Case Report

DOI: 10.7860/JCDR/2026/82640.22768

Dentistry Section

An Atypical Peripheral Ossifying Fibroma of the Mandible in a Male Patient: A Clinico-pathologic and Surgical Case Report

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ABSTRACT

Peripheral Ossifying Fibroma (POF) is an uncommon reactive, inflammatory gingival lesion believed to originate from the periodontal ligament in response to chronic irritation such as plaque, calculus, trauma, or defective restorations. It belongs to the spectrum of reactive gingival overgrowths, including pyogenic granuloma and Peripheral Giant Cell Granuloma (PGCG). Pathologically, POF displays a fibrocellular connective tissue matrix with characteristic areas of mineralisation. These may appear as immature woven bone, cementum-like deposits, or dystrophic calcifications. The presence of these mineralised foci is a key histopathological hallmark and reflects the osteogenic potential of periodontal ligament fibroblasts, which are capable of differentiating into cementoblasts or osteoblast-like cells under persistent inflammatory stimulation. POF typically occurs in young females and most frequently involves the maxillary anterior region. This case report describes an atypical presentation of POF in a 54-year-old male who reported a slowly enlarging, asymptomatic gingival mass in the mandibular canine–premolar area. Clinical examination revealed a firm, sessile, well-circumscribed nodular overgrowth in the attached gingiva of the mandibular left canine-premolar region, with normal surface appearance and no bleeding. Adjacent teeth showed pathologic migration, and periodontal support appeared compromised clinically. As part of the diagnostic work-up, an intraoral periapical radiograph was obtained, which demonstrated bone loss, widening of the periodontal ligament space, and migration of adjacent teeth, but no radiopaque material. Complete surgical excision was performed, and the specimen was submitted for histopathological evaluation. The gingival origin, clinical behaviour, radiographic and histopathological evaluation supported the diagnosis of POF. The patient was followed up regularly for three years and demonstrated no recurrence. This case underscores the importance of detailed clinical, radiographic and histopathologic evaluation when assessing atypical gingival enlargements occurring outside their usual demographic and anatomical patterns.

Keywords: Calcification foci, Gingival growth, Reactive lesion

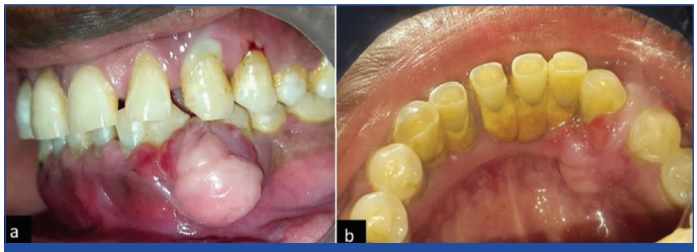
CASE REPORT

A 54-year-old male reported with a chief complaint of painless gingival overgrowth in the lower front teeth region for the past four months. The patient reported that the swelling initially appeared as a small, painless nodule on the gingiva, which gradually increased in size over a period of four months to its present dimensions. The onset was insidious, and the growth remained non-tender throughout its progression. The patient denied any history of pain, pus discharge, ulceration, foul taste, or spontaneous bleeding. There were no associated systemic symptoms such as fever, malaise, weight loss, or difficulty in swallowing, chewing, or speech. The patient did not recall any recent trauma to the area, nor did he experience any sudden increase in size of the lesion. He acknowledged poor oral hygiene habits, reporting long-standing plaque accumulation and calculus deposits.

