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PYOGENIC GRANULOMA: A CASE REPORT AND BRIEF REVIEW OF ETIO-PATHOGENESIS

Periodontology

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ABSTRACT

Pyogenic granuloma (PG) is a non- specific conditioned gingival enlargement, that occurs as a reaction to a localized irritation or injury, without a specific underlying cause and appears as sessile or pedunculated growth which vary from deep red to reddish purple or pale pink in colour ranging from few millimeters to several centimeters, with more predilection to maxillary gingiva. After conservative excision, many lesions recur with the rate of 15.8% and gingival sites has more recurrence than other oral sites. In our case, we had observed recurrence of the PG after the initial excision, which was then treated with minimally invasive approach of Scaling, root planing and focusing on the curettage more, after which the lesion was completely subsided which was observed after total duration of 4 weeks after the minimally invasive approach with healthy sulcus depth, absence of signs of inflammation and establishment of crestal bone radiographically without use of any biological mediators.

KEYWORDS

Pyogenic granuloma, Recurrence, Minimal Invasive Approach, Regeneration.

INTRODUCTION:

Pyogenic granuloma is an inflammatory hyperplasia that occurs due to various stimuli such as chronic low-grade irritating factors[1], traumatic injury[2], hormonal factors[3] or certain kind of drugs[4]. It is the most common gingival tumors of the oral tissues accounting for 75%[4] where predominant cause is calculus or foreign material in gingival crevice[2]. Hartzell coined the term "Pyogenic Granuloma" or Granuloma Pyogenicum[5]but the term is considered as misnomer, as it is not related to any infection that contain pus[6]. It is also known with various terminologies like Crocker and Hartzell's disease[5] Hemangiomatous granuloma[7], Granuloma telangiectacticum[8], Granuloma Pediculatum Benignum, Benign vascular tumor and as Granuloma gravidarum during pregnancy[6].

Etio-Pathogenesis:

Several authors have described the pathogenesis behind different etiological factors of pyogenic granuloma.

Etiological Factors:

- Local irritants like calculus, poor oral hygiene, nonspecific infection etc[2].
- Physical trauma like over hanging restorations, cheek biting, foreign material in the gingiva, micro trauma from habitual tooth brushing[2,9].
- Hormonal factors during pregnancy or use of Oral contraceeptives[3].
- Drugs like isotretinoin[10].

Pathogenesis:

The exact pathogenesis remains unclear for the development of the lesion, but several authors had given various concepts for etio-pathogenesis.

Though pyogenic granuloma has multiple etiological factors, it is now generally agreed that some minor trauma to the tissues is primary factor for the development of lesion. Minor trauma to the tissues provide a pathway for the invasion of non-specific type of micro-organisms and when infection occurs, tissue responds to it either as a stimulus or destructive or both.

As per the normal physiological response, when infected tissue has many cells in small volume and there is a relative reduction of blood flow through the area of the inflammation, the concentration of stimulus will be high, and tissue responds to it by growth rather than destruction. In Pyogenic granuloma, because of persistent irritation for a long period of time, inflammatory response of tissue increases [11] and as a response to the stimulus, tissue responds by the overzealous proliferation of vascular endothelium.[12] Mast cells and macrophages exerts their effect on vascular endothelial cells and fibroblasts leading to the synthesis extracellular matrix and this is controlled by various cytokines, out of which growth factors like bFGF- a heparin binding angiogenic protein, has been found to be highly mitogenic for capillary endothelial cells to induce angiogenesis and immune-localization of these growth factors released from the macrophages and mast cells was noticed in extracellular matrix during neovascularization of the granulation tissue.

Mast cell mediator, namely tryptase, promotes fibroblast activation, collagen deposition, and fibrosis. Fibroblasts also promote neovascularization through pro-angiogenic cytokines primarily Vascular Endothelial Growth Factor (VEGF) [13,14] . Mutual mast cell–fibroblast interactions promotes granulation tissue formation is a hallmark in the development of the pyogenic granuloma[15]. Regezi et al., demonstrated a strong activity of angiogenesis in oral pyogenic granulomas by demonstrating prominent capillary growth in the hyperplastic granulation tissue[2]. Jafarzadeh et al., stated the association of decorin, VEGF, connective tissue growth factors, bFGF basic fibroblast growth factor on angiogenesis in profound inflammation[6]. Yuan et al., stated that Other factors like Vascular morphogenesis factors Tie-2, angiopoietin 2, ephrin B2 and ephrin were found to be up-regulated in oral pyogenic granulomas compared to healthy gingiva and emphasized the role of two angiogenesis enhancers VEGF and bFGF, and two angiogenesis inhibitors, TSP-1 and angiostatin in mechanism for angiogenesis and stated that the imbalance between angiogenesis enhancers and angiogenesis inhibitors was also found to be an etiology for pyogenic granuloma[16].

