Case Report

Indian Journal of Dental Education
Volume 8 Number 3, July - September 2015
https://dx.doi.org/10.21088/ijde.0974.6099.8315.7

Recurrent Pyogenic Granuloma

Ratheesh A. V.*, Sujatha G. P., Shiva Prasad Shankarmurthy**, Ashok Lingappa*****

Abstract

Pyogenic granuloma is one of the common gingival epulis that occur as inflammatory hyperplasias seen in the oral cavity which appears as an overgrowth of tissue due to irritation, physical trauma or hormonal factors. The term pyogenic granuloma is misleading as it is not a true granuloma. The treatment being surgical excision, recurrence occurs in 16% of the lesions. This case report describes a recurrent pyogenic granuloma in a 12 year old boy of 8months duration.

Keywords: Pyogenic Granuloma; Gingival Hyperplasia; Recurrent Epulis; Gingival Epulis.

Introduction

Pyogenic granuloma is one of the inflammatory hyperplasias seen in the oral cavity which appears as an overgrowth of tissue due to irritation, physical trauma or hormonal factors. The term pyogenic granuloma is misleading as it is not a true granuloma. The growth is typically seen in young adults especially in pregnancy. In the oral cavity, it occurs most commonly in gingiva, followed by buccal mucosa,tongue. After excision, recurrence occurs in up to 16% of the lesions so in some cases re-excision is necessary.Recurrence may be due to incomplete excision, failure to remove etiologic factors, or re-injury of the area.

Case Report

A 12-year-old, apparently healthy boy reported to the Department of Oral Medicine and Radiology complaining of painless growth in the upper right back tooth gum region since two weeks which was insidious in onset that gradually grew in size. There was occasional bleeding from the same region while brushing teeth.

Past history revealed that similar type of swelling had appeared 8 and 3 months back which were excised from a private dental practioner.

On extra oral examination, no abnormalities were detected. Intraorally all the teeth were present and were normal. Intraoral soft tissue examination revealed a solitary pedunculated growth about 2.5 x 2.5 cm in size arising from the marginal gingiva and interdental papilla in relation to buccal side of 16 & 15 [Figure 1]. Mucosa over the growth was pale pink in color and surface appears was grainy with interspersed areas of erythema and ulceration present over the anterior part covered by yellowish grey slough. Areas of bleeding were evident. On palpation, it was soft in consistency and non tender. There was presence of bleeding on mild provocation. Grade 1 mobility was seen in relation to 15.

Routine hematological investigations were carried out and revealed normal values.

Intraoral periapical radiograph revealed 16, 15 & 14 with incomplete root formation in relation to 14 & 15. Mild widening of periodontal ligament space is present on cervical third on distal side of 15 [Figure 2].

An excision of intraoral mass was performed along with oral prophylaxis. Histopathological examination of haematoxylin and eosin stained tissues revealed parakeratinized stratified squamous epithelium with ulceration in few areas [Figure 3]. Connective tissue was composed of numerous

Author's Affiliation: *PG student **Professor ***Professor and Head, Dept. of Oral Medicine and Radiology, Bapuji Dental College and Hospital, Davangere.

Reprints Requests: Ratheesh A. V., PG student, Oral Medicine and Radiology, Bapuji Dental College and Hospital, Davangere, Karnataka - 577004.
E-mail: ratheeshav@gmail.com

dialated blood vessels and budding capillaries. The deepest part of connective tissue is highly cellular with composed of numerous plump epithelial cells and dense acute and chronic inflammatory cells. In some areas the subepithelial connective tissue shows numerous spindle shaped cells resembling myofibroblasts. There was also evidence of hemmorhagic areas. These features were suggestive of pyogenic granuloma. There was no recurrence during the past 2 years follow up of the patient.

