REVIEW ARTICLE

Peripheral Ossifying Fibroma: A Review

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ABSTRACT

The gingiva is often the site of localized growths that are considered to be reactive rather than neoplastic in nature. Chronic irritation from various sources like masticatory forces, entrapment of debris, ill-fitting dental appliances, dental calculus etc. is considered to be responsible for it. Peripheral ossifying fibroma is such a gingival lesion with a high recurrence rate. Its origin is still controversial, though periodontal ligament is considered to be the tissue of origin.

KEYWORDS: Peripheral Ossifying Fibroma, Reactive, Origin, Periodontal Ligament

INTRODUCTION

There are two types of ossifying fibromas, central and peripheral. The central type arises from the endosteumor periodontal ligament adjacent to the root apex and expands from the medullary cavity of the bone, whereas peripheral variant occurs exclusively on the soft tissue covering the alveolar process.[1] Peripheral variant originates in reaction to local irritation which may be in the form of a traumatic event, plaque, calculus, bacterial, restorations and various prosthetic and orthodontic appliances.[2] Ossifying fibromas of the oral cavity have been reported in the scientific literature since middle of the twentieth century. Many names have been given to similar lesions, such as peripheral ossifying fibroma(Eversole LR 2006), epulis or peripheral fibroma with calcification **(Bhaskar and Jacoway2008**), peripheral cemento-ossifying fibroma**(Feller L et al., 2008)**, calcifying fibroblastic granuloma**(Lee KW 2008),** Peripheral cementifying fibroma, peripheral fibroma with cementogenesis**(Kumar SK et al., 2009).** The sheer number of names used for fibroblastic gingival lesions indicates that there is much controversy regarding the classification of these lesions. As it commonly occurs in interdental papilla area so it is thought to originate from periodontal ligament.

CLINICAL FEATURES

A POF may occur at any age but exhibits a peak incidence between the second and third decades. Almost 60%of the lesions occur in the maxilla and mostly occurs anterior to the molars in the second decade of life. The lesion affects females more commonly than males (5:1 respectively) **(Buchner & Hansen, 1987)**.Clinically, POF is sessile or pedunculated, usually ulcerated and erythematous or exhibits a colour similar to that of surrounding gingiva. It does not blanch on palpation **(Walters *et al*.; Kenney *et al*., 1989)**. The lesions of POF are usually less than 1.5-2 cm in diameter, but have been known to grow to larger sizes **(Poon *et al*., 1995).** POF can cause resorption of the alveolar crest and separation of adjacent teeth with pathologic migration **(Poon *et al*.)**.[3]

RADIOGRAPHIC FEATURES

Radiographically the features of POF tend to vary. Foci of calcifications have been reported to be scattered in the central area of the lesion, but not in all lesions. Underlying bone involvement is usually not visible on a radiograph but in rare instances,

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superficial erosion of bone can be seen.[4]

HISTOLOGIC FEATURES

Gardner (1982) stated that cellular connective tissue of POF is so characteristic that a histological diagnosis can be made with confidence, regardless of the presence or absence of calcification. The peripheral variant is characterized by the presence of collagenous connective tissue, proliferation of endothelial cells and formation of a mineralized product. Peripheral ossifying fibroma can mimic pyogenic granuloma because sometimes endothelial proliferation is dense in areas of ulceration. The mineralized component of peripheral ossifying fibroma varies from 23% to 75 % (Farquhar et al).[5]

ORIGIN

The widely accepted etiopathogenesis for POF is the inflammatory hyperplasia of the cells of the periosteum or periodontal ligament.,[6,7,8] as there is excessive proliferation of mature fibrous connective tissue in response to gingival injury, gingival irritation, sub gingival calculus or a foreign body in the gingival sulcus. Chronic irritation of the periosteal and periodontal membrane causes metaplasia of the connective tissue and result ant-initiation of formation of bone or dystrophiccalcification.[4,6] An origin from cells of periodontal ligament has been suggested because of exclusive occurrence of POF from interdental papilla, the proximity of gingiva to PDL, the presence of oxytalan fibres within the mineralized matrix of some lesions, the age distribution which is inversely related to the number of lost permanent teeth, and the fibro-cellular response similar to other reactive gingival lesions of periodontal ligament origin.[1,9]According to **Marcos A. Jose *et al*., 2010** the proliferating cells of connective tissue in POF are of myo-fibroblastic nature ((i.e., cells sharing morphological characteristics with fibroblasts and muscle cells). An immuno-histochemical study made to determine the nature of these proliferating spindle shaped cells showed the cells to be positive to vimentin and actin suggesting the myo-fibroblastic nature.

TREATMENT

Treatment of POF consists of elimination of etiological factors, scaling of adjacent teeth and total aggressive surgical excision along with involved periodontal ligament and periosteum to minimize the possibility ofrecurrence.[10] Long term postoperative follow-up is extremely important because of the high growth potential of incompletely removed lesion and a relatively high recurrence rate of approximately 20%.[1]

CONCLUSION

POF is a progressing lesion with high recurrence rate. It should be differentiated clinically, radiographically and histologically from various other gingival lesions, so that appropriate treatment can be carried out at the earliest. Thorough surgical removal should be done along with removal of all the irritants and etiological factors so as to prevent the recurrence. Though its most accepted origin is from periodontal ligament but still further research is needed regarding its origin by using various advanced techniques like immunohistochemistry. High rate of occurrence among females may indicate some hormonal role in its etiopathogenesis but yet to be proved.

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