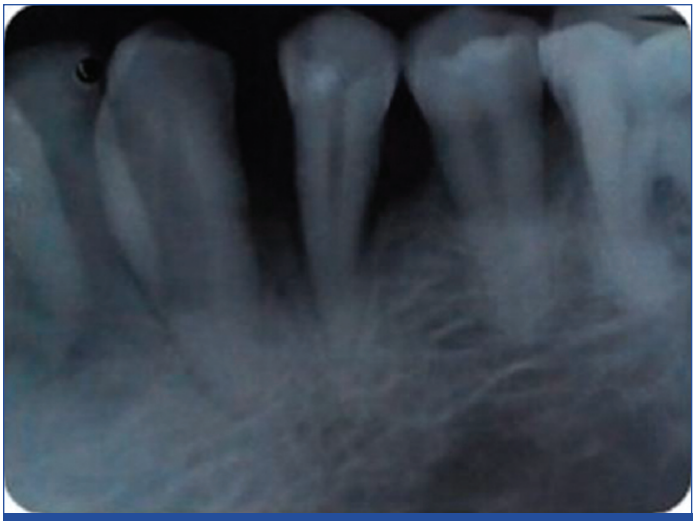
The patient reported no known systemic illnesses, including diabetes, hypertension, cardiovascular disease, thyroid disorders, or bleeding abnormalities. He denied any drug allergies and was not on any long-term medications. There was no history of previous hospitalisations or major surgeries.

The patient admitted to irregular dental visits and poor oral hygiene practices. He had not undergone prior periodontal therapy, nor did he have a history of trauma or similar swellings in the past. No dental prostheses, orthodontic appliances, or restorations were present that could have acted as irritants. The patient denied habits such as smoking, alcohol consumption, betel nut/areca nut chewing, nail biting, lip biting, or any parafunctional habits associated with chronic gingival irritation. Extraoral examination revealed no facial asymmetry, swelling, discolouration, or palpable lymphadenopathy. The patient appeared in good general health, and the overlying skin and facial musculature were normal. There was no tenderness on palpation of the submandibular or submental regions.

Intraoral examination showed moist oral mucosa with no ulcerations or abnormalities in the buccal mucosa, tongue, palate, or floor of the mouth. Clinical examination revealed clinical attachment loss involving the mandibular anterior teeth, which the patient stated had been present for several years. The patient presented with poor oral hygiene. A sessile overgrowth was noticed in the attached gingiva of mandibular left canine-premolar region, extending from the buccal to the lingual side measuring about 1.7×1.5 cm buccally and 1.2×0.8 cm lingually. The surface appeared pink and smooth with regular borders [Table/Fig-1]. The lesion was firm in consistency and non-tender to palpation. Spacing was present between 33 and 34, but the associated teeth were non tender without mobility. Intraoral Peri Radiograph (IOPA) revealed horizontal bone loss between 33 and 34 with interdental spacing [Table/Fig-2].



[Table/Fig-1]: a) Preoperative photograph showing the lesion on the buccal side; b) Preoperative photograph showing the lesion on the lingual side.



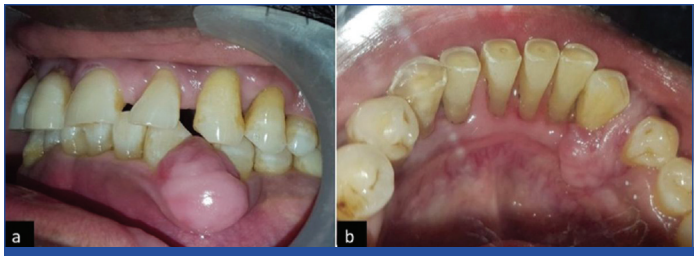
[Table/Fig-2]: Intraoral Periapical radiograph (IOPA) revealing horizontal bone loss between 33 and 34.