Fig. 1: Solitary well defined pedunculated swelling is seen to arise from interdental papilla and attached gingiva in relation to 15,16

Fig. 1A

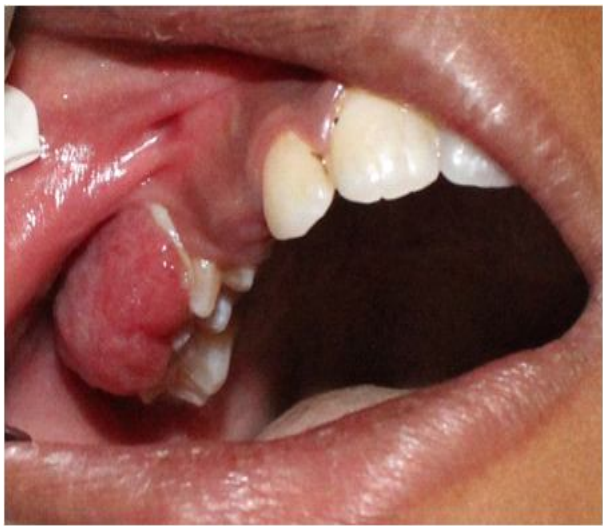


Fig. 1B

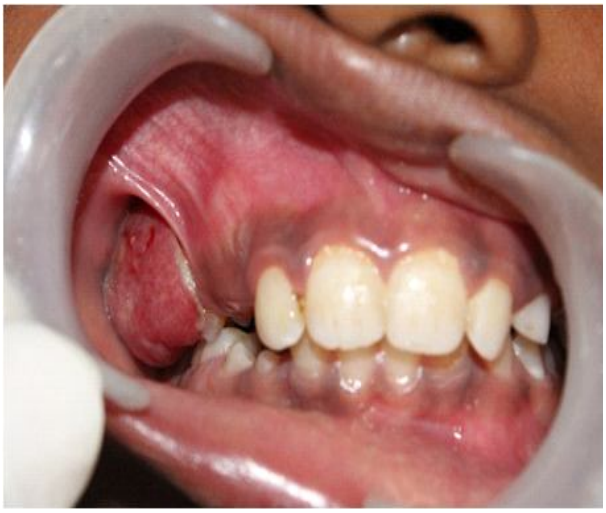


Fig. 1C

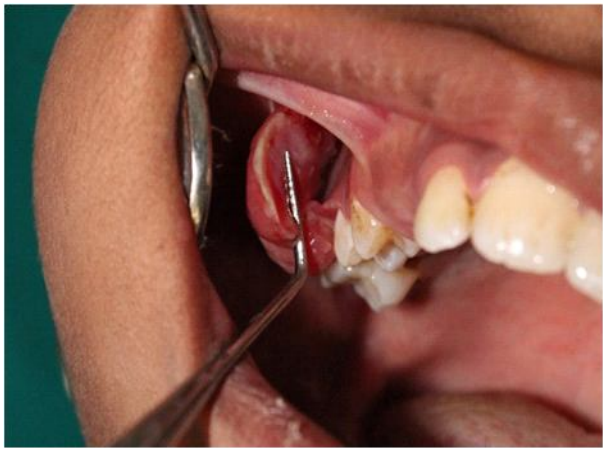


Fig. 2: IOPAR irt 15, 16 reveals mild widening of periodontal ligament space at cervical third of distal side of 15

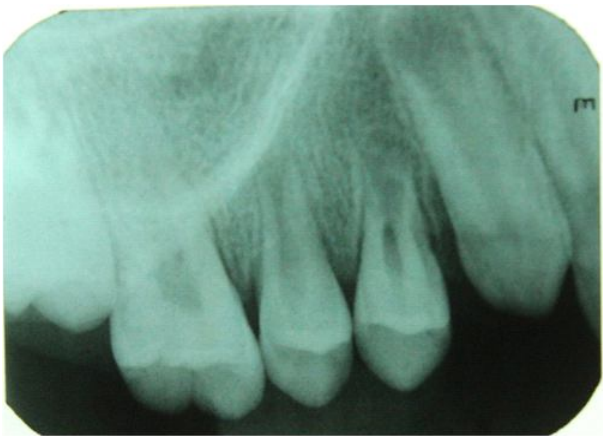
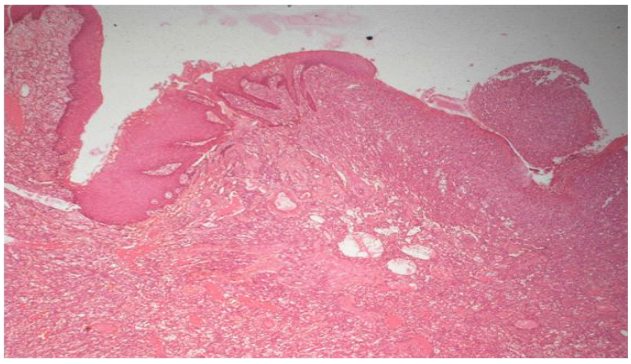


