OSSIFYING FIBROMA REVISITED: AN UPDATE ON CLASSIFICATION, PATHOGENESIS & MANAGEMENT

Dr. Nagasaireddy Konda MDS[1], Dr. Anil BudumuruMDS [2], Dr. Mohana Kondapally MDS[3], Dr. Poojasree chunduru MDS[4], Dr. Jyothirmai Nagisetty MDS[5], Dr. Musunuri Manoj Kumar MDS FTMJF RCSEd[6]

[1,4,5]Assistant Professor, Department of Oral & Maxillofacial Surgery, Sibar Institute of Dental Sciences, India.

[2]Associate Professor, Department of Oral and Maxillofacial Surgery, ESIC Dental College, Kalaburagi, India.

[3]Consultant, Department of Periodontics and Implantology, India.

[6]Associate Professor, Department of Oral & Maxillofacial Surgery, Sibar Institute of Dental Sciences, India.

ABSTRACT

Ossifying fibroma (OF) is a benign fibro-osseous neoplasm characterized by the replacement of normal bone with a fibrous stroma containing mineralized material in the form of bone and cementum-like tissue. It commonly affects the jaws, particularly the mandible, and originates from the periodontal ligament, which has the potential to form both osseous and cementum-like structures. Clinically, OF presents as a slow-growing, painless swelling causing facial asymmetry and tooth displacement. Radiographically, it appears as a well-circumscribed unilocular or multilocular radiolucent lesion that gradually becomes mixed radiopaque as calcification progresses. The lesion's well-defined margins help distinguish it from fibrous dysplasia and other fibro-osseous pathologies. Histopathologically, the lesion consists of fibro cellular tissue containing trabeculae of woven or lamellar bone and cementum-like spherules. Based on clinical and histologic characteristics, OF is classified into conventional Cemento-Ossifying Fibroma (COF), Juvenile Trabecular Ossifying Fibroma (JTOF), and Psammomatoid Ossifying Fibroma (POF). Conventional COF is slow-growing and well-encapsulated, whereas the juvenile form exhibits aggressive behaviour with rapid expansion and a higher recurrence rate. Treatment primarily involves surgical excision, and recurrence is uncommon in conventional types but more frequent in juvenile variants. Early diagnosis through clinical, radiographic, and histopathological correlation is essential for effective management. Although prognosis is generally favourable, long-term follow-up is recommended to monitor for recurrence, especially in juvenile cases. Understanding the biological behaviour of each variant aids in selecting appropriate surgical strategies and improving patient outcomes.

Keywords: Ossifying fibroma, Cemento-ossifying fibroma, Juvenile ossifying fibroma, Fibro-osseous lesion, Jaw tumors.

Introduction

Fibro osseous lesions are poorly defined lesions that include fibrous dysplasia, ossifying fibroma & osseous dysplasias that affect the jaws and craniofacial bones [1]. It is characterised by the replacement of normal bone and marrow with a connective tissue matrix and mineralization with woven bone or acellular structures [2,3]. The recent WHO (2022) classification describes ossifying fibromas as not being a single entity; instead, distinct subtypes are recognized, especially according to location, behaviour, and origin. This classification distinguishes between odontogenic and non-odontogenic / craniofacial fibro-osseous lesions. The term "juvenile" has been dropped from the psammomatoid variant, and it is now renamed simply as psammomatoid ossifying fibroma (POF), as it occurs across a wider age range than just children. The cemento-ossifying fibroma (COF) remains the variant considered to have odontogenic origin and is restricted to the tooth-bearing parts of the jaws [3]. Thus, the term OF is used if the predominant component is bone. Cemento-ossifying fibroma refers to lesions that contain both bone and cementum. Peripheral ossifying fibroma is an extraosseous soft tissue lesion that accounts for 9.6% of all the gingival lesions [4]. Ossifying fibroma (OF) is a benign, non-odontogenic fibro-osseous lesion with unknown cause. It seems to emerge from the mesenchymal blast periodontal ligament cells, which consist of fibrous tissue, bone, and cementum-like substance. This unusual tumour may be challenging to diagnose due to its overlapping clinical and histomorphological features [5]. The lesion often appears as a slow-growing, well-defined intra-bony mass in the third to fourth decades of life, with a 5:1 female predisposition. They are typically asymptomatic until they develop large enough to cause facial deformities or restrict function [7]. Later, the lesion may cause additional symptoms, including pain, tooth mobility, and discharge of pus. Radiologically, it appears as radiolucency, mixed radiolucency, opacity, and total radiopacity, depending on the degree of mineralization. Initially, the lesion is well defined due to its encapsulation [7]. Management for OF involves surgical excision and extended resection, depending on the size and location of the lesion [6]. Our review aims to compare and contrast the clinical characteristics, imaging results, and detailed histological features of three recently described subtypes of ossifying fibromas of the maxillofacial skeleton: COF, JTOF, and POF.

