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Maxillary peripheral ossifying fibroma: A case report and review of literature

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Abstract

Fibro-osseous lesions of the maxillofacial bones comprise a diverse group of pathologic conditions that include developmental lesions, reactive or dysplastic diseases, and neoplasms. The concept of fibro-osseous lesions has evolved over the last several decades and now includes two major entities: fibrous dysplasia and ossifying fibroma. The less common lesions include florid osseous dysplasia, periapical dysplasia, focal sclerosing osteomyelitis, proliferative periostitis of Garre, and osteitis deformans. We report a case on Peripheral ossifying fibroma in Maxilla.

Keywords: Craniofacial, Fibrous, Maxilla, Ossifying, Peripheral

Introduction

Peripheral ossifying fibroma (POF) is a non-neoplastic entity, which occurs on the gingiva in response to trauma or irritation. It is a reactive lesion of connective tissue and is not the soft-tissue counterpart of central ossifying fibroma. It is reported under bewildering array of terms in literature, which includes peripheral cementifying fibroma, calcifying or ossifying fibroid epulis, mineralizing ossifying pyogenic granuloma, peripheral fibroma with calcifications and calcifying fibroblastic granuloma^[1]. POF was first reported by the Shepherd in 1844 as alveolar exostosis^[1]. Eversol and Robin in 1972, later coined the term peripheral ossifying fibroma^[1]. It occurs in the younger age group with a female preponderance. It has a predilection for maxillary arch and most of them occur in the incisor-cuspid region. It presents as a painless mass on gingiva or alveolar mucosa measuring not exceeding 3 cm. It can be pedunculated or sessile. Earlier lesions appear irregular and red and older lesions have a smooth pink surface. Surface ulceration may be present^[2]. POF occurring in an older age group in male patient involving posterior maxilla is rare and an unusual clinical presentation. We attempt to report one such rare case with emphasis on compilation of interesting facts pertaining to POF.

Case Report

A 30-year-old man had developed a swelling on the left side of the face that had persisted for one year [Figure 1]. There was no history of pain, trauma, epistaxis, loosening of teeth, trismus, or diminished vision. Examination of the region revealed a smooth bone hard swelling involving the right maxilla, zygoma, temporal. The skin over the swelling was normal. The swelling was bulging into the gingivo-buccal sulcus area [Figure 2]. The teeth grade 2 mobility in relation to 24,25. There was no other swelling in the body and cafe-au-lait spots were not seen. The throat and nose examination was found to be normal. The blood and urine investigations were normal. On CBCT examination [Figure 3] A prominent mixed density dome shaped labially and palatally expanding area was noted starting from alveolar crest leading to complete obliteration of floor of left maxillary sinus and completely involved left maxillary sinus cavity. The lesion starting anteriorly from distal to 22 and posteriorly mesial to 28 region. (Cross section no 7-31) (Axial section no 3-21) (sagittal section no 9-46). Lesion measuring 40.3mm in anteroposteriorly direction and 16.1 mm in buccolingual direction and 38.3 mm superoinferiorly. Internal trabecular pattern is altered having numerous hyper dense foci which is varying in size and density ranging from 200-400 (grayscale value) giving a "Drivensnow appearance" and Osseous remodelling. Large interdental space is observed in the region between 25 and 26.

The 24 and 25 are displaced more mesially secondary to osseous changes. The 26 is displaced more distally. An incisional biopsy was subsequently performed, providing a diagnosis of fibrous dysplasia. Based on the clinical history, radiographic, and histologic features of the lesion, a diagnosis of craniofacial peripheral ossifying fibroma.



Fig 1: Pre-operative extra oral frontal view



Fig 2: pre-operative intra oral appearance buccal vestibule obliteration



Fig 3: Radiographic appearance-cone beam computer tomography



Fig 4: intra operative exposure of the lesion

The case was taken under general anaesthesia nasal intubation, maintaining standard aseptic protocols, Incision was placed over labial frenum extended distal till mesial aspect of 27 to raise full thickness trapezoidal mucoperiosteal flap to exposed the

pathologic lesion [Figure 4], Enucleation and curettage of the lesion done over right side of maxilla, followed by extraction of 24,25 done [Figure 5] with thermal cauterisation was done followed by smoothing of bone margins of cavity. Ribbon gauge soaked in 5% betadine with lignocaine gel 2% and placed in the pathologic cavity one end is exposed intraorally [Figure 6] and closure were done with 3.0 vicryl suture. On second post-operative day ribbon gauge is removed and suture closer done with oral hygiene maintained Fig. 7 Post-operative inhalational agents, antibiotics IV, NSAIDS IV is maintained, follow up was done after 7 days where wound healing is satisfactory, no swelling, paresthesia, nasal discharge, haematoma, nasal bleeding, sinus discharge, with suture removal done. Post operatively OPG was taken which shows certain amount of bone formation. Fig 8