Histologically, there are two types of pyogenic granuloma (LCH-Lobular capillary Haemangioma) and (Non-LCH- Non Lobular capillary Haemangioma). The LCH and the non-LCH pyogenic granulomas have different pathways of evolution[17].

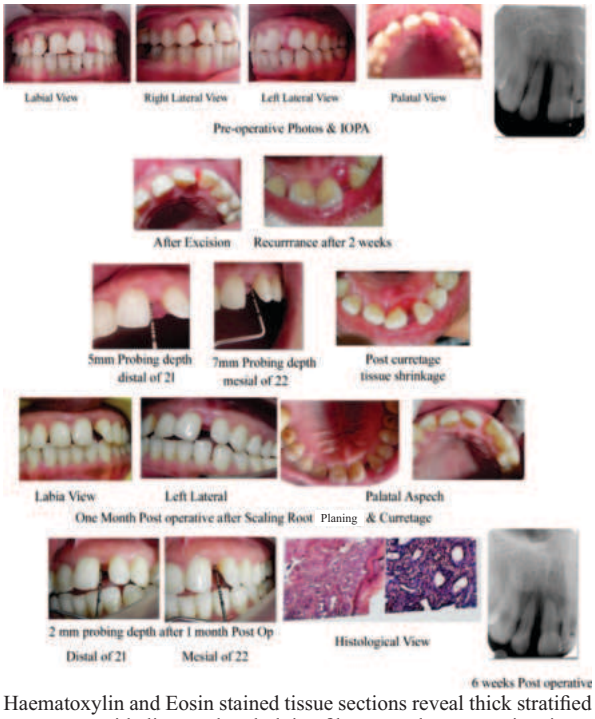
Most oral pyogenic granulomas are LCH type histologically. On investigating the relationship of human endothelial receptor tyrosine kinase Tie2 and its expression in the lobular capillary haemorrhage, it was observed that the expression of Tie2 in the ovoid cells with the presence of alpha SMA antibodies in the development and progression of the LCH type of pyogenic granuloma[18]. The non-LCH type consisted of a vascular core resembling granulation tissue with foci of fibrous tissue[19].

Case History:

A 48 years old female patient reported to the department of periodontology with the chief complaint of tissue growth and pain in upper front teeth region, which was associated with difficulty in mastication and increase in the space between the upper front teeth.

On intra oral examination, a 1.5×2cm solitary sessile nodular mass which is bluish red, lobulated, fleshy and not associated with any surface alterations and no spontaneous bleeding was observed in the interdental region of maxillary left central and lateral incisor extending from buccal to palatal aspect with grade I mobility of maxillary left lateral incisor and pathological migration of maxillary left central and lateral incisor was observed. Clinical probing depth was not measured because of obstruction of tissue mass. The patient has poor oral hygiene with abundant local factors. On radio-graphical examination, horizontal bone loss was observed which extended till apical third. A routine hemogram was found to be normal. Patient's had given history of hypertension since 4 years and was under medication for the same. The patient's blood pressure was 150//80 mm Hg. Initially, surgical excision with a scalpel under plain lignocaine was performed followed by sub-gingival scaling and root planing. Postoperative instructions given and advised amoxicillin 500mg BID for 5 days with analgesics BID for 3 days and patient was recalled after two weeks for review. Patient was motivated and instructed to maintain the oral hygiene. On 2 weeks review, there was recurrence of a small vascular rich papule of 5 mm in the interdental palatal aspect of maxillary left central and lateral incisor with a probing depth of 5mm on distal and mesial aspect of maxillary left central and left lateral incisor. During the second visit, a minimally invasive approach was planned in which only thorough sub-gingival scaling, root planing and curettage was performed under plain lignocaine and the end point of shrinkage of the tissue mass was noted after the treatment. Post-operative instructions given and patient was kept under analgesics and topical application of Chlorhexidine gel. After 4 weeks of SRP and Curettage, a satisfactory healing without any recurrence of growth was observed, healthy gingival tissues restored without any signs of inflammation and with a sulcus depth of 2mm was observed and patient oral hygiene compliance is excellent. Surprisingly, on radiographic evaluation done after 1 month of SRP and curettage, there was regeneration and restoration of crestal bone till the junction of middle and apical third without use of any biological mediators and patient was satisfied with the outcome, as treatment approach was minimal.