Classification

In 1872, OF was first described by Menzel, but was reported by Montgomery in 1927. Numerous classifications have applied the term “OF,” while in the 3rd edition of the WHO classification 2005, the term “cementifying OF” was replaced with OF. However, COF was reclassified as a benign mesenchymal odontogenic tumour in the 2017 WHO classification. This clearly distinguished COF from OFs, which are categorized as benign fibro- and chondro-osseous lesions. Peripheral OF is the extraosseous variety, while intraosseous OF has conventional and juvenile forms. Juvenile OF (JOF) refers to two separate clinicopathological entities: juvenile trabecular OF (JTOF) and juvenile psammomatoid OF(JPOF) [2]. The term 'juvenile' is removed from the psammomatoid variant in the recent 2022 WHO classification. The current classification of ossifying fibroma is illustrated in the following (**Fig. 1)**

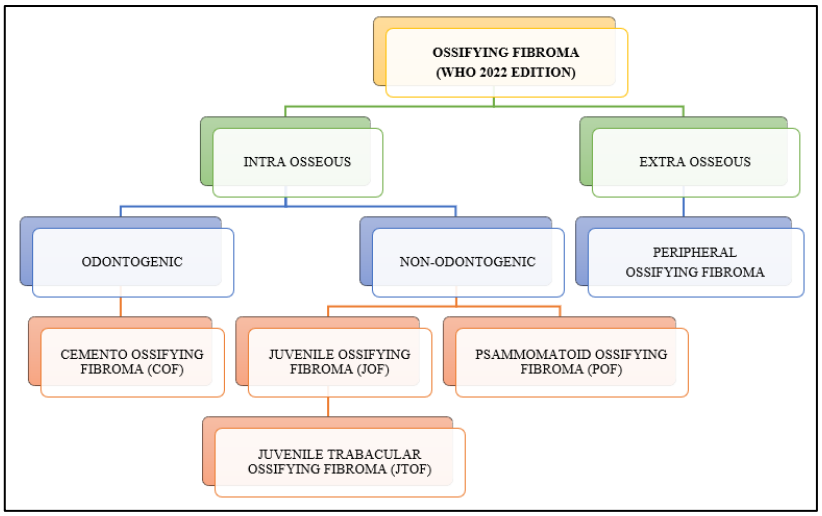

FIG.1 Classification of ossifying fibromas

Etiopathogenesis

The etiopathogenesis of ossifying fibroma is most effectively elucidated as a neoplastic proliferation of multipotential mesenchymal cells, primarily of periodontal ligament origin, that is influenced by genetic, molecular, and potentially hormonal factors (TABLE-1) & (FIG-2) The most widely accepted hypothesis suggests that OF arises from the periodontal ligament, which contains pluripotent cells capable of forming cementum, bone, and fibrous tissue. Costa-Guda et al. (2021) found that in OFs, four showed complete loss of nuclear parafibromin expression and CDC73 mutations, indicating that loss of the parafibromin pathway may play a role in a subset of OFs [23

