



PYOGENIC GRANULOMA OF MARGINAL GINGIVA: REVIEW OF LITERATURE WITH A CASE REPORT OF A CLASSICAL OCCURRENCE.

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ABSTRACT

Pyogenic granuloma (PG) is described as a reactive tumor like lesion of the oral cavity occurring as a result of mild local irritation, hormonal changes or in some cases trauma. It is most commonly seen in association with the gingiva, but buccal mucosa, lips and tongue can also be affected. The current case study reports an inflammatory hyperplastic lesion of the anterior maxillary gingiva in a 23-years-old female patient which was clearly visible and was interfering with the aesthetics. An excisional biopsy of the lesion was performed and the histopathological findings confirmed Pyogenic Granuloma. There were no sign of recurrence in a year of follow-up. This article will try to explain the etiology, differential diagnosis, clinical features and treatment of oral Pyogenic Granuloma and review the currently available literature.

KEYWORDS : Inflammatory hyperplasia, tumor-like, pyogenic granuloma, nodular growth

Introduction

Pyogenic granuloma (PG) is an inflammatory hyperplastic lesion, describing a wide range of oral nodular growths.^{1,2} It is a common non-neoplastic lesion of the oral cavity, and was first described by Hüllihen in 1844.³ Various terms have been proposed earlier for this lesion, but it was in 1904 that Hartzell gave the current term of Pyogenic Granuloma or granuloma pyogenicum.⁴

It is a commonly occurring disorder of the skin but is rarely seen in the gastrointestinal tract, the oral cavity being an exception,⁵ with keratinized mucosa being the most common site.⁶ Various factors contribute to the development of oral PGs including chronic mild irritation,^{7,8} hormones, trauma,⁹ and certain drugs.¹⁰ 75% of the cases of Oral PGs are in the gingiva, precipitating factors mainly being local irritants, poor oral hygiene, and foreign bodies.¹¹ This article presents a case report of a moderately sized Pyogenic granuloma in the marginal gingiva in a 23-years-old female patient and also will review the currently available literature relevant in particular to describe the concerned case.

Case-report

A 23-years-old female patient reported to the clinic with the chief complaint of a growth in her gums in the upper right front tooth region since 8 months. It initially began as a small growth in upper front jaw region which progressively increased to attain the present size. There was no pain until 1-month back, but since the growth increased in size, it further got associated with mild, intermittent non-radiating pain and bleeding on chewing food and pressing the swelling making it difficult for the patient to brush the teeth that area. Patient had also undergone excision for same 8 months back from some local doctor but the growth again reoccur after 3 days. 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 fair oral hygiene. Soft tissue examination revealed a well-defined gingival growth in the 11, 12 teeth region extending on the labial aspect [Figure 1]. The growth was reddish pink in color around 2 cm × 1.5 cm in size with an irregular and nodular surface [Figure 1]. The growth was Soft in consistency non tender & compressible. Bleeding on provocation was present. No osseous bony involvement was felt. A provisional diagnosis of PG of the gingiva was given. Differential diagnosis of irritational fibroma Hemangioma, Peripheral Giant cell Granuloma, Peripheral Ossifying Fibroma and Lymphangioma were given. Hematological and biochemical investigations were all within normal limits. A surgical excisional biopsy was carried out, and the excised tissue specimen was sent for histopathological investigation [Figure 2]. The histopathological slides revealed parakeratinized epithelium and connective tissue showing engorged dilated blood vessels, extravasated red blood cells, inflammatory cells, and collagen fibers [Figure 3]. Blood vessels

showed endothelial cell proliferation along with areas of necrosis [Figure 4]. A diagnosis of PG (LCH Type) was histologically confirmed. The patient was recalled after 3 months, and from then, with regular visits for the past 1 year and there was no recurrence of the lesion so far.

Discussion

Pyogenic Granuloma is a kind of inflammatory hyperplastic lesion, 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, Angelopoulos described it as hemangiomatous granuloma formed because of increase in blood vessels.¹⁴ Cawson *et al.* termed it as granuloma telangiecticum.¹⁵

PG is classified in 2 categories- the lobular capillary hemangioma (LCH) and the non-LCH.¹¹

PG is caused by stimulants such as calculus or foreign material in the gingival crevice resulting in a proliferation of connective tissue.⁸ Also one-third of these lesions occur due to trauma. As per Ainamol, routine tooth brushing habit lead to repeated trauma to gingiva, forming these lesions.¹⁶ Some studies discuss the role of factors like release of variety of endogenous substances and angiogenic factors, trauma to deciduous teeth,¹⁷ aberrant tooth development,¹⁸ occlusal interferences,¹⁹ drugs such as cyclosporine²⁰ and selection of wrong healing cap for implants as factors for development of oral PG.²¹