Fig. 3: H & E section of biopsy specimen reveals parakeratinized epithelium with areas of ulceration and adundant endothelial cell proliferation in connective tissue



Discussion

Soft tissue enlargements of the oral cavity may represent a variation of normal anatomic structures, inflammation, cysts, developmental anomalies, and neoplasm. Within these lesions is a group of reactive hyperplasias, which develop in response to a chronic, recurring tissue injury that stimulates an exuberant or excessive tissue repair response. Pyogenic granuloma (PG) is of the most common entities responsible for causing soft tissue enlargements. PG is a common tumor like growth seen in oral cavity and skin which is non neoplastic in nature [1,2]. In 1844 Hullihen first described PG [3]. Poncet and Dor described it as botryomycosis hominis [4]. In 1904, Hatzell termed 'pyogenic granuloma' or 'granuloma pyogenicum' [5]. Angelopoulos AP proposed the term "hemangiomatous granuloma" that accurately expresses the histopathologic picture (hemangioma like) and the inflammatory nature (granuloma) of oral pyogenic granuloma [6]. Cawson et al. suggested that since the blood vessels are so numerous in oral pyogenic granuloma, alternative term for pyogenic granuloma is granuloma telangiectacticum [7]. There are two kinds of PG namely, lobular capillary hemangioma (LCH) type and non-LCH type, which differ in histological features [8].

Incidence and Prevalance

In an analysis of 244 cases of gingival lesions in south Indian population, Shamim et al., found that nonneoplastic lesions accounted for 75.5% of cases with oral pyogenic granuloma being most frequent lesion, accounting for 52.71% cases [9]. According to Cawson et al. oral pyogenic granuloma is relatively common. It represents 0.5% of all skin nodules in children. The pregnancy tumor variant of pyogenic granuloma occurs in up to 5% of pregnancies [7]. Esmeili et al. in their review stated that hyperplastic reactive lesions represent as a group the most common oral lesions, excluding caries, periodontal, and periapical inflammatory disease. In this group, the second most common group is represented by hyperplastic reactive gingival/alveolar lesions, including inflammatory gingival hyperplasia, oral pyogenic granuloma, peripheral giant-cell lesion and peripheral cemento-ossifying fibroma [9].

Etiopathogenesis

Kerr reported staphylococci and botryomycosis, foreign bodies, and localization of infection in walls of blood vessel as contributing factors in the development of PG [10]. According to Shafer et al., oral pyogenic granuloma arises as a result of infection by either staphylococci or streptococci, partially because it was shown that these microorganisms could produce colonies with fungus-like characteristics.[11]It is also stated that oral pyogenic granuloma arises as a result of some minor trauma to the tissues that provide a pathway for invasion of nonspecific types of microorganisms. The tissues respond by the overzealous proliferation of a vascular type of connective tissue to these low virulence microorganisms.

Regezi et al., suggest that pyogenic granuloma represents an exuberant connective tissue proliferation to a known stimulus or injury like calculus or foreign material within the gingival crevice [12]. Several "etiologic factors" such as trauma, injury to a primary tooth, chronic irritation, hormones, drugs, gingival inflammation, pre-existing vascular lesions, chronic irritation due to exfoliation of primary teeth, eruption of permanent teeth, defective fillings in the region of tumor, food impaction, total periodontitis, toothbrush trauma, etc. have been suggested as etiological factors where patients presented with these findings.