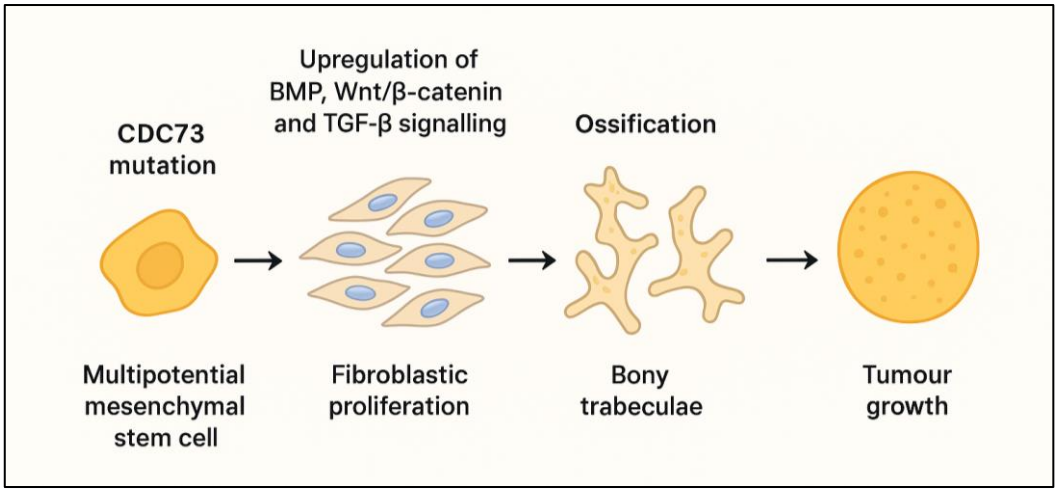

FIG.2 Pathogenesis of tumour growth

- CDC73/HRPT2 mutation and parafibromin loss are the most consistent findings, confirming the neoplastic nature in a subset of OFs.
- Absence of GNAS mutations differentiates OF from fibrous dysplasia.
- BMP and Wnt signalling activation drives aberrant ossification within the lesion.
- Juvenile variants may harbour unique molecular events (e.g., SETD2 mutation)

GENE / PATHWAY	MOLECULAR ALTERATION / FINDING	FUNCTIONAL / PATHOGENETIC ROLE	REFERENCE
CDC73 (HRPT2) [23]	Germline or somatic inactivating mutations; loss of parafibromin expression	Tumour suppressor gene regulating cell cycle via PAF1 complex; loss promotes neoplastic proliferation	Costa-Guda *et al.*, 2021
Parafibromin (protein product of CDC73	Loss of nuclear staining (IHC)	Supports HRPT2/CDC73 inactivation; marker for neoplastic nature	Costa-Guda *et al 2021*
SETD2	Frameshift mutation (novel	Implicates chromatin remodeling and	Kato *et al.*, *Diagnostic Pathology*, 2021

		epigenetic regulation in aggressive variants	
Wnt/β-catenin Pathway	β-catenin nuclear localization observed	Promotes osteogenic differentiation of fibroblasts; linked to aberrant ossification	Pereira *et al.*, *Virchows Archiv*, 2019
BMP-2, BMP-4 (Bone Morphogenetic Proteins)	Overexpressed in OF stroma	Induces osteoblastic differentiation and bone matrix formation	Pereira *et al.*, *Virchows Archiv*, 2019

TABLE 1 Molecular and Genetic factors associated with ossifying fibromas.

Clinical features

Cemento-ossifying fibroma can manifest in both jaws; however, it is more commonly found in the mandible, particularly in females [24]. It can occur in all ages, with the highest incidence in the second & third decades of life. The most commonly reported initial sign of COF is swelling and pain in the affected region, accompanied by gross displacement of the teeth. Asymmetry in the face may result from large, persistent lesions. Juvenile ossifying fibromas (JOFs) occur in an age range of 3-70 years of life. There are two histological variants, trabecular and psammamotiod OF. POF more commonly occurs in extra gnathic areas compared to JTOF, which primarily affects the jaws with a slight predilection to the maxilla than the mandible **(TABLE 2).**