Oral PGs are seen in all ages, but are most commonly seen in females in the second decade due to increased hormonal changes.²² It is usually a sessile or pedunculated mass appearing as a erythematous papule, sometimes showing ulcerations and is covered by a fibrinous membrane.^{7,8} The color variations range from pink to red to purple, depending on the lesion's vascularity.²³ Gingiva is mostly affected, especially the maxillary marginal gingiva compared to mandibular gingiva. Anterior jaw region is more frequently affected than posterior with facial aspect affected more commonly than the lingual aspect.⁷ Radiographic findings are not usually seen,²⁴ however, in long standing gingival lesions a localized alveolar bone resorption is sometimes visible.¹⁴

Differential diagnosis comprises of lesions like hyperplastic gingival inflammation, peripheral giant cell granuloma, peripheral ossifying fibroma, hemangioma, pregnancy tumor, bacillary angiomatosis, Kaposi's sarcoma, metastatic cancer, angiosarcoma, and non-Hodgkin's lymphoma.²⁵ Peripheral giant cell granuloma clinically appears very similar to PG, the only differentiating features being radiographic bone resorption and presence of the multinucleated giant cell.⁸

Fibroma on the other hand can be marked off on the basis of the

consistency, texture, and the lighter color.⁷ Hemangiomas are of developmental origin and are commonly seen on the tongue presenting as a multinodular, bluish red growth.¹ Pregnancy tumor occurs towards the end of pregnancy with the tendency to shrink after delivery indicating the definite etiology of lesions. Also, this lesion is usually limited to the interdental papilla.²⁶ Bacillary angiomatosis and Kaposi's sarcoma are AIDS associated and can be differentiated.¹² Metastatic tumors, microscopically resemble the tumor of origin.⁷ PG shows a lobular growth pattern, bland endothelial cells and well-formed vessels and can be clearly differentiated from angiosarcoma.²⁷ Gingival non-Hodgkin's lymphomas are usually found to be present as an asymptomatic gingival lesion resembling a PG.¹² Oral PG can be classified as an LCH and non-LCH only after histopathology.⁷ LCH shows blood vessels proliferating in a lobular pattern in contrast to Non-LCH type that shows a vascular core resembling granulation tissue with foci of fibrous tissue. Most oral PGs are LCH type. Pyogenic granuloma shows a developmental course in three phases namely as cellular phase, vascular phase and phase of involution.²⁸ Treatment recommended for small painless lesions includes surgical excision of the lesion with the removal of irritants and for larger lesions excision of lesions up to periosteum followed by thorough scaling and root planning of adjacent teeth to remove all visible sources of irritation.⁷ Various other treatment modalities are also present including Nd: Yttrium-aluminum-garnet lasers, carbon dioxide lasers, flash lamp, pulse dye laser, cryosurgery, sodium tetradecyl sulphate sclerotherapy.²⁹ Oral PG during pregnancy can be treated with 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 Warner Wilson James syndrome shows a recurrence rate of 16% and also a case of multiple deep satellite lesions surrounding the original excised lesion.³¹ Regular follow-up is also necessary because of higher recurrence rate, especially in gingival lesions.³²

Conclusion

This article reports a classical case of Pyogenic Granuloma in the anterior maxillary gingiva with a detailed review of literature explaining the etiologies, clinical features, histopathological presentations, differential diagnoses, treatment modalities, and recurrence rate.

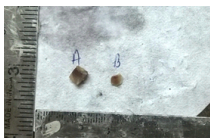
Figures

Figure 1



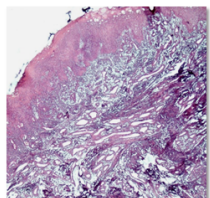
A well-defined gingival growth in the 11, 12 teeth region, extending on the labial aspect.

Figure 2



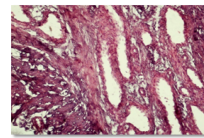
Excised soft tissue biopsy specimens.

Figure 3



The H&E stained sections show the presence of parakeratinized stratified squamous epithelium overlying connective tissue showing vessels of varying diameters.

Figure 4



The given H&E stained slide shows blood vessels showing endothelial cell proliferation along with areas of necrosis and chronic inflammatory cell infiltrate.