Trauma has also been implicated in etiopathogenesis of multiple and satellite oral pyogenic granuloma, although, exact etiopathogenesis that whether it occurs following treatment or de novo, is not clearly understood. But various theories have been proposed. Ainamo suggested that trauma can cause release of various endogenous substances including angiogenic factors from the tumor cells and it may also cause disturbances in the vascular system of the affected area. As there is a site predilection for labial gingiva in the anterior region of the oral vestibule, some authors have postulated that habitual tooth brushing may also be considered as a significant cause of micro-trauma and irritation to the gingiva [13]. In Whitaker et al., study, it was suggested that the quantity of estrogen or progesterone receptors in oral pyogenic granuloma is not the determining factor in its pathogenesis. Rather, such a role could be attributed to the levels of circulating hormones. The levels of estrogen and progesterone are markedly elevated in pregnancy and could therefore exert a greater effect on the endothelium of oral pyogenic granuloma [14]. Inpregnancy there is inhibition of the migration of inflammatory cells and fibroblasts. Hence, it seems the co-existence of the two factors prevent acute type of tissue reaction (which keep tissues clinically healthy) to plaque, but allows an increased chronic reaction resulting clinically in an exaggerated appearance of inflammation [15].

Histopathologically there will be prominent capillary growth in hyperplastic granulation tissue in PG suggesting a strong activity of angiogenesis [12]. Angiogenesis enhancers, VEGF (decorin) and bFGF, and angiogenesis inhibitors, TSP-1 and angiostatinis associated with angiogenesis. Vascular morphogenesis factors Tie-2, angiopoietin-1, angiopoietin-2, ephrinB2, and ephrinB4 were found upregulated in pyogenic granuloma compared to healthy gingiva [16,17].

Clinical Features

Oral pyogenic granuloma occurs over a wide age range of 4.5 to 93 years with highest incidence in second and fifth decades. Females are slightly more affected than males. PG appears as elevated, smooth or exophytic, sessile or pedunculated growth covered with red hemorrhagic and compressible erythematous papules, which appear lobulated and warty showing ulcerations and covered by yellow fibrinous membrane [12,18]. The color varies from red, reddish purple to pink depending on the vascularity of the growth [19]. The gingiva, especially the marginal gingiva is affected more than the alveolar part. It is also seen in lips, tongue, buccal mucosa, hard plate, cheek, mucobuccal fold and frenum. The size varies from a few millimeters to several centimeters and it is usually slow growing, asymptomatic, painless growth, but at times it grows rapidly [20].

Radiographic Features

Usually the radiographic findings are absent in PG. But rarely it can cause localized alveolar bone resorption especially in large and long standing cases.

Histopathological Features

PG is covered by parakeratotic or non-keratinized stratified squamous epithelium. Lobulated or a non-lobulated mass of angiomatous tissue form the major bulk. 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 pyogenic granuloma is usually sparse. Surface can be ulcerated and in such ulcerated lesions, edema was a prominent feature and the lesion is infiltrated by plasma cells, lymphocytes and neutrophils [4].

Immunohistochemistry reveals factor VIII – related antigen positivity in the endothelial cells lining large vessels, but are negative in the cellular areas, whereas Ulexeuropaeus 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 ofBax/Bcl-2 proteins and strong expression of phosphorylated mitogen activated protein kinase [21].

Differential Diagnosis

Differential diagnosis of PG includes peripheral giant cell granuloma, peripheral ossifying fibroma, metastatic cancer, hemangioma, conventional granulation tissue, hyperplastic gingival inflammation, kaposi's sarcoma, angiosarcoma and non hodgkins lymphoma.

Peripheral giant cell granuloma (PGCG) is an exophytic lesion exclusively seen in gingiva and is clinically similar to PG. PGCG is bluish purple when compared to bright red color of PG. PGCG is more likely to cause bone resorption. Distinguishing features for PGCG are lack of infectious source and presence of multinucleated giant cells [12]. PG can be soft to firm in consistency and can be suggestive of peripheral odontogenic or ossifying fibroma. But the later entities are paler in color. These are also seen commonly in women like PG but are exclusively seen in gingiva only. Also there will be evidence of calcifications present in radiographs in these lesions along with involvement of periodontal ligament space. Vascular component is minimum in these types of fibromas [12,19].

