

Original Research Paper

Dental Science

A RARE CASE OF RECURRENT PERIPHERAL OSSIFYING FIBROMA: A CASE REPORT

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In the oral cavity, gingival growth is one of the most commonly encountered lesions, most of these lesions are benign, but some do have malignant potential. Peripheral ossifying fibroma (POF) is a benign neoplasm that usually develops from reactive gingival overgrowth. Microscopically, the tumour shows stratified squamous epithelium and highly cellular fibrous stroma, sparse endothelial proliferation with fibroblasts and dystrophic calcifications. It has to be differentiated histopathologically from pyogenic granuloma, fibroma, peripheral giant cell granuloma, peripheral odontogenic fibroma and fibrous hyperplasia. This report presents a case of gingival overgrowth in a 56-year-old woman which reoccurred for 4th time. Treatment included excision of the gingival growth and also extraction of the tooth in relation to the growth as it presented with cervical caries and root resorption. Based on histopathological examination a definitive diagnosis of POF was established.

KEYWORDS: Peripheral ossifying fibroma, oral cavity, gingival overgrowth, Reactive lesion.

INTRODUCTION

Increase in size is a common feature of gingival disease and the accepted terminology for this condition is gingival enlargement or gingival overgrowth. Benign neoplasm of the periodontal tissues are characterised by progressive growth without remarkable symptoms. The growth is measured in terms of months or years and they are often found incidentally on routine examination. They may be diffuse or localised. Gingiva is a common site for neoplastic and non neoplastic lesions. Peripheral Ossifying Fibroma (POF) is a nonneoplastic gingival growth which is more commonly seen in relation to the gingiva. Peripheral ossifying fibroma is a reactive lesion and originates from the periodontal ligament. The site for its occurrence is mostly on the gingiva, anterior to the molars, interdental papillary region and in maxilla.² From the Indian perspective, it is usually noticed in 5th-6th decades of life with female predilection.³ They are slow growing, spherical in shape, pink in colour, surface may be ulcerated and base may be sessile or pedunculated. Usually these lesions occur as a result of irritants, plaque, calculus, trauma, microorganisms, restorations and dental appliances. The confirmatory diagnosis is based on the histopathological examination.⁴ A conservative surgical management provides an excellent prognosis, though the recurrence rate can reach 20-22%.

CASE REPORT

A 56 year old female patient reported to the Department of Periodontics with a chief complaint of growth in the anterior front tooth region of the lower jaw. The growth was painless and initially smaller in size, which increased to the present size over a period of 8 years. The patient gave history of similar growth three times in the past which was excised and had now reoccurred for the fourth time (Figure 1). Initially the growth was smaller in size and occurred for the first at the age of 15 years and was excised 8 years hence. The growth then reoccurred and was subsequently excised twice. The first three times the growth was a single isolated growth however the fourth time when it reoccurred was 2 in number. Patient also gave the history of bleeding while brushing.



Figure 1: Pre-operative view

Patients medical history revealed that she was diabetic since 10 years and was on medication for the same and had also undergone hysterectomy 1 year back. Patient past dental history did not reveal any significant except for repeated excision of similar intraoral growth in the same region.

On extraoral examination no gross asymmetry of the face was detected, the lips were competent and lymph nodes were not palpable. Intra examination revealed two solitary pedunculated, reddish pink gingival growth measuring about 1cm×1cm in dimension in 42 to 44 region. On palpation both the growths were firm in consistency and non-tender.

On radiographic examination radiolucency was seen in the cervical area of 43 suggestive of cervical caries. And bone loss was seen on the mesial aspect of 43 (Figure 2)



Figure 2: Radiographic view

The provisional diagnosis of peripheral ossifying fibroma was given. The differential diagnosis consisted of pyogenic granuloma, peripheral giant cell granuloma, peripheral odontogenic fibroma and irritational fibroma.

TREATMENT

Excision of the lesion was planned. Initial treatment included scaling and root planning so as to eliminate the local etiological factors such as plaque and calculus. Patient was recalled after a weak for the excision of the lesion. The lesion was excised completely using scalpel, following which a periodontal flap was reflected in order to curette the bone. On reflection of the flap cervical caries lesion and external root resorption exposing the root canal was noted. The cavity was filled with granulation tissue suggesting granuloma formation in relation to lateral aspect of 43. Hence extraction of 43 was planned. 43 was extracted and the socket was curetted (Figure 3). The excised growth was submitted for histopathological examination.



