# Juvenile Peripheral Ossifying Fibroma – A Rare Case Report & Review.

Abstract: Peripheral ossifying fibroma, first reported by Shepherd in 1844 as alveolar exostosis, is a non-neoplastic reactive lesion arising as a focal exophytic mass exclusively on the gingiva originating from the interdental area and shows no bone involvement in most cases. The lesion shows propensity for maxilla and incisor-cuspid region with female predilection. The etiological factors include local factors causing gingival irritation like calculus, plaque, illfitting dentures or orthodontic appliances; and hormonal influence, initiate exhuberant connective tissue response. The lesion shows high recurrence potential , necessitating proper identification, treatment and effective long-term recall protocol. This case report presents a relatively rare case of juvenile peripheral ossifying fibroma in relation to mandibular central & lateral incisors in an adolescent female child followed for upto 1 year after surgical excision.

Key-words: Gingiva, exophytic mass, ossifying fibroma, peripheral ossifying fibroma, reactive lesions

### Introduction:

Peripheral ossifying fibroma (POF) is a non-neoplastic, slow growing solitary mass, which is usually sessile with a smooth or ulcerated surface, typically seen on the interdental papilla and accounts for about 9% of all growths of the gingiva.[1,2] POF is usually sessile with a smooth or ulcerated surface, and thought to originate from periodontal ligament. It does not affect the adjacent teeth, but sometimes, may cause migration and mobility, and lead to delayed eruption of permanent teeth.[3]

Ossifying fibroma is classified as central and peripheral; the central type arises from the endosteum or PDL near root apex and extends from the medullary cavity of the bone and the peripheral type is in contiguous relationship with the PDL, involving soft tissues overlying the alveolar process.[4]

The purpose of this article is to present a rare case of juvenile POF in a 14 year old female patient in the mandibular incisor region and reviewing the available literature.

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https://doi.org/10.21276/ujds.2020.6.3.19	

### **Case report :**

A female aged 14 yrs, reported to the Department of Periodontology & Oral Implantology, RUHS college of Dental Sciences, Jaipur with the chief complaint of a swelling of the gums in the lower left front teeth region since 3-4 months. Her medical and family history was in significant & belonged to low socioeconomic class. The history revealed that the lesion first appeared as a small, painless gingival swelling 2 yrs back. The growth expanded in size gradually & caused spacing between lower anterior teeth (31, 32).She had got excision of the growth 1 yr back, but the growth recurred after 6-7 months & developed into its present size (Fig 1).

 <sup>1</sup>SAINI, N., <sup>2</sup>SAINI, V., <sup>3</sup>JAIN, S., <sup>4</sup>JAIN, T.
<sup>1</sup>Department of Periodontics & Implantology, Govt Dental College & Hospital , (RUHS-CODS), Jaipur.
<sup>2</sup>NIMS Dental College and Hospital, Jaipur.
<sup>3</sup>Medical Officer (Dental), Community Health Centre, Nawacity, Nagaur
<sup>4</sup>Govt District Hospital, Pali.

Address for Corresponding : Dr. Neha Saini Department of Periodontology Govt Dental College & Hospital, (RUHS-CODS), Jaipur. Email : drneha.perio@gmail.com

Received : 3 Nov. 2020, Published : 31 Dec. 2020

How to cite this article: Saini, N., Saini, V., Jain, S., & Jain, T. (2021). Juvenile Peripheral Ossifying Fibroma – A rare case report and review. UNIVERSITY JOURNAL OF DENTAL SCIENCES, 6(3)



Fig 1: Exophytic pedunculated gingival growth between lower anterior teeth (31, 32)

Intraoral examination revealed that the growth was pedunculated, with its base of origin interdentally between 31, 32; extending labially from the mesial aspect of 41 up to mesial aspect of 33. Open contacts were present between 41, 31, 32, 33 with 31 and 32 displaced lingually. The growth measured 11 mm\*6mm, was painless, reddish pink with nodular & irregular shiny surface (fig 2). The gingiva with respect to 42, 41,31,32,33,34 was red and erythematous, soft in consistency with rolled out margins. Bleeding on probing was present with a probing depth of 3-4 mm and grade I mobility in relation to 31, 32. The oral cavity presented inadequate oral hygiene status, and rest of the oral cavity showed no unusual features. There was no accompanying cervical lymphadenopathy and no relevant systemic history. Routine hemogram was found to be normal.



Fig. 2 : Painless, reddish pink gingival growth with nodular & irregular shiny surface measuring 11 mm\*6mm.

Widening of periodontal ligament, mild horizontal bone loss with cuffing in the crestal region was seen in relation to 31,32 in the intraoral periapical radiograph (fig 3).



Fig.3: IOPA showing widened periodontal ligament, mild horizontal bone loss with cuffing in the crestal region

The case was provisionally diagnosed as peripheral ossifying fibroma based on the history, radiographic and clinical features. Pyogenic granuloma and peripheral giant cell granuloma were among the differential diagnosis.

The treatment plan included scaling and root planing (Phase I therapy), surgical excision of the lesion and strict recall protocol. Consent for the surgical procedure was obtained from the patient after proper counselling.