Metastatic tumors are most commonly seen in attached gingiva and tongue and can resemble hyperplastic or reactive lesions like PG. it is usually seen at 5^{th} to 7^{th} decade of life [22] Hemangiomas are developmental disorders were small lesions are indistinguishable from PG. Hemangiomas give positive diascopy test. Most hemangiomas are located in tongue and are multinodular and bluish red in color. Histopathological features will be more histiocytoid, endothelial cell proliferation without an acute inflammatory cell infiltrate [1].

Conventional granulation tissue can also be considered in differential diagnosis but PG shows rapid growth, multiple occurrence and frequent recurrence [20]. Hyperplastic gingival inflammation can sometimes histopathologically mimic PG. But PG will be a distinct clinical mass compared to hyperplastic gingivitis.Kaposi's sarcoma of AIDS can also mimic clinically and histopathologically like

PG.[17]PG can be distinguished from angiosarcoma by its lobular growth pattern. Well-formed vessels and cytologically bland epithelial cells [17]. In Non-Hodgkin's lymphoma (NHL), primary sites in head and neck are waldeyer'sring,paranasal sinuses, salivary glands, oral cavity and larynx. Clinically gingival NHL varies in appearance but is usually found to be asymptomatic enlargement or mass similar to PG [23].

Treatment

Conservative surgical excision and removal of causative irritants (plaque, calculus, foreign materials, source of trauma) are the usual treatments for gingival lesions, the excision should extend down to the periosteum and the adjacent teeth should be thoroughly scaled to remove the source of continuing irritation [19]. Nd:YAG laser for excision of this lesion because of the lower risk of bleeding compared to other surgical techniques [24]. Use of flash lamp pulsed dye laser on a mass of granulation tissue which did not respond to the usual treatment methods concluded that previously resolute tissue responded well to a series of treatments with the pulsed dye laser [25]. Conservative treatment by techniques such as cryosurgery, laser surgery, and electrodessication are usually adequate, excisional treatment can often result in scars [26]. Injection of ethanol in recurrant lesions is found to be less invasive than surgical excision [27]. Another alternative is sclerotherapy with sodium tetradecy sulfate which also gives promising results [28].

Treatment considerations during pregnancy are very important. During this period, careful oral hygiene, removal of dental plaque, and use of soft toothbrushes are important to avoid occurrence of a pregnancy tumor.

Recurrence

After excision, recurrence occurs in up to 16% of the lesions so in some cases re-excision is necessary [17]. Recurrence is believed to result from incomplete excision, failure to remove etiologic factors, or re-injury of the area. Gingival cases show a much higher recurrence rate than lesions from other oral mucosal sites [2].

Conclusion

Pyogenic granuloma is a benign swelling which can cause alarming size, pain and discomfort. Thus early intervention through reinforcement of adequate oral hygiene measuresalong with surgical excision down to the periosteum is necessary to minimize the recurrence.

References

1. Neville BW, Damm DD, Allen CM, Bouquot JE Oral & maxillofacial pathology. 2nd ed, WB Saunders, Philadelphia, 2002; 437-495.
2. Vilmann A, Vilmann P, Vilmann H Pyogenic granuloma: evaluation of oral conditions. Br J Oral MaxillofacSurg 1986; 24, 376-382.
3. Hullihen SP Case of aneurism by anastomosis of the superior maxillae. Am J Dent Sc 4, 1844; 160-162.
4. Bhaskar SN, Jacoway JR. Pyogenic granuloma – clinical features, incidence, histology, and result of treatment: Report of 242 cases. J Oral Surg. 1966; 24: 391–82.
5. Hartzell MB Granuloma pyogenicum. J Cutan Dis Syph 1904; 22, 520-52.
6. Epivatianos A, Antoniades D, Zaraboukas T, Zairi E, Poulopoulos A, Kiziridou A, Iordanidis SPyogenic granuloma of the oral cavity: comparative study of its clinicopathological and immunohistochemical features. PatholInt 2005; 55: 391-397.
7. Cawson RA, Binnie WH, Speight PM, Barrett AW, Wright JM. Lucas Pathology of tumors of oral tissues. 5th ed. Missouri: Mosby; 1998. pp. 252-4.
8. Epivatianos A, Antoniades D, Zaraboukas T, Zairi E, Poulopoulos A, Kiziridou A, Iordanidis S Pyogenic granuloma of the oral cavity: comparative study of its clinicopathological and immunohistochemical features. PatholInt 205; 55: 391-397.
9. Esmeili T, Lozada-Nur F, Epstein J. Common benign oral soft tissue masses. Dent Clin North Am. 2005; 49: 223–40.
10. Kerr DA. Granuloma Pyogenicum. Oral Surg. 1951; 4: 158.
11. Shafer, Hine, Levy . Shafer's Textbook of Oral pathology. 5th ed. Amsterdam: Elsevier Health Sciences, 2006; pp. 459–61.
12. Regezi JA, Sciubba JJ, Jordan RC.Oral pathology: Clinical pathologic considerations. 4th ed. Philadelphia: WB Saunders, 2003; pp. 115–6.
13. Ainamo J. The effect of habitual tooth cleaning on the occurrence of periodontal disease and dental caries.Suom Hammaslaak Toim. 1971; 67: 63.