Figure 3: Excision of the growth and extraction of 43

The patient presented for follow up examination 14 days postoperatively. On postoperative evaluation, the surgical site appeared to be healing well. Patient was followed up for 1 year and showed no evidence of recurrence. (Figure 4)



Figure 4: 1 year Follow up

HISTOPATHOLOGY

The microscopic examination revealed nonkeratinised stratified squamous epithelium and underlying connective tissue stroma (Figure 5). The epithelium is hyperplastic and appeared ulcerated in few areas. The connective tissue was highly cellular and composed of collagen fibers and associated fibroblasts. Few areas in the subepithelial connective tissue showed round to ovoid calcified bodies while sheets of plasma cells were seen in other areas. Diffuse inflammatory cell infiltrate composed of neutrophils, eosinophils, lymphocytes and plasma cells as well as discreate areas of fibrosis were seen in the connective tissue stroma. Hence a histopathological diagnosis of peripheral ossifying fibroma was given.

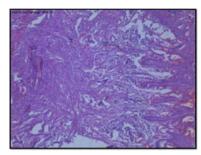


Figure 5: Histopathological view

DISCUSSION

Peripheral ossifying fibroma is thought to be either reactive or neoplastic in nature. In 1872 Menzel first described the lesion ossifying fibroma, but its terminology was given by Montgomery in 1927.5 There are two types of ossifying fibroma, the central and the peripheral. POF is not a counterpart of the central ossifying fibroma but a reactive lesion of the gingiva. The term peripheral ossifying fibroma was given in the year 1982 by Gardner for a lesion that is reactive in nature and is not the extraosseous counterpart of a central ossifying fibroma (COF) of the maxilla and mandible. Considerable confusion has prevailed in the nomenclature of peripheral ossifying fibroma with various synonyms being used, such as peripheral cementifying fibroma, ossifying fibroepithelial polyp, peripheral fibroma with osteogenesis, peripheral fibroma with cementogenesis, peripheral fibroma with calcification, calcifying or ossifying fibrous epulis and calcifying fibroblastic granuloma.1

Majority of the reports suggest POF is commonly seen in the second decade of life, with a reduced incidence with age. In the present case, incidence of peripheral ossifying fibroma was in the fifth decade, which was comparatively older than that reported by Ababneh.⁷ The size of the POF ranges 0.4-4.0cm. At its greatest dimension, the average lesion measures approximately 1.0cm.8 In the present case, the dimension of the lesions were well within above mentioned ranges. POF can become large, causing extensive destruction of adjacent bone and significant functional or esthetic alterations. Though the etiopathogenesis of POF is uncertain, an origin from cells of the PDL has been suggested. The reasons for considering PDL origin for POF include exclusive occurrence of POF in the interdental papilla gingiva, the proximity of gingiva to the PDL, and the presence of oxytalan fibres within the mineralized matrix of some lesions. Excessive proliferation of mature fibrous connective tissue is a response to gingival injury, gingival irritation, subgingival calculus or a foreign body in the gingival sulcus. Chronic irritation of the periosteal and periodontal membrane causes metaplasia of the connective tissue and resultant irritation of formation of bone or dystrophic calcification. It has been suggested that the lesion may be caused by fibrosis of the granulation tissue.8 High female predilection, rare occurrence in the first decade, and decline in incidence after age 30 suggest that hormonal influence may be a lesional growth factor.9 In the present case, the patient had abundant supragingival and subgingival calculus which probably contributed to etiopathogenesis of the lesion. After excision of the growth it was noted that there was abundant granulation tissue from the cervical caries of 43 which might have caused chronic irritation leading to the formation of such a reactive gingival lesion.

Radiographic features of the POF vary. Radiopaque foci of calcifications have been reported to be scattered in the central area of lesion, but not all lesions demonstrate radiographic calcifications. Underlying bone involvement is usually not visible on a radiograph. In rare instances, superficial erosion of bone is noted. 10 In the present case, there was mild bone loss on the mesial aspect of 43 which indicating superficial erosion of the bone. If POF is suspected a histopathologic diagnostic approach should always be adopted. It rests on several criteria including intact or ulcerated stratified squamous surface epithelium, benign fibrous connective tissue with varying numbers of fibroblasts, sparse to profuse endothelial proliferation, mineralized material consisting of mature, lamellar or woven osteoid, cementum like material or dystrophic calcifications and acute or chronic inflammatory cells in lesion. All the above features were present in the present case. Follow-up is essential because of the recurrence rates vary from 8 to 20%, recurrence are primarily due to incomplete excision, and or persistence of local factors. The present case was unusual because the lesion had reoccurred for the 4th time. It was completely excised along with extraction of the tooth and the socket was curetted in order to prevent recurrence. The patient was followed for 1 year period without recurrence.

CONCLUSION

POF is a slowly progression lesion, the growth of which is generally

limited. Many cases will progress for long periods of time before patient seeks treatment. Treatment consists of surgical excision which should include the periosteum, and scaling of adjacent teeth. Close postoperative follow-up is required because of the recurrence potential of incompletely removed lesions.

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