Provisional Diagnosis

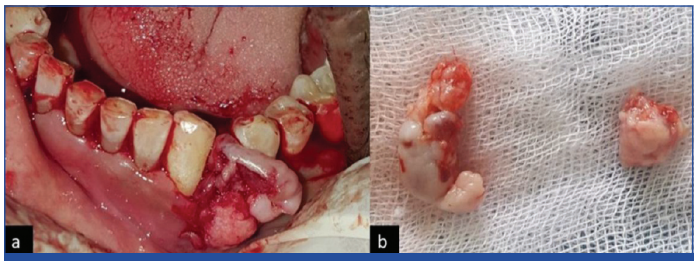
The clinical appearance of a firm, sessile gingival overgrowth in the mandibular anterior region required consideration of several potential differential diagnosis. A pyogenic granuloma was initially considered; however, the lesion lacked the characteristic bright red, soft, and highly vascular appearance, and did not exhibit spontaneous bleeding. A PGCG was another strong differential diagnosis, yet the absence of a bluish-purple coloration and the firm rather than soft consistency made this diagnosis less probable. A fibroma, typically arising in response to trauma, was also contemplated, but its usual presentation on the buccal mucosa or areas prone to irritation made it less compatible with the location and behaviour of this lesion. Peripheral odontogenic fibroma was included due to its firm texture, though it is far less common and often presents with associated displacement of teeth or calcifications. Ultimately, the lesion's firm consistency, gingival location, gradual enlargement, and clinical characteristics made Peripheral Ossifying Fibroma (POF) the most likely provisional diagnosis, later confirmed histopathologically.

Investigations and Treatment

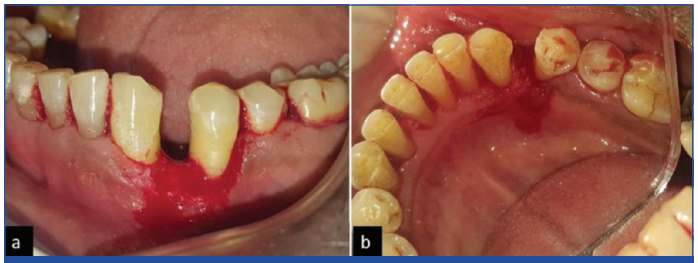
Informed consent was obtained before the procedure, following an explanation of the planned intervention. Also, consent was obtained from the patient after explaining the purpose of the case report, the intended use of clinical data and images, and ensuring confidentiality. Scaling and root planing were performed using ultrasonic scalers with standard periodontal tips, followed by hand instrumentation with Gracey curettes to ensure thorough root surface debridement [Table/Fig-3]. The patient was advised to use 0.12% chlorhexidine mouthrinse twice daily for one week. The patient was then recalled for the surgical phase, and routine blood investigations, including a mini surgical profile consisting of complete blood count, bleeding time, clotting time, and blood sugar estimation, were completed before surgery to confirm the patient's systemic fitness for the planned procedure. The procedure was carried out under local anaesthesia (2% lignocaine with 1:80,000 adrenaline). Following adequate anaesthesia, a full-thickness mucoperiosteal flap was carefully reflected using a No. 15 Bard-Parker blade and periosteal elevator to expose the lesion. The lesion was excised en bloc along with the underlying periosteum using surgical curettes and tissue forceps [Table/Fig-4]. The area was then thoroughly degranulated and debrided with Gracey curettes and bone curettes to ensure complete removal of inflamed tissue and to create a clean, healthy surgical bed [Table/Fig-5]. Haemostasis was achieved with light pressure and suction. A periodontal pack was subsequently placed for wound protection [Table/Fig-6]. Postoperative instructions were explained to the patient, and a course of analgesics and antibiotics was prescribed as per standard protocol.



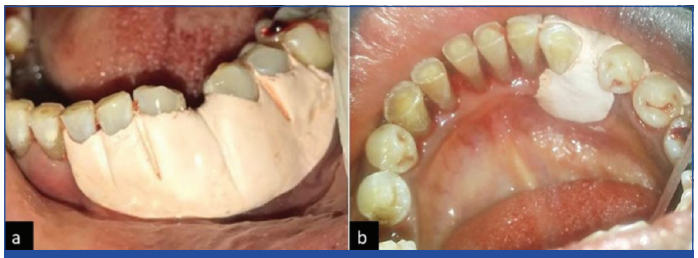
[Table/Fig-3]: a) Post Phase1 therapy (Scaling and root planing) –buccal view; b) Post Phase1 therapy (Scaling and root planing)- lingual view.



[Table/Fig-4]: a) Intraoperative view during excision; b) Excised lesion.