FEATURE	**CEMENTO-OSSIFYING FIBROMA (COF)**	**JUVENILE TRABECULAR OSSIFYING FIBROMA (JTOF)**	**PSAMMOMATOID OSSIFYING FIBROMA (POF)**
NATURE	Benign, slow-growing fibro-osseous neoplasm	Benign but locally aggressive fibro-osseous tumor	Benign, locally aggressive, may extend into paranasal sinuses
AGE GROUPS	3rd–4th decades, occasionally in young adults	8–20 years, peak in the first two decades	5–25 years, often in adolescents and young adults

GENDER	Female > Male	Slight male predominance or equal	No significant gender bias
COMMON SITE	Mandible > Maxilla, especially premolar–molar region	Maxilla > Mandible, often alveolar process	Paranasal sinuses, orbit, ethmoid and frontal bones; occasionally maxilla
ONSET AND GROWTH RATE	Slow, progressive swelling	Rapid growth, sometimes aggressive	Slow to moderate, expansile due to sinus involvement
SYMPTOMS	Painless swelling, facial asymmetry, and tooth displacement	Pain, facial swelling, cortical expansion, sometimes paresthesia	Facial swelling, proptosis, nasal obstruction, sinus symptoms
CORTICAL BONE CHANGES	Expansion with thinning, rarely perforation	Expansion with possible perforation of cortex	Expansion of sinus walls, orbital or nasal displacement
EFFECT ON TEETH	Displacement or root divergence; rarely resorption	Tooth displacement is common and may cause mobility	Displacement of adjacent teeth or sinus floor elevation
CONSISTENCY ON PALPATION	Firm to hard	Firm to hard	Firm to hard
AGGRESSIVENESS	Usually, non-aggressive	Aggressive can recur if incompletely removed	Locally aggressive, especially in confined sinus/orbital spaces
RECURRENCE RATE	Low (0–10%) after complete excision	High (30–50%) if incomplete excision	Moderate (20–30%), especially with incomplete removal
PROGNOSIS	Excellent with complete excision	Good but requires close follow-up	Good, but may recur due to difficult surgical access

TABLE 2 Clinical features of ossifying fibroma subtypes

Radiographic features

Cemento-ossifying fibroma (COF) appears radiographically as a well-defined, expansile lesion that is initially radiolucent but subsequently acquires mixed radiopaque areas as the lesion evolves due to mineralized material deposition. It often appears unilocular or multilocular, with a corticated border, producing cortical expansion, weakening, and displacement of adjacent teeth. Juvenile Trabecular Ossifying Fibroma (JTOF) has a more aggressive radiographic appearance, with ill-defined margins and radiolucent/radiopaque density. The lesion can cause rapid bone expansion, cortical perforation, and invasion of surrounding structures, such as the nasal cavity or orbit. The interior framework shows uneven, patchy regions of calcification that correlate to juvenile trabecular bone. Juvenile Psammomatoid Ossifying Fibroma (JPOF), on the other hand, usually affects the paranasal sinuses, orbit, or fronto-ethmoidal region and appears as a well-defined, mixed-density or purely radiopaque lesion. It often has a lobulated or "ground-glass" look, with small concentric radiopacities that resemble psammoma-like ossicles. Despite its well-defined boundaries, JPOF can cause substantial growth and thinning of cortical plates. The well-defined borders of ossifying fibromas distinguish them from fibrous dysplasia, which usually merges with the surrounding bone **(TABLE 3).**

RADIOGRAPHIC FEATURE	CEMENTO-OSSIFYING FIBROMA (COF)	JUVENILE TRABECULAR OSSIFYING FIBROMA (JTOF)	PSAMMOMATOID OSSIFYING FIBROMA (POF)
Lesion outline/margins	Well-defined, often corticated or encapsulated	Well-demarcated, may appear corticated or scalloped	Sharp, well-defined margins, sometimes lobulated
Border relationship to the surrounding bone	Clearly separated from the surrounding bone by a thin radiolucent line (fibrous capsule)	May show a fine radiolucent halo; distinct separation from adjacent bone	Distinct interface from normal bone; may expand sinus walls

