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Original Article

Rare Case of Mandibular Peripheral Ossifying Fibroma in a 70-Year-Old Male: Diagnostic Challenges

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ABSTRACT

Peripheral ossifying fibroma (POF) is a benign growth localized to the gingiva and alveolar mucosa, often arising from periodontal ligament cells. It most commonly occurs in women in their twenties, making diagnosis challenging due to overlapping features with other oral lesions. Here, we report an unusual case of POF in the left lateral mandible of a 70-year-old male with two semicircular implant-supported bridges in both jaws. CBCT and panoramic imaging revealed no intraosseous abnormalities. Histopathological evaluation confirmed the lesion as POF. This case is remarkable because POF is predominantly observed in younger female patients, highlighting the atypical presentation in an elderly male.

Keywords: Peripheral ossifying fibroma, Irritation fibromatosis, Giant cell lesions, CBCT

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Introduction

Peripheral ossifying fibroma (POF) is a benign growth that arises locally from the gingiva and alveolar mucosa. Clinically, it appears as a firm, slow-expanding nodule, usually under 2 cm, and is generally asymptomatic. Under the microscope, the lesion is composed of fibrous connective tissue with varying numbers of fibroblasts. The presence of distinct areas of metaplastic bone is often a key feature in establishing a diagnosis [1, 2]. In the literature, POF has been described using multiple terms, including peripheral cementifying fibroma, peripheral fibroma with cementogenesis, peripheral fibroma with osteogenesis, peripheral fibroma with calcification, calcified or ossified fibrous epulis, and calcified fibroblastic granuloma [1].

POF occurs most frequently in women during their twenties but can manifest in a variety of intraoral locations such as the tongue, lips, floor of the mouth, palate, and the alveolar ridges of both jaws [3]. Genetic alterations in the SATB2 gene, leading to its functional inactivation through various molecular pathways, may contribute to the lesion's development. Proliferating cell nuclear antigen (PCNA)-positive cells indicate high cell turnover, which can affect treatment planning. Such mutations may be linked to SATB2-associated syndromes [4], which can present with neurodevelopmental delays, behavioral issues, palatal clefts, and dental or bone abnormalities, though involvement of other organs is uncommon [5].

Differential diagnosis of POF is complicated because it shares clinical and histological features with other lesions, including pyogenic granuloma, peripheral giant cell granuloma, irritation fibroma, and certain soft-tissue metastases [6]. Despite this, POF is understood as a reactive, non-cancerous mass of soft tissue that generally originates in the interdental papilla. Its surface may feel soft or firm, and the coloration can range from light pink to deep red [7].

Case study

A 70-year-old male presented to the University Hospital Centre Zagreb's Oral Surgery Department with a large fibrous mass in the distal left mandible **(Figure 1)**. The patient had two acrylic bridges supported on four implants. The implants had been placed six months before, prior to the appearance of the mass, and the bridges were three months old. The patient reported noticing swelling in the posterior left mandible for approximately three months. Panoramic X-ray and CBCT revealed no radiolucency, radiopacity, or periimplant bone changes **(Figure 2)**. The lesion had a smooth surface, a broad base, and extended toward the left sublingual area. The patient had poor oral hygiene, smoked, and consumed two to three alcoholic drinks daily.

On palpation, the mass was firm, fixed to the alveolar crest, and extended to the sublingual region, measuring 3.5 × 2 cm. Differential diagnosis included irritation fibromatosis, peripheral giant cell fibroma, peripheral ossifying or non-ossifying fibroma, or a malignant lesion, with definitive diagnosis pending histopathology. Surgical excision was performed **(Figure 3)**. Submucosal layers were dissected using scalpel and electrocautery. A feeding artery from the sublingual area was ligated with 4/0 resorbable suture, and the mass was removed entirely **(Figure 4)**. Some areas were allowed to heal by secondary intention, while the majority of the incision was sutured with 4/0 silk **(Figures 5 and 6)**. Hemostasis was ensured via electrocautery. The procedure was conducted under local anesthesia. Follow-up clinical review and suture removal were completed seven days post-surgery **(Figure 7)**.

a) b)

Figure 1. Initial clinical presentation of the lesion.

Figure 2. Panoramic X-ray of the patient's mandible.