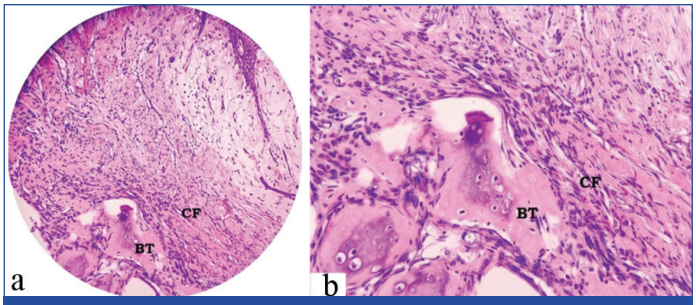
[Table/Fig-5]: a) Site degranulated after excision-buccal; b) Site degranulated after excision-lingual.



[Table/Fig-6]: Periodontal pack placed: a) Buccal view; b) Lingual view.

Histopathology and Final Diagnosis

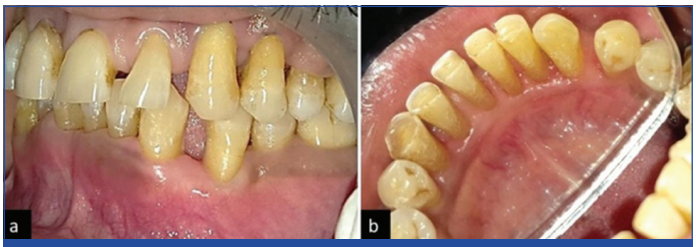
Histopathological examination of the excised specimen was performed using routine Haematoxylin and Eosin stain. Under low-power magnification (10x), the section showed a connective tissue stroma and foci of calcifications with bony trabeculae. The underlying connective tissue stroma, examined under high-power magnification (40x), demonstrated densely packed collagen fibres, many of which were hyalinised and irregularly arranged. Multiple areas of calcification were evident throughout the lesion, ranging from immature basophilic calcified deposits to well-formed bony trabeculae, consistent with the ossifying nature of the lesion. Several budding capillaries with endothelial proliferation were observed, indicating the reactive and vascular component of the lesion. Scattered multinucleated giant cells were also identified within the fibrous stroma [Table/Fig-7]. Histopathological examination confirmed the diagnosis of POF. Although a few scattered multinucleated giant cells were present, their sparse distribution does not exclude POF, as occasional giant cells may appear in reactive gingival lesions. The lesion predominantly showed multiple foci of calcification, immature ossification, and well-formed bony trabeculae within a dense collagenous stroma, features characteristic of POF and distinct from PGCG, which typically exhibits numerous clustered giant cells in a vascular, haemorrhagic stroma. Clinically, the firm, pale-pink lesion arising from the interdental papilla further supported POF.



[Table/Fig-7]: Stain: Haematoxylin and eosin; a) Under low-power magnification (10x), the section showing connective tissue stroma and foci of calcifications with Bony Trabeculae (BT). b) Under high-power magnification (40x), the section shows densely packed Collagen Fibers (CF) and well-formed BT.

Follow-Up

The patient was consistently followed up [Table/Fig-8], and the healing was uneventful without recurrence. The patient was monitored for a period of three years following surgical excision to evaluate healing and detect any signs of recurrence. Follow-up visits were scheduled at two-week intervals during the initial healing phase, then monthly for the first six months, and subsequently every six months as part of supportive periodontal therapy. To minimise the likelihood of recurrence, an outcome known to be relatively common in POF, several preventive measures were implemented. These included complete excision of the lesion along with removal of the periosteum, thorough degranulation and debridement of the underlying tissues, and elimination of all local irritants. The patient underwent repeated sessions of professional plaque and calculus removal and received continuous reinforcement of oral hygiene instruction. Through this structured maintenance programme and careful long-term surveillance, the patient remained free of recurrence throughout the entire three-year follow-up period.



[Table/Fig-8]: a) Postoperative view-buccal side; b) Postoperative view-lingual side.