After the gingival inflammation subsided, surgical procedure was performed under local anaesthesia. The periodontal local flap surgery extending from 41 to 33 was done with complete excision of the growth, the area was aggressively debrided up to the bone level and crestal osteoplasty was done. It was made sure that all the excessive soft tissue was removed interdentally resulting in close to normal gingival architecture at the time of suturing. A frenotomy was performed with the respect 41 and 31 to address the high frenal attachment. Direct interrupted sutures were given and periodontal dressing was applied. Excised tissue was stored in the 10% formalin and sent to the department of oral pathology for histopathological examination.

Diclofenac 50mg with 325mg paracetamol was prescribed three times a day for 3 days to prevent post-operative discomfort, amoxicillin with clavulanic acid 625 mg bd for 5 days to prevent infection and the patient was advised to rinse with 0.2% chlorhexidine gluconate solution twice daily for 14 days to aid in plaque control. Sutures were removed after 7 days & one month follow up showed uneventful healing at the surgical site.

Histopathology of the biopsied specimen showed hyperplastic stratified squamous epithelium with elongated rete ridges extending into the underlying connective tissue, which was extremely cellular with numerous ossifications of varying sizes and shapes. Abundant blood vessels and chronic inflammatory cells were seen. Overall histopathological features were suggestive of Juvenile peripheral ossifying fibroma (figure 4).



Fig 4 :Histopathological picture showing hyperplastic stratified squamous epithelium with elongated rete ridges ,inflammatory cells & numerous ossifications

The patient was followed as per Merin's recall protocol at every 3 months after surgical procedure for one year & no recurrence was observed (figure 5,6).



Fig 5: postop uneventful healing



Fig6:postop uneventful healing

# **Discussion:**

JPOF is a reactive focal overgrowth, one of its kind occurring on the gingiva, among other reactive lesions including pyogenic granuloma, fibrous hyperplasia & peripheral giant cell granuloma. [1,5,6] These reactive lesions appearing as hyperplastic growth in localised area,[5] occur as a response to various irritants like dental calculus, plaque, microorganisms, food debris, dental appliances & restorations and also have been attributed to hormonal causes (supported by higher incidence among females, in second decade, and declining incidence after the third decade).[7]POF may also occur due to genetic mutations.[8] Other such lesions are seen in pyogenic granuloma, fibrous hyperplasia (fibrous epulis), peripheral giant cell granuloma (PGCG). POF is a nonneoplastic tumor-like growth of the soft tissue which is composed of fibrocellular tissue and contains one or more mineralized tissues.

The lesion was first reported as "alveolar exostosis" by Shepherd in 1844 [9]. The term Peripheral Ossifying Fibroma was coined by Eversol and Robin in 1972.[5] POF is a misnomer and it is not considered a true neoplasm.[10] The various synonymous nomenclatures given to POF include epulis, peripheral fibroma with calcification, peripheral fibroma with cementogenesis, peripheral cementifying fibroma, and peripheral cemento-ossifying fibroma.[11]

POF accounts for 10%–18% of the reactive lesions of gingiva.[12] It shows predilection for maxilla (60%), incisor cuspid area (>50%) and females (1.3–1.7 times more than males).[13,14] The clinical appearance of POF is characteristic but not pathognomonic. POF is a slow growing progressive lesion, sessile or pedunculated, smooth surfaced, usually firm and nontender to palpation. Most of the POF cases have been reported to have a size <2 cm in diameter.[15] The lesion may be of the same color as the surrounding mucosa or appear red or reddish blue with intact or ulcerated surface characteristics due to trauma to the growth.[16] The lesion occurs between the age of 5-25 yrs with peak incidence at 13 yrs of age with a decreasing trend with increase in age.[17]

Radiographically, POF may not yield significant changes, but in some cases there may be varying radiodensity within the lesion, depending on the degree of mineralization. No underlying bone involvement is seen on the radiograph, but rarely there can be superficial erosion of the bone or migration and mobility of adjacent teeth.[15,17] Superficial bone changes, and focal areas of calcification have been rarely reported as in the present case which showed bone involvement with cuffing defect. The presence of small radiopaque lesion may be detected by decreasing kilovoltage and increasing milliamperes.[18] Computed tomography and magnetic resonance imaging are also helpful in larger lesions.[19,20]

POF occurs mainly on the interdental gingiva and though etiopathogenesis is uncertain, it has been suggested to be arising from the periodontal ligament (PDL) [1,5] (due to its occurrence only on the gingiva, its proximity to the PDL, presence of oxytalan fibres and the fibrocellular response similar to other reactive lesions of PDL origin).[21] Chronic irritation caused by the etiological factors leads to connective tissue metaplasia with bone formation and calcification.[22] Histologically, POF is nonencapsulated mass of highly cellular fibroblastic connective tissue covered by stratified squamous epithelium, which when ulcerated may have fibrinopurulent membrane and granulation tissue. Gardner 6 stated that POF cellular connective tissue is so characteristic that a histologic diagnosis can be made without ambiguity, regardless of the presence or absence of calcification. Buchner and Hansen[13] observed randomly distributed mineralised material of 3 types - consisting of bone (woven, lamellar, trabecular), cementum-like material, or dystrophic calcifications are dispersed in the connective tissue.

# **Conclusion:**

The definitive diagnosis of POF is made by the histologic evaluation of biopsy specimen. Treatment includes addressing the etiologic factor (i.e. removal of calculus, illfitted dental appliances, and rough restorations) and complete surgical excision of POF including the aggressive curettage of the involved PDL and periosteum for preventing recurrence. The recurrence rate of POF has been considered high for reactive lesions making postoperative follow-up mandatory.

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