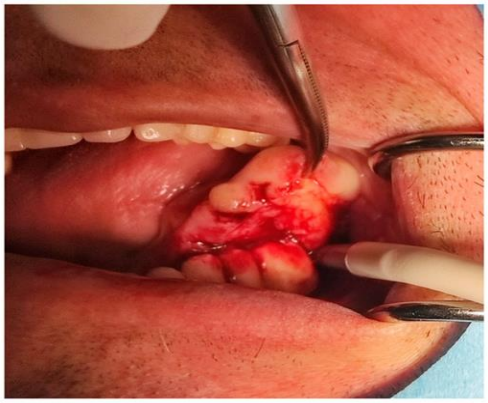

Figure 3. Surgical removal of the fibrous mass.

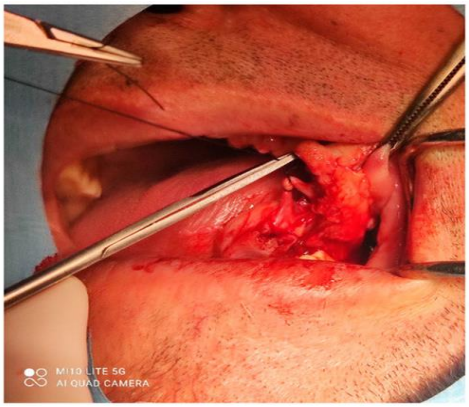

Figure 4. Ligation of the feeding artery during excision.

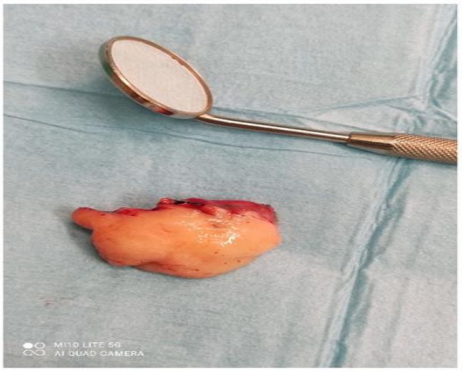

Figure 5. Measurement of the excised lesion.

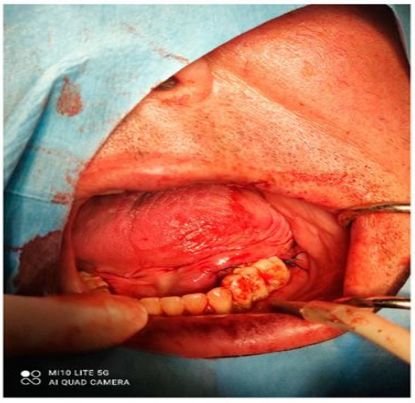

Figure 6. Immediate post-suturing view of the surgical site.

Figure 7. Follow-up evaluation seven days after surgery, including suture removal.

The excised tissue was submitted for histopathological examination. The specimen showed epithelium with reactive changes and a multilayered structure. The subepithelial connective tissue contained a dense linear infiltration of mononuclear inflammatory cells. No signs of Lichen were observed. Within the thick fibrous stroma, small areas resembling cemento-osseous lacunae were present. Centrally, clusters of hemosiderin deposits and multinucleated giant cells were identified. Interestingly, a small salivary gland with expanded intra- and interlobular ducts was noted. Peripheral regions of the specimen contained remnants of the mucocele wall **(Figure 8)**.

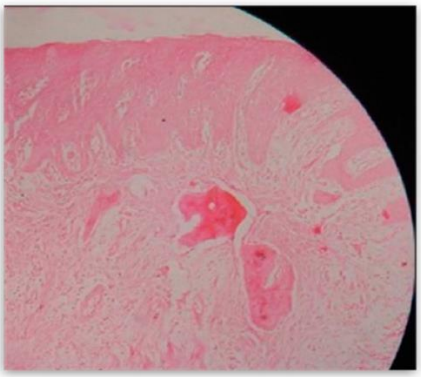

Figure 8. Histopathology of the lesion at 4× magnification.

Malalignment between the patient's upper and lower acrylic bridges, together with parafunctional habits such as bruxism, likely contributed to the lesion's development.

The precise cause of peripheral ossifying fibroma (POF) is unclear, though factors such as mechanical irritation, plaque accumulation, and poorly fitted prostheses are considered contributors. In the literature, this lesion is also referred to as fibrous epulis or calcifying fibroblastic granuloma [8]. Immunohistochemical studies have linked peripheral fibromas and fibromatoses to markers including CD34, α-smooth muscle actin (α-SMA), vimentin, Ki-67 (Mib1), and TGF-α, which may stimulate fibroblast proliferation and fibrous tissue activity [9].