Internal structure	Initially radiolucent, later mixed radiolucent–radiopaque as calcifications increase.	Mixed density lesion with fine trabecular or granular radiopacities ("honeycomb" / "ground-glass")	Heterogeneous mixed density, often with concentric or psammomatoid radiopacities
Calcification pattern	Cementum-like and bone-like radiopacities scattered in a fibrous matrix	Irregular, wispy, trabecular bone-like calcifications	Numerous small psammoma body–like round calcifications

TABLE 3 Radiographic features of ossifying fibroma subtypes

Histopathological features

Cemento-ossifying fibroma (COF) is characterized histologically by a well-circumscribed lesion composed of a moderately cellular fibrous stroma containing varying amounts of mineralized material, including trabeculae of woven bone and rounded basophilic cementum-like calcifications. Osteoblasts often rim the bony trabeculae, and the lesion is usually separated from the surrounding bone by a fibrous capsule, indicating its benign and slow-growing nature [18]. In contrast, Juvenile Trabecular Ossifying Fibroma (JTOF) exhibits a highly cellular fibroblastic stroma with irregular trabeculae of immature osteoid and woven bone resembling fetal bone. Plump osteoblasts typically line the trabeculae, and mitotic figures may be seen, reflecting the lesion's rapid and locally aggressive growth [18,25]. Juvenile Psammomatoid Ossifying Fibroma (JPOF), on the other hand, demonstrates a densely cellular fibrous stroma containing numerous small, round, concentric ossicles resembling psammoma bodies. These "psammomatoid" calcifications are typically eosinophilic with a lamellar pattern, and the lesion is well circumscribed, though potentially locally invasive. While COF tends to occur in the jaws of adults, JTOF and JPOF are observed in younger patients and exhibit more aggressive behaviour, with a higher recurrence potential.

Discussion

Ossifying fibroma is the most common benign fibro-osseous lesion that is characterized by replacement of normal bone with a connective tissue matrix containing mineralized bone or cementum [14]. Various names, including osteofibrous dysplasia, non-osteogenic fibroma, cemento-ossifying fibroma, osteofibroma, fibro-osteoma, and benign fibro-osseous lesion of periodontal ligament origin, have been used to describe this lesion. Menzel first described a variant of ossifying fibroma and called it COF. The term "COF" refers to a range of fibro-osseous lesions caused by the periodontal ligament, including those with just cementum or bone deposition [9]. The term "cemento-ossifying fibroma" has been modified to "ossifying fibroma" (OF) in the 2005 WHO classification. OFs and COFs are primarily odontogenic, originating from the periodontal ligament. In the 2017 edition by the WHO, COF was included in the odontogenic mesenchymal origin. Juvenile OF (JOF) refers to two different entities: juvenile trabecular OF (JTOF) and juvenile psammomatoid OF(JPOF) [2]. In the WHO's new classification, the term juvenile was dropped & renamed as psammomatoid OF (POF). The cemento-ossifying fibroma (COF) remains the variant considered to have odontogenic origin and is restricted to the tooth-bearing parts of the jaws [3]. It can occur in two types: a central variant is from the periodontal ligament cells around the root apex, and a peripheral variant that only affects the soft tissues of the jaw. The central variant is a neoplastic entity, whereas the peripheral variant is a reactive non-neoplastic proliferation of soft tissues [10]. It can include mature collagen, cellular fibroblastic tissue, mineralized tissue, and multinucleated giant cells. Ossifying fibroma is often seen in the third to fourth decade of life, with a male predominance (5:1), with the mandible being the preferred jawbone in 70-90% of instances. Initially, it is painless and may appear as an incidental radiographic finding. As the lesion grows, it can cause pain, bone expansion, jaw deformities & facial disfigurement [14]. It occurs more frequently in the tooth-bearing area of the mandible rather than the maxilla, with the most common site being the mandibular premolar and molar area [11]. Some authors suggest that OF in the maxilla is more aggressive and exhibits more visible symptoms than in the mandible due to its physiological differences. The mandible's robust outer cortex and loose inner marrow act as a barrier to lesion formation and expansion. On the contrary, in the maxilla, it can expand more easily in the thin cortex and inner cancellous bone [12]. In our series, the mean age of the patients was 30.3 years. The age distribution is similar to that in other studies, with a notable difference in gender distribution attributed to demographic factors. In our study, the maxilla is predominantly affected than the mandible in ¾ cases, which is inconsistent with other studies.