REFERENCES

- Greenberg MS, Glick M. *Burket's Oral Medicine. Diagnosis and Treatment*. 11 th ed. McGraw Hill; 2003. p. 130-4.
- Eversole LR. *Clinical Outline of Oral Pathology. Diagnosis and Treatment*. 3 rd ed. Hamilton: BC Decker; 2002. p. 113-4.
- Hullihen SP. Case of aneurism by anastomosis of the superior maxillae. *Am J Dent Sci* 1844;4:160-2.
- Hartzell MB. Granuloma pyogenicum. *J Cutan Dis Syph* 1904;22:520-5.
- Yao T, Nagai E, Utsunomiya T, Tsuneyoshi M. An intestinal counterpart of pyogenic granuloma of the skin. A newly proposed entity. *Am J Surg Pathol* 1995;19:1054-60.
- Fowler EB, Cuenin MF, Thompson SH, Kudryk VL, Billman MA. Pyogenic granuloma associated with guided tissue regeneration: A case report. *J Periodontol* 1996;67:1011-5.
- Neville BW, Damm DD, Allen CM, Bouquot JE. *Oral and Maxillofacial Pathology*. 2nd ed. Philadelphia: Saunders; 2002. p. 437-95.
- Regezi JA, Sciubba JJ, Jordan RC. *Oral Pathology and CLINICAL Pathological Considerations*. 4th ed. Philadelphia: WB Saunders; 2003. p. 115-6.
- Mussalli NG, Hopps RM, Johnson NW. Oral pyogenic granuloma as a complication of pregnancy and the use of hormonal contraceptives. *Int J Gynaecol Obstet* 1976;14:187-91.
- Miller RA, Ross JB, Martin J. Multiple granulation tissue lesions occurring in isotretinoin treatment of acne vulgaris - successful response to topical corticosteroid therapy. *J Am Acad Dermatol* 1985;12 (5 Pt 1):888-9.
- Gomes SR, Shakir QJ, Thaker PV, Tavadia JK. Pyogenic granuloma of the gingiva: A misnomer? - A case report and review of literature. *J Indian Soc Periodontol* 2013;17:514-9. [PUBMED]
- Jafarzadeh H, Sanatkhami M, Mohtasham N. Oral pyogenic granuloma: A review. *J Oral Sci* 2006;4:167-75.
- Bouquot JE, Nikai H. Lesions of oral cavity. In: Gnepp DR, editor. *Diagnostic Surgical Pathology of Head and Neck*. Philadelphia: WB Sanders; 2001. p. 141-233.
- Angelopoulos AP. Pyogenic granuloma of the oral cavity: Statistical analysis of its clinical features. *J Oral Surg* 1971;29:840-7.
- Cawson RA, Binnie WH, Speight PM, Barrett AW, Wright JM. *Ucas Pathology of Tumours of Oral Tissues*. 5th ed. Missouri: Mosby; 1998. p. 252-4.
- Ainamo J. The effect of habitual toothcleansing on the occurrence of periodontal disease and dental caries. *Suom Hammaslaak Toim* 1971;67:63-70.
- Aguilo L. Pyogenic granuloma subsequent to injury of a primary tooth. A case report. *Int J Paediatr Dent* 2002;12:438-41.
- Milano M, Flaitz CM, Bennett J. Pyogenic granuloma associated with aberrant tooth development. *Tex Dent J* 2001;118:166-72.
- Widowati W, Ban T, Shareff A. Epulis and pyogenic granuloma with occlusal interference. *Maj Ked Crigi* 2005;38:52-5.
- Bachmeyer C, Devergie A, Mansouri S, Dubertret L, Aractingi S. Pyogenic granuloma of the tongue in chronic graft versus host disease. *Ann Dermatol Venerol* 1996;123:552-4.
- Dojcinovic I, Richter M, Lombardi T. Occurrence of a pyogenic granuloma in relation to a dental implant. *J Oral Maxillofac Surg* 2010;68:1874-6.
- Ojanotko-Harri AO, Harri MP, Hurttia HM, Sewon LA. Altered tissue metabolism of progesterone in pregnancy gingivitis and granuloma. *J Clin Periodontol* 1991;18:262-6.
- Mubeen K, Vijayalakshmi KR, Abhishek RP. Oral pyogenic granuloma with mandible involvement. An unusual presentation. *J Dent Oral Hyg* 2011;3:6-9.
- Kamal R, Dahiya P, Puri A. Oral pyogenic granuloma: Various concepts of etiopathogenesis. *J Oral Maxillofac Pathol* 2012;16: 79-82. [PUBMED]
- Calonje E, Wilson Jones E. Vascular tumours: Tumours and tumour like conditions of blood, vessels and lymphatics. In: Elder D, Elenitsas R, Jaworsky C, Johnson BJ, editors. *Lever's Histopathology of the Skin*. 8th ed. Philadelphia: Lippincott-Raven; 1997. p. 895.
- Sonis ST, Fazio RC, Fang LS. *Principles and Practice of Oral Medicine*. 2nd ed. Philadelphia: WB Saunders; 1995. p. 416.
- Pilch BZ. *Head and Neck Surgical Pathology*. Philadelphia: Lippincott Williams & Wilkins; 2000. p 389-90.
- Sternberg SS, Antonioli DA, Carter D, Mills SE, Oberman H. *Diagnostic Surgical Pathology*. 3rd ed. Philadelphia: Lippincott Williams and Wilkins; 1999. p. 169-74.