POF represents approximately 3.1% of all oral tumors and 9.6% of gingival lesions [10]. Unlike central ossifying fibroma, which is an osteogenic neoplasm originating near the root apex and expanding within bone, POF is a reactive, soft-tissue growth confined above the alveolar ridge. Peripheral POF, like peripheral giant cell granuloma (PGCG), arises from the periodontal ligament and manifests only in the soft tissue. Clinically, POF is seen as a slow-growing, well-circumscribed nodule with a broad base, firm consistency, smooth surface, and mucosa that generally appears normal [11].

Since its first descriptions in the late 1940s, POF has been identified under multiple names, including epulis, peripheral fibroma with calcification, peripheral fibroma with osteogenesis, calcified fibroblastic granuloma, peripheral cementifying fibroma, peripheral fibroma with cementogenesis, and peripheral cemento-ossifying fibroma [8, 12]. Approximately 60% of cases are located in the maxilla, and most affect the interdental papilla of incisors and canines. Typically, POF occurs in patients aged 10–30, with a peak around 20 years, and incidence decreases significantly after 30; only 0.5% of cases appear in elderly patients [13–16]. Hormonal influences contribute to the higher prevalence in women [17]. Most lesions are under 1.5 cm in diameter, although rare "giant" POFs have been reported, measuring up to 9 cm [18]. This case is unusual due to the patient being a 70-year-old male with elevated parathyroid hormone (PTH), highlighting an atypical presentation.

Ogbureke *et al*. documented a case involving a 44-year-old male who presented to the emergency room due to a swelling in the posterior right mandibular region, persisting for approximately three months. His medical history included a family background of type 2 diabetes, hypertension, and cardiovascular disease. Notably, the patient had received two dental implants in the right distal mandibular quadrant three months

prior to noticing the mass. Clinically, distinguishing between a peripheral giant cell lesion and peripheral ossifying fibroma (POF) proved challenging. Histopathological examination is required for definitive differentiation: both lesions exhibit giant cells, but only POF contains cemento-ossifying lacunae [19].

Gulati *et al*. presented a 56-year-old female patient with a firm oral mass, measuring 3.5 × 4 × 3 cm, located in the region of teeth 13–23, all of which were affected by advanced periodontal disease (stage III). The lesion was sessile, erythematous, and asymptomatic. Panoramic radiography indicated no abnormal findings in the underlying bone. Surgical excision was performed using a 940-nm diode laser (Biolase®, Foothill Ranch, CA, USA). Histology revealed fibrocellular stroma interspersed with islands of osteoid tissue, both mature lamellar and immature bone, with peripheral osteoblastic activity in certain areas. Small clusters of endothelial and inflammatory cells were also observed. The findings confirmed the diagnosis of POF [20].

According to Prasad *et al*., POF may originate as a pyogenic granuloma, which subsequently undergoes fibrous maturation and mineralization [21].

Satish *et al*. proposed that POF arises from inflammatory hyperplasia of periodontal ligament or periosteal tissue, with fibrous tissue undergoing metaplasia, leading to dystrophic calcification and formation of bone-like structures [22].

Rallan *et al*. described a case of a 12-year-old boy who observed swelling in the anterior maxilla approximately one month before his dental visit. Intraoral examination revealed a well-circumscribed, firm, erythematous, oval mass on the palatal aspect of the maxillary incisors, measuring about 2 × 2 cm. The lesion was sessile, asymptomatic, and caused occlusal interference. They concluded that the growth represented a reactive connective tissue lesion, commonly occurring in the anterior maxilla, particularly among young females. Excisional biopsy followed by histopathological analysis is considered the standard approach. Due to the recurrence risk, periodic follow-up is essential [23].

Nadimpalli and Kadakampally reported a 23-year-old female with a recurrent POF in the mandibular right premolar region. The case highlighted the clinical, radiographic, and histological features, emphasizing differential diagnosis, treatment, and follow-up. Early recognition, complete surgical excision, and curettage of adjacent tissue are critical to reduce recurrence, which has been reported at 8–20%. Continuous monitoring after surgery is recommended due to the lesion's growth potential [24].

Sudhakar *et al*. reported a case involving a 55-year-old woman who presented with a growth in the upper jaw, localized at the left maxillary second incisor. Imaging via intraoral periapical radiograph (IOPAR) and panoramic X-ray revealed no abnormal findings aside from generalized horizontal alveolar bone loss. Histopathological examination, similar to our case, showed highly cellular connective tissue with plump fibroblasts embedded in a delicate fibrillar stroma, interspersed with regions of woven trabecular bone and osteoid material.