14. Whitaker SB, Bouquot JE, Alimario AE, Whitaker TJ., Jr Identification and semi quantification of estrogen and progesterone receptors in pyogenic granuloma of pregnancy.OralSurg Oral Med Oral Pathol.1994; 78: 755–60.

15. Ojanotak-Harri AO, Harri MP, Hurttia HM, Sewon LA. Altered tissue metabolism of progesterone in pregnancy gingivitis and granuloma.JClin Periodontol.1991; 18: 262–6.

16. Yuan K, Jin YT, Lin MT. Expression of Tie-2, angiopoietin-1, angiopoietin-2, ephrinB2 and ephrinB4 in pyogenic granuloma of human gingiva implicates their roles in inflammatory angiogenesis.J Periodontal Res.2000; 35: 165–71.

17. Jafarzadeh H, Sanatkhani M, Mohtasham N. Oral pyogenic granuloma: A review.J Oral Sci.2006; 48: 167–75.

18. Mubeen K, Vijaylakshmi KR, Abhishek RP. Oral pyogenic granuloma with mandible involvement: An unusual presentation. J Dent Oral Hyg 2011; 3: 6-9.

19. Greenberg MS, Glick M Burket's oral medicine: diagnosis and treatment. 10th ed, BC Decker, Hamilton, 203; 141-142.

20. Kapadia SB, Heffner DK Pitfalls in the histopathologic diagnosis of pyogenic granuloma. Eur Arch Otorhinolaryngol 1992; 249: 195-200.

21. Sato H, Takeda Y, Satoh M. Expression of the endothelial receptor tyrosine kinase Tie2 in lobular capillary hemangioma of the oral mucosa: An immunohistochemical study. J Oral Pathol Med. 2002; 31: 432–8.

22. Hirshberg A, Buchner A (1995) Metastatic tumors to the oral region. An overview. Eur J Cancer B Oral Oncol 1995; 31: 355-360.

23. Raut A, Huryn J, Pollack A, Zlotolow I Unusual gingival presentation of post-transplantation lymphoproliferative disorder: a case report and review of the literature. Oral Surg Oral Med Oral Pathol Oral RadiolEndod20000; 90: 436-441.

24. White JM, Chaudhry SI, Kudler JJ, Sekandari N, Schoelch ML, Silverman S Jr Nd:YAG and CO2 laser therapy of oral mucosal lesions. J Clin Laser Med Surg1998; 16: 299-304.

25. Meffert JJ, Cagna DR, Meffert RM Treatment of oral granulation tissue with the flashlamp pulsed dye laser. DermatolSurg 1998; 24: 845-848.

26. Ishida CE, Ramos-e-Silva M Cryosurgery in oral lesions.Int J Dermatol 1998; 37: 283-285

27. Ichimiya M, Yoshikawa Y, Hamamoto Y, Muto M (2004) Successful treatment of pyogenic granuloma with injection of absolute ethanol. J Dermatol 2004; 31: 342-344.

28. Moon SE, Hwang EJ, Cho KH Treatment of pyogenic granuloma by sodium tetradecyl sulfate sclerotherapy. Arch Dermatol 2005; 141, 644-646.