DISCUSSION

Reactive gingival overgrowths occur in response to local irritants such as plaque, calculus, faulty restorations or prosthesis, trauma and iatrogenic factors. Literature speaks of many such growths, each exhibiting a specific histopathology. These include pyogenic granuloma, fibrous epulis, PGCG, POF, etc., [1-3]. In the present case, the most probable etiological factors contributing to the development of the lesion were chronic plaque accumulation, persistent local irritation from long-standing poor oral hygiene, and underlying periodontal attachment loss in the mandibular anterior region. No history of trauma, parafunctional habits, or ill-fitting dental restorations was reported by the patient, thereby supporting chronic inflammatory irritation as the primary precipitating factor. A thorough clinical examination to assess the lesion's size, surface texture, consistency, colour, base, and exact location, along with evaluation of local irritants such as plaque, calculus, or defective restorations, is mandatory. Periodontal assessment should include probing depths, attachment levels, bleeding tendency, and mobility of adjacent teeth. Radiographic evaluation, typically using an intraoral periapical radiograph, is performed to rule out underlying bone involvement, identify focal radiopacities suggestive of calcifications, and assess the periodontal status. Definitive diagnosis requires complete surgical excision of the lesion followed by histopathological examination to distinguish POF from clinically similar entities such as pyogenic granuloma, PGCG, and peripheral odontogenic fibroma [Table/Fig-9] [4-8]. Although a few multinucleated giant cells were observed in the histopathological section, this finding does not preclude the diagnosis of POF. Occasional giant cells may be seen in reactive gingival lesions as part of a localised inflammatory or reparative response. In the present case, the dominant microscopic features strongly favoured POF, including the presence of multiple foci of calcification, immature ossification, and well-formed bony trabeculae within a densely collagenous and partially hyalinised connective tissue stroma. These mineralised components are characteristic of POF [9] and are not typical of PGCG, which usually presents with numerous giant cells arranged in clusters within a highly vascular, haemorrhagic background. Furthermore, the giant cells in this case were few in number and scattered, lacking the extensive distribution expected in PGCG [10]. Clinically, the lesion exhibited a firm consistency, pale-pink colour, and origin from the interdental papilla, features consistent with POF rather than the bluish-red, highly vascular appearance commonly associated with PGCG. Therefore, integrating both the clinical presentation and the predominance of ossifying elements in the histology, the diagnosis of POF is substantiated despite the presence of a small number of giant cells. POF is a reactive gingival lesion with a prevalence rate of 10% to 18% and has a higher predilection for females and is known to occur in the second and third decades of life, showing a propensity for the anterior maxilla [11,12]. In contrast to these facts, this is a unique case report describing a male in his fifth decade of life with the lesion occurring in the mandible, which could be a rare occurrence. Various theories explain the development of the lesion. POF is considered to be a non-neoplastic growth of the gingiva, contributing to 9% of all gingival growths and is thought to originate from the periodontal ligament [13]. Chronic irritation resulting from gingival trauma, calculus and foreign body in the gingival sulcus could cause inflammatory hyperplasia of the periosteal and periodontal membrane and provoke metaplasia in connective tissue, leading to the formation of bone or dystrophic calcification [14].

In this case, the patient had poor oral hygiene, which may be the aetiology. The plaque and calculus deposits could have been a source of irritation. This theory is widely accepted because of the close proximity of the gingiva to the periodontal ligament and the occurrence of the lesion from the interdental papilla. Also, the occurrence of oxytalan fibres within the mineralised matrix in some

Study (Year)	Age/Sex	Site	Size/ Duration	Treatment Performed	Follow-up	Recurrence
Takagi R et al., (2024) [4]	68/male	Right maxillary gingiva	60 mm diameter (giant lesion)/ rapidly progressive for 6 months	Wide excision with thorough curettage	10 months	None
Xavier SA et al., (2024) [5]	50/Female	Posterior mandibular gingiva	2.1 x 1.5 x 1.2 cm/5 months	Excisional biopsy to periosteum + curettage	4 months	None
El Gaouzi R et al., (2024) [6]	42/Female	Anterior and posterior mandibular gingiva (large lesion)	6 cm × 4 cm/ 2 years	Complete excision+ curettage	3 Months	None
Pandey R et al., (2024) [7]	(2 cases) 22/male and 33/male, respectively	Posterior mandibular gingiva and anterior maxillary gingiva, respectively	2x1 cm/2 months and 6x7x3 mm/2 weeks respectively	Excision to periosteum (scalpel technique)	1 year and 2 years, respectively	None
Balachandran A et al., (2023) [8]	28/Female	Maxillary posterior gingiva	1.5cmx1.5 cm/18 months	Complete excisional biopsy+ curettage	1 year	None

