's sarcoma and bacillary angiomatosis can be differentiated histopathologically and are also AIDS related.[12] Pregnancy tumor occurs towards the end of pregnancy, and the tendency for this lesion to shrink after delivery indicates the definite role in etiology of lesions. Also, pregnancy tumor is usually confined to the interdental papilla.[26] PG can be distinguishable from angiosarcoma by its lobular growth pattern, well-formed vessels and cytologically bland endothelial cells.[27] Clinical appearance of gingival non-Hodgkin's lymphoma varies but is usually found to be an asymptomatic gingival enlargement or mass resembling a PG.[12] Histopathologically, it can be classified as an LCH and non-LCH.[7] LCH has proliferating blood vessels in lobular aggregates, no specific changes such as edema and capillary dilatation. Non-LCH type consists of vascular core resembling granulation tissue with foci of fibrous tissue. The lobular area of LCH type has a greater number of blood vessels. Oral PGs are mainly LCH type.[11] The natural course of the lesion can be in three phases of development as cellular phase, vascular phase and phase of involution.[28]

Treatment includes surgical excision of the lesion with the removal of irritants recommended for small painless lesions. Excision of gingival lesions up to periosteum with thorough scaling and root planning of adjacent teeth to remove all visible sources of irritation. [7] Various other treatment modalities include Nd: Yttrium-aluminum-garnet lasers, carbon dioxide lasers, flash lamp, pulse dye laser, cryosurgery, sodium tetradecyl sulphate sclerotherapy,[29] and use of intralesional steroids have been proposed by clinicians. Treatment of oral PG during pregnancy would depend on preventive measures such as careful oral hygiene, removal of dental plaque, and use of soft toothbrush. In some cases, shrinkage of the lesion after pregnancy may make surgical treatment unnecessary.[30] Incomplete excisions, failure of removal of etiological factors contribute to the recurrence of these lesions.[8] A recurrence rate of 16% and also a case of multiple deep satellite lesions surrounding the original excised lesion in a case of Warner Wilson James syndrome have been reported. [31] A need for regular follow-up is also emphasized because of higher recurrence rate, especially in the gingiva.[32]

4. Conclusion

This article seeks to report a large PG in the maxillary gingiva with a detailed review on the etiologies, clinical features, histopathological features, differential diagnoses, treatment modalities and recurrence rate. Even though PG is a relatively common presentation, a thorough understanding of the lesion is important to differentiate it from similar clinical presentations with proper treatment modalities gives excellent results.

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Figures

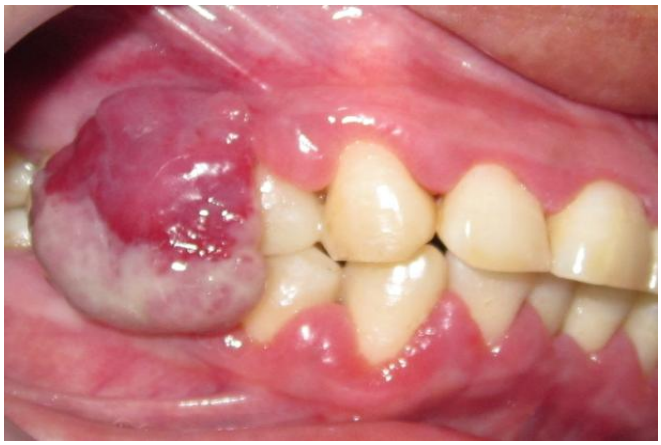

Figure 1: Clinical view of gingival growth in maxillary jaw

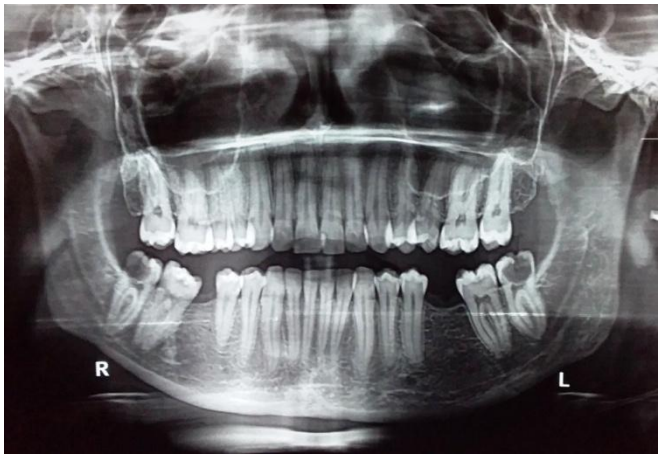

Figure 2: OPG shows no bony involvement in the maxillary region

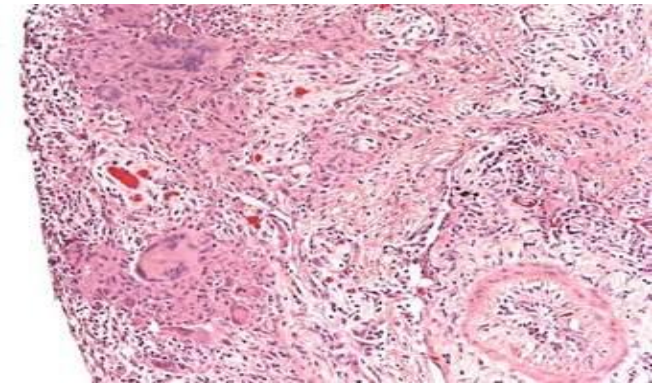

Figure 3: Photomicrograph of oral pyogenic granuloma

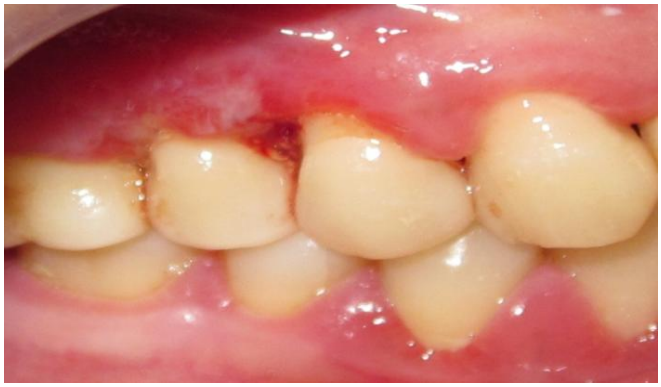

Figure 4: Post operative clinical view after 1 month.

Table 1

Hemoglobin- 13.8 gm.	WBCs- 8500/mm^3
DLC:	Neutrophils- 53%
Lymphocytes- 44%	Monocytes- 01%
Basophils- 00%	Eosinophils- 00%
CT- 6 min.40 sec.	BT- 4 min. 20 sec.
RBS- 88mg/dl	