While POF has a known predilection for females in the second and third decades, implicating hormonal influences as a possible contributing factor [25], our patient is an elderly male in his seventies, with the lesion appearing on the left lateral mandible. Previous literature indicates that ossifying and cemento-ossifying fibromas are most frequently observed in women aged 20–50, with common locations including the anterior maxilla and distal hard palate.

Agarwal *et al*. described a 68-year-old female with a soft tissue mass on the left posterior palate. The lesion developed over four months from a small nodule to a larger growth. She had no relevant medical history. Intraoral examination revealed a shiny, elongated pink mass extending from teeth 23 to 27 anteroposteriorly, and from 1 cm lateral to the palatal midline to the occlusal surfaces of the left maxillary molars. Histology confirmed a fibroblastic stroma with overlying epithelium, containing bony trabeculae with osteoblastic rimming, consistent with POF. Postoperative follow-up showed uneventful healing [26].

Katanec *et al*. reported a case of symmetrical palatal fibrous hyperplasia in a 47-year-old patient. Bilateral palatal masses had been present for three years, enlarging over the previous year to exceed 5 cm in diameter. The lesions were firm, sessile, and broadly attached to the palatal artery, with no signs of acute inflammation. Surgical excision, including the removal of adjacent periosteum and periodontal ligament, was performed to reduce recurrence, emphasizing the need to control local irritants [27].

Dutra *et al*. found hyperplastic oral lesions occur more frequently in females and commonly affect the anterior alveolar ridge. Among these, inflammatory fibrous hyperplasia represented 72.09%, pyogenic granuloma 11.79%, giant cell fibroma 7.30%, and POF 5.24% of cases [28].

Borghesi *et al*. documented a unique case of a 50-year-old female with four benign lesions in one hemimandible identified on CBCT: peripheral osteoma (PO), compound odontoma (CO), focal cemento-osseous dysplasia (FocCOD), and cemento-ossifying

fibroma (COF). This case underscores the diagnostic complexity of multiple concurrent oral lesions, highlighting the importance of thorough differential diagnosis [29].

Sangle *et al*., in a retrospective study and literature review, noted that traumatic fibromas are the most frequently observed oral lesions, supporting the idea that chronic irritation is a major contributing factor for fibrous proliferative growths [30].

Conclusion

We report a 70-year-old male patient with histologically confirmed POF on the lingual aspect of the left mandible. This case is noteworthy because POF typically affects females aged 20–30 years. The patient, overweight, had two Toronto bridges on four implants in the upper and lower jaws, with misaligned occlusal surfaces that may have caused irritation, serving as a likely predisposing factor for POF development. Laboratory evaluation revealed elevated parathyroid hormone (PTH) levels, which could have contributed to lesion formation. After complete surgical removal, the site healed uneventfully, with no signs of recurrence, and proper occlusal alignment of the existing prosthetic bridges was achieved.

