Hard Palate Pyogenic Granuloma in 11 Year Old Girl: A Case Report, and Compendious Insight

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Abstract:- Pyogenic Granuloma is a unique clinical entity originating as response of tissues to non specific infection, occurring most commonly in the gingiva, lips, tongue and buccal mucosa. It is a tumor like growth considered as an amplified, conditioned response to minor trauma by the overproduction and proliferation of vascular type of connective tissue. The clinical diagnosis and differentiation of these lesions is at times difficult. The purpose of this article is to report a case of benign tumor occurring on hard palate of an 11 year old female patient clinically diagnosed as oral pyogenic granuloma. Excisional biopsy of the lesion revealed findings suggestive of PG. This article intends to give a comprehensive review of oral PG with its etiology, etiopathogenesis, clinical features, multiple differential diagnosis and treatment.

Keywords:- Oral, Pyogenic Granuloma(PG), Non specific infection, Hard plate, Benign tumor, Etiological factors, Etiopathogenesis, Treatment.

I. INTRODUCTION

Pyogenic granuloma (PG) phrase was introduced by Hartzell in 1904,[1] PG is a proliferative lesion of nonneoplastic nature, common in the oral cavity, usually located in the gingiva.[2]

Oral PG in children represents one of the most frequent benign oral tumors because of their inflammatory origin. The inflammatory hyperplasia common in mouth affects the oral epithelium, the connective and glandular tissue.[3] It is considered a reactive tumor as its appearance is generated through various physical stimuli (trauma, tartar, and low intensity chronic irritation), chemicals (drugs, hormones) or dental and periodontal treatment.[4] There are cases where even dental eruption can be blamed for Pyogenic Granuloma appearance.[5]

Clinically presents as erythematous nodule, which develops gradually as a pedunculated lump with erosive surface which tends to bleed. It presents with a smooth or lobulated aspect like a cauliflower. The shade of the tumor varies from pink to bright red according to the age of the lesion.[6] Oral PGs have intense vasculature, bleed spontaneously or after minor trauma. Most often occur in young women, because of the role that hormones have on vascular development.[7] PGs can appear at any age with a predilection for young adults and children. The oral gingiva has high affinity for PGs, but can also prevail on other parts of oral cavity, with 30% of these tumors developing after trauma and proliferating to reach upto 2cm of size within few days. (Schoen, 1994) Fore sites of mouth are affected more than hind sites, and the maxilla is more commonly affected than mandible. Gingival PGs account for approximately 75-85% of all oral PGs.[8] Although many lesions occurring in the oral cavity have similar appearance as PG, a detailed history, clinical examination, and proper treatment plan will be helpful to pinpoint the disease.

Case Report

A female patient aged 11 year old, attended by her Mother reported to the Department of Pedodontics and Preventive dentistry, A J Institute of Dental Sciences, Mangalore, with a chief complaint of presence of a swelling behind the upper left back tooth region from 2 months. The swelling was minute when first noted, but had grown within 2 weeks of time span to reach the current size. Swelling was not associated with any episodes of pain but with incidental bleeding on brushing. The lesion is presented in Figure 1.

The patient’s medical history was nothing significant, personal history revealed the patient had a habit of eating chips constantly at least 2 to 3 packets a day, which was reduced due to bleeding from the lesion with no other specific factors detected. Intracoral soft tissue examination revealed a lone, pedunculated, spherical-shaped, reddish pink nodule with distinct border and uneven surface, in the posterior half of hard palate just sidealong to the erupting left canine on the palatal aspect of maxillary first premolar measuring 1.2 cm x 2 cm in size.

The swelling on palpation was infrangible, soft in consistency, and blanched with pressure. Hard tissue examination presented generalized debris deposition with no decay. All hematomal investigations carried out were normal confirming a good immunocompetent status of the patient. Provisional diagnosis of PG or Hemangioma was formed taking into account clinical signs and symptoms. Treatment opted was surgical removal of the lesion. Verbal informed consent of the patient and a signed informed consent of the caregivers (the mother) for treatment and for the use of medical information in the datasheet was obtained.
The surgical excision followed by curettage was performed under local anesthesia. The histopathological examination of excised mass described stratified squamous parakeratinized spongiosic epithelium with presence of numerous budding capillaries, lined by plump endothelial cells accompanied by chronic inflammatory cell infiltrate in the underlying connective tissue stroma suggestive of PG.