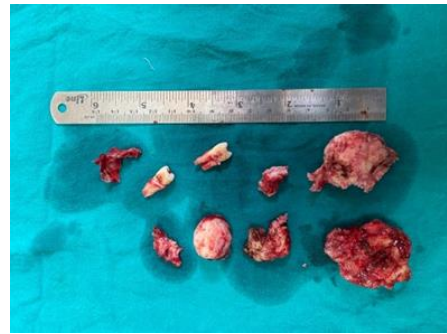


Fig 5: specimen and extracted tooth



Fig 6: Packing placed with closure



Fig 7: Suture paced and pack removed



Fig 8: Post Operative OPG

Discussion

Gingiva is one of those anatomical regions in the oral cavity with the broadest array of lesions occurring ranging from inflammatory to neoplastic. POF is one such reactive lesion, which occurs exclusively on gingiva. It accounts for 9.6% of gingival lesions^[3]. Histogenesis remains controversial and there are two schools of thought proposed to understand the histogenesis of POF. POF may initially develop as pyogenic granuloma that undergoes subsequent fibrous maturation and calcification. It represents the progressive stage of the same spectrum of pathosis^[4]. POF is due to inflammatory hyperplasia of cells of periodontal ligament/periosteum. Metaplasia of the connective tissue leads to dystrophic calcification and bone formation^[5]. Triggering factors such as subgingival plaque and calculus, dental appliances, poor quality of dental restorations, micro-organisms and food lodgement initiate the inflammatory response^[6]. Clinically POF presents as smooth lobulated pink mass on a pedunculated or sessile base. It has an increasing incidence in II decade and declining incidence after III decade.^[7] Only 0.5% cases are reported in older age group^[1]. There is a female predilection for the lesion due to the hormonal influences^[8]. POF is usually seen anterior to molars, especially in incisor-canine region^[9]. Kfir *et al.* reported that the size of the POF is usually smaller than 1.5 cm in diameter. A case of giant POF of 9 cm is reported in the literature^[10]. Multicentric POF can also occur in oral and maxillofacial region^[7] and is observed in genetic associated conditions like: Nevroid basal cell carcinoma syndrome Multiple endocrine neoplasia-type II Neurofibromatosis Gardner syndrome^[7]. POF has to be differentiated from other reactive lesions of a gingiva such as pyogenic granuloma, peripheral giant cell granuloma (PGCG) and peripheral odontogenic fibroma. Pyogenic granuloma shows red mass with surface ulceration clinically and microscopically exhibit vascular proliferation resembling granulation tissue. PGCG shows scattered giant cells in a fibrous stroma. Peripheral odontogenic fibroma contains prominent islands of odontogenic epithelium^[5]. Bone involvement, though not significant in most of the cases, some alterations are noted like: Superficial erosion of bone Foci of calcifications Widening of the periodontal ligament space and thickened lamina dura^[5] Migration of teeth with Interdental bone loss^[2]. The basic microscopic pattern of the POF is fibrous proliferation associated with the formation of mineralized components. Mineralized component varies from 23 to 75%^[7]. Butcher and Hansen reported three types of components in POF^[9]. Dystrophic calcifications Bone (woven/lamellar) Cementum.

Conclusion

POF is a pathological entity whose histogenesis is yet to be delineated. It shares a varied clinic-pathological presentation. Substantial overlap exists between various focal reactive overgrowths of gingiva. Clinic-pathological characteristics may vary and on the contrary to the usual presentation, our case presented a different age, sex and site of POF. Surgical excision is considered curative and may present a high recurrence rate compared with other reactive lesions. Identification of any reactive lesions requires the formulation of differential diagnosis to enable accurate patient evaluation and management.

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