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Ethics Statement: None

References

1. Buchner A, Hansen LS. The histomorphologic spectrum of peripheral ossifying fibroma. Oral Surg Oral Med Oral Pathol. 1987;63(4):452–61.
2. Bhaskar SN, Jacoway JR. Peripheral fibroma and peripheral fibroma with calcification: report of 376 cases. J Am Dent Assoc. 1966;73(6):1312–20.
3. Maturana-Ramírez A, Adorno-Farías D. A retrospective analysis of reactive hyperplastic lesions of the oral cavity: study of 1149 cases diagnosed between 2000 and 2011, Chile. Acta Odontol Latinoam. 2015;28(2):103–7.
4. Zarate YA, Fish JL. SATB2-associated syndrome: Mechanisms, phenotype, and practical recommendations. Am J Med Genet A. 2017;173(2):327–37.
5. Zarate YA, Kalsner L. Genotype and phenotype in 12 additional individuals with SATB2-associated syndrome. Clin Genet. 2017;92(4):423–9.
6. Pal S, Hegde S, Ajila V. The varying clinical presentations of peripheral ossifying fibroma: a report of three cases. Rev Odonto Ciênc. 2012;27(3):251–5.
7. Neville BW, Damm DD, Allen CM, Bouquot JE. Oral and maxillofacial pathology. Philadelphia (PA): WB Saunders Co; 1995. p. 374–6.
8. Lee KW. The fibrous epulis and related lesions. Periodontics. 1968;6(6):277–92.
9. Rotaru H, Choi JY, Hong SP, Lee YC, Yun KL, Kim SG. Transforming growth factor-α and oral fibroma: immunohistochemical and in situ hybridization study. J Oral Maxillofac Surg. 2003;61(12):1449–54.
10. Walters JD, Will JK, Hatfield RD, Cacchillo DA, Raabe DA. Excision and repair of the peripheral ossifying fibroma: a report of 3 cases. J Periodontol. 2001;72(6):939–44.
11. Baumgartner JC, Stanley HR, Salomone JL. Peripheral ossifying fibroma. J Endod. 1991;17(4):182–5.
12. Gardner DG. The peripheral odontogenic fibroma: an attempt at clarification. Oral Surg Oral Med Oral Pathol. 1982;54(1):40–8.
13. Kfir Y, Buchner A, Hansen LS. Reactive lesions of the gingiva: a clinicopathologic study of 741 cases. J Periodontol. 1980;51(11):655–61.
14. Kendrick F, Waggoner WF. Managing a peripheral ossifying fibroma. ASDC J Dent Child. 1996;63(2):135–8.
15. Jain A, Deepa D. Recurrence of peripheral ossifying fibroma: a case report. People's J Sci Res. 2010;3(1):23–5.
16. Effiom OA, Adeyemo WL, Soyele OO. Focal reactive lesions of the gingiva: an analysis of 314 cases at a tertiary health institution in Nigeria. Niger Med J. 2011;52(1):35–40.
17. Shetty DC, Urs AB, Ahuja P, Sahu A, Manchanda A, Sirohi Y. Mineralized components and their interpretation in the histogenesis of peripheral ossifying fibroma. Indian J Dent Res. 2011;22(1):56–61.
18. Poon CK, Kwan PC, Chao SY. Giant peripheral ossifying fibroma of the maxilla: report of a case. J Oral Maxillofac Surg. 1995;53(6):695–8.
19. Ogbureke EI, Vigneswaran N, Seals M, Frey G, Johnson CD, Ogbureke KU. A peripheral giant cell granuloma with extensive osseous metaplasia or a hybrid peripheral giant cell granuloma–peripheral ossifying fibroma: a case report. J Med Case Rep. 2015;9(1):14.
20. Gulati R, Khetarpal S, Ratre MS, Solanki M. Management of massive peripheral ossifying

fibroma using diode laser. J Indian Soc Periodontol. 2019;23(2):177–80.
21. Prasad S, Reddy SB, Patil SR, Kalburgi NB, Puranik RS. Peripheral ossifying fibroma and pyogenic granuloma: Are they interrelated? N Y State Dent J. 2008;74(2):50–2.
22. Satish BN, Kumar P. Peripheral ossifying fibroma of hard palate: a case report. Int J Dent Clin. 2010;2(1):30–4.
23. Rallan M, Pathivada L, Rallan NS, Grover N. Peripheral ossifying fibroma. BMJ Case Rep. 2013;2013(1):bcr2013009010.
24. Nadimpalli H, Kadakampally D. Recurrent peripheral ossifying fibroma: case report. Dent Med Probl. 2018;55(1):83–6.
25. Das UM, Azher U. Peripheral ossifying fibroma. J Indian Soc Pedod Prev Dent. 2009;27(1):49–51.
26. Agarwal P, Chug A, Kumar S, Jain K. Palatal peripheral ossifying fibroma: a rare occurrence. Int J Health Sci. 2019;13(3):63–6.
27. Katanec T, Bakula A, Filipović-Zore I, Kuna T. Symmetrical fibrous hyperplasia of the palate. Acta Stomatol Croat. 2021;55(3):207–11.
28. Dutra KL, Longo L, Grando LJ, Rivero ERC. Incidence of reactive hyperplastic lesions in the oral cavity: a 10-year retrospective study in Santa Catarina, Brazil. Braz J Otorhinolaryngol. 2019;85(3):399–407.
29. Borghesi A, Tonni I, Pezzotti S, Maroldi R. Peripheral osteoma, compound odontoma, focal cemento-osseous dysplasia, and cemento-ossifying fibroma in the same hemimandible: CBCT findings of an unusual case. Radiol Case Rep. 2017;12(4):756–9.
30. Sangle VA, Pooja VK, Holani A, Shah N, Chaudhary M, Khanapure S. Reactive hyperplastic lesions of the oral cavity: a retrospective survey study and literature review. Indian J Dent Res. 2018;29(1):61–6.