[Table/Fig-9]: Reported cases of Peripheral Ossifying Fibroma (POF) showing patient demographics, lesion site, size, duration, treatment, follow-up, and recurrence [4-8].

lesions and the fibrocellular response similar to gingival lesions arising from the periodontal ligament strengthens this theory [8,15]. The other theory states that POF could occur secondary to the fibrosis of the granulation tissue of pyogenic granuloma [16]. This is because both lesions exhibit similarity in terms of site of occurrence, gender involved, and clinical and histological features. POF can cause resorption of the alveolar crestal bone involved, which, in turn, could cause pathological migration of the adjacent teeth [17]. The present case also shows radiographic evidence of bone loss associated with pathologic migration of adjacent teeth, along with widening of the periodontal ligament. But the radiograph didn't reveal any radioopaque material. According to Kumar SKS et al., not all POF lesions show radioopacity unless they are significantly large and long-standing and this explains the present case [18]. The histological diagnosis, irrespective of the presence or absence of calcification, can be confirmed because of the characteristic cellular connective tissue of POF. Highly cellular fibroblastic connective tissue is observed in ulcerated lesions, whereas non-ulcerated lesions may exhibit partly collagenised tissue. The mineralised component may contain lamellar or woven bone, cementum-like material or even dystrophic calcifications. Dystrophic calcifications are mostly seen in ulcerated lesions. Also, the mineralised component of POF could differ from 23% to 75% [7]. The current case showed histological evidence of bony trabeculae suggesting lamellar/woven bone and there were no dystrophic calcifications or clinical evidence of ulceration. Treatment demands the complete excision of the lesion as POF has high recurrence rates (8% to 20%) [2]. With regards to the present case, the lesion was completely excised with the affected periosteal component along with removal of all irritants to prevent recurrence. The patient was regularly recalled for periodontal maintenance, and no recurrence was noted during the three-year follow-up period. The present case highlights how subtle clinical features, absence of early radiopacity, and proximity to periodontal structures can lead to diagnostic uncertainty unless corroborated by histology. Clinically, this reinforces the need for routine periodontal evaluation, meticulous plaque control, and follow-up after excision. Future implications include the need for standardised diagnostic criteria, exploration of molecular markers that may clarify the true origin of POF, and long-term multicentre studies to better understand its biological behaviour and recurrence patterns. Documenting such cases contributes to improved diagnostic accuracy and strengthens evidence-based periodontal practice.

CONCLUSION(S)

The present case highlights the importance of thorough clinical assessment and histopathological evaluation in diagnosing POF, especially when classical radiographic mineralisation is absent in early or intermediate stages. Its significance lies in the unusual presentation in a 54-year-old male, occurring in the canine-premolar region, which is less commonly reported compared with the typical predilection for younger females. Despite being long-standing, the lesion showed no radiopacity, underscoring the variability in the maturation and mineralisation patterns of POF. The absence of recurrence after three years of follow-up further reinforces the importance of complete surgical excision, including the periosteum and meticulous elimination of local irritants. Future implications include the need for comprehensive studies exploring site-specific behaviour, gender variation, and molecular pathways to enhance understanding of POF pathogenesis and long-term outcomes.

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PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Sep 02, 2025
- Manual Googling: Dec 20, 2025
- iThenticate Software: Dec 22, 2025 (7%)

ETYMOLOGY: Author Origin

EMENDATIONS: 6

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. Yes

Date of Submission: **Aug 09, 2025**
Date of Peer Review: **Nov 11, 2025**
Date of Acceptance: **Dec 24, 2025**
Date of Publishing: **Mar 01, 2026**