II. DISCUSSION

Familiar foreign materials like calculus acts as a stimulant in the gingival sulcus resulting in procreation of connective tissue leading to PG.[9] One-third of the lesions occur after injury. Ainamol suggested that routine tooth brushing habit resulted in these lesions due to repeated injury to gingiva.[10] Some of the triggering factors for PG are, release of diverse endogenous substances, angiogenic factors,[11] injuries to the primary teeth,[12] ectopic tooth development, and occlusal disharmony.[13]

Obscure traumas to the tissues are the most common etiologies which provide pathways for breach of nonspecific types of microorganisms, with tissues responding to these organisms by overgrowth of vascular type of connective tissue. This restates the well-known biological principle that any irritant may act either as stimulus or as destructive agent or both when applied to living tissue. Growth of oral PG occurs when the cells within the tissues differentiate and mature.[14]

Differential diagnosis includes peripheral ossifying fibroma, hemangioma, peripheral giant cell granuloma, hyperplastic gingival inflammation, Kaposi’s sarcoma, bacillary angiomatosis.[15] Peripheral giant cell granuloma is clinically similar to PG, but bone resorption on radiograph and appearance of many multinucleated giant cells and numerous capillaries in cytology are differentiating features.[5] Fibroma can be identified by the consistency, texture, and lighter color than PGs. Peripheral ossifying fibromas also appears as overgrowth on gingiva, but are distinguished by a high degree of cellularity and display of bone formation. Plump proliferating fibroblasts intermingled throughout a very delicate fibrillar stroma in cytology differentiate fibromas from PGs.[6] Hemangiomas are true neoplasms of endothelial cells, exhibit a rapid proliferative phase and slowly involute to non existent and do not recur after involution or removal, whereas PG is more stable lesion and fails to regres and chances of recurrence are more. Multinodular, deep red or bluish red appearance of hemangiomas and chairside diagnostic procedure diascopy helps to differentiate hemangiomas from PG.[16]

The histologic features of hemangioma include numerous capillaries lined by single layer of endothelial cells, whereas that of PG include a great number of endothelium lined vascular spaces with extreme proliferation of fibroblasts and budding endothelial cells.[17]

Treatment of oral PGS in many instances is surgical excision of the lesion followed by removal of all visible sources of irritation that can evoke the lesion.[5] Other diverse treatment modalities include use of intralesional steroids, flash lamp, cryosurgery, sclerotherapy, and different types of lasers like Nd: Yttrium-aluminium-garnet, Argon, CO2.[18] Positive results exist with use of Erbium:YAG ablative laser for the treatment of isolated mucosal PGs of pediatric patients. Failure of removal of etiological factors, incomplete excisions contribute to the recurrence of PGs. Bhaskar and Jacoway have documented that after conservative excision, 15.8% recurrence rate was seen. Vilmann et al. noticed lesser recurrence rate with lesions from other oral mucosal sites when compared to gingival cases. Sapp et al. asserted relatively high recurrence rate after simple excision.[19] Higher recurrence rate makes the follow-ups essential part of treatment.

III. CONCLUSION

PG is a relatively common presentation and are to be treated soon to avoid the distress and complications they cause to the patients, like recurrent bleeding or obstruction to erupting teeth or other structures. A thorough understanding of the lesion is prudent to differentiate it from similar clinical presentations in children and to select appropriate treatment modalities.

REFERENCES

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Fig 1: Pyogenic granuloma mesiopalatal to 1st premolar

Fig 2: Palatal view of Pyogenic granuloma
Fig 3: Histopathologic view of Pyogenic Granuloma

Fig 4: Excised Pyogenic Granuloma