Histopathological report



Haematoxylin and Eosin stained tissue sections reveal thick stratified squamous epithelium and underlying fibro vascular connective tissue with diffuse dense chronic inflammatory cell infiltrate. Connective tissue has loosely arranged fibers with numerous endothelial lined blood vessels of varying sizes and shapes.

DISCUSSION:

Pyogenic granuloma is an inflammatory hyperplasia of oral tissues appears as elevated smooth or exophytic, sessile or pedunculated growth, non-lobulated or lobulated and warty with or without ulcerations and sometimes covered by yellow fibrin[20]. It varies from deep red to reddish purple or pale pink depending upon the vascularity and maturation of the lesion. Size varies from few millimeters to several centimeters. Usually grow slowly and painless, sometimes shows rapid growth. Lesions are more common on maxillary gingiva than mandibular, anteriors most common than posteriors, also facial aspect is most common than lingual though some extend between the teeth extending from facial to palatal/lingual aspect[21]. Majority of lesions are most common on marginal gingiva with only 15% on alveolar part[22]. Other sites include lips, tongue or buccal mucosa, palate affecting the maxilla more than the mandible. The pregnancy tumour variant of pyogenic granuloma occurs in up to 5% of pregnancies[8].

In the gingiva, labial gingiva in the anterior region has more predilection and this attributed to habitual tooth brushing causing micro trauma and irritation to the gingiva[9]. After conservative excision, it was observed that the recurrence rate is 15.8%[23] and gingival sites has more recurrence than other oral sites. If the patient is pregnant, recurrence is common.

Sternberg et al. described the three phases of the lesion as the natural course involving cellular phase, vascular phase and involution phase. In cellular phase or early phase, there will be a compact cellular stroma with little lumen formation. The vascular/ capillary phase consists of lobules, which are highly vascular with rich intraluminal red blood cells. The “involutionary phase” is suggestive of healing phase, consisting of intra- and peri-lobular fibrosis. On correlation of various stages with clinical appearance, younger lesions appear as red to purple and soft, older lesions appear as pink and firm because of more collagenisation. Because of this, sometimes-older lesions appear as oral fibroma, peripheral ossifying fibroma. The only way to differentiate the older lesions is relative increase in vascular component and inflammatory infiltrate as they are indicative of pyogenic granuloma though the lesion is older[24].

Different treatment approaches are simple conservative surgical excision with curettage in the presence of sub-gingival irritation factors. Using Argon laser photocoagulation is one of the effective therapies[25] and 585 nm flashlamp-pumped pulsed-dye laser surgery is beneficial. The disadvantage is it requires multiple treatments and can be used for small lesions[26].

In the presence case report, the local irritation factor for the development of the lesion was the presence of sub-gingival calculus and the lesion developed interdentally rather than the usual appearance of labial gingiva. The lesion was soft, reddish-purple with a lobular appearance with minor aberration in the surface texture without any surface alterations and the lesion was painful because of occlusal interference. The lesion recurred after initial excision, this could be attributed to, failure to remove etiologic factors completely in the first visit[2] as treatment was planned for a shorter duration because of the stage 1 hypertension condition of the patient. On recurrence of the growth during the second visit, a conservative approach of sub-gingival scaling, and curettage with topical appliance of chlorhexidine gel for 1 week was done similar to the minimally invasive approach suggested by Chandrashekar B for the treatment of PG[27]. After 4 weeks follow up of SRP and curettage, there was no any recurrence and the area is healthy without any signs of inflammation and radiographically there is restoration of lost crestal bone without use of any biological mediators or bone grafts. This could be attributed to body's healing potential in the absence of irritational factors according to the study done by Kantro M et al where restoration of crestal bone noticed after the removal of co-destruction factors[28].

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