On radiograph, in their early stages, OF may be completely radiolucent when it matures, it becomes mixed, and at later stages it becomes more radio-opaque [14]. According to Guanseelan et al, the most common finding was a well-defined radiolucent lesion and jaw bone expansion with or without a sclerotic border, and mixed radiolucent and radiopaque components in later stages [13]. According to Marx and Stern [15], OFs are likely caused by the widespread induction of mesenchymal cells into bone and cementum during odontogenesis. Errors during tissue induction can lead to the development of an OF in the jaws. The neoplastic nature of the OF is attributed to the fact that larger lesions exhibit aggressive behaviour and cause significant bone destruction [16]. "Juvenile" OF is a subtype that affects the craniofacial bones of children under the age of 15 years. As the name suggests, the most striking feature of JOF is its greater incidence in children and young adults. However, their occurrence in the older age group has also been reported. Johnson et al. [20] have reported JOFs occurring at any age, from 3 months to 72 years. The JOFs are classified into two distinct clinicopathologic entities: the trabecular and the psammomatoid types. The presence of trabeculae, fibrillar osteoid, and woven bone defines Trabecular JOF. Psammomatoid JOF is characterized by the presence of small, homogeneous, spherical ossicles that resemble psammoma bodies [17,18]. The predominant anatomical site of lesions differentiates the two types of JOF. JPOF is more common in the fronto-Naso-orbito-ethmoidal area compared to JTOF, which mostly affects the jaws with a slight predilection to the maxilla [8]. Approximately 75% of JPOFs develop in the orbit, paranasal sinuses, and calvaria, with just 25% affecting the jawbone, predominantly the maxilla. JPOF mostly affects the sinonasal and orbital bones of the skull, while JTOF is mostly a gnathic lesion affecting the jaws [19]. The local invasion can range from 'bowing' or 'pushing' of surrounding bony limits to invasion through osseous delimiting walls and extension into adjacent anatomical compartments. According to Chrcanovic BR & Gomez RS et al [8], 42% JTOF and 22% JPOF cases involved the mandible. Many authors have suggested that JPOFs are more commonly reported in adults than JTOFs and exhibit a wider age distribution. In a large systematic review, Charcanovic reported that the mean age of patients with JPOF (18.9 ± 12.0 years) was significantly higher than that of patients with JTOF (11.5 ± 6.0 years). Research suggests that fibroosseous lesions may coexist with other giant cell lesions in the jaws, including ABC and CGCG. This coexistence may be due to a stromal alteration within the initial lesion. Kaplan et al. reported 3 cases of CGCG in OF among 51 intraosseous lesions. They believed that in mixed lesions, the primary lesion is OF, and due to an unknown trigger,

the mesenchymal spindle cells of the tumour release cytokines that induce differentiation toward osteoclast giant cells [21].

Aneurysmal bone cyst (ABC) formation has also been observed in some cases. Initially, they appear as focal myxoid changes in the stroma, followed by haemorrhage and osteoclastic giant cells. These cells gradually expand and form cysts with thin fibrous walls [8,18]. According to Padwa et al., 22% of jaw ABCs reported in the literature have been associated with other bone lesions, such as fibrous dysplasia, ossifying fibroma, or giant cell tumor. Lesions with secondary aneurysmal bone cysts had a higher chance of recurrence than lesions without cysts, and these cysts are more common in JPOF than JTOF [20]. But in our case series, no such secondary ABC lesions were identified in any variants of OFs.

The management mainly depends on both clinical and radiographic findings, comprising one of the following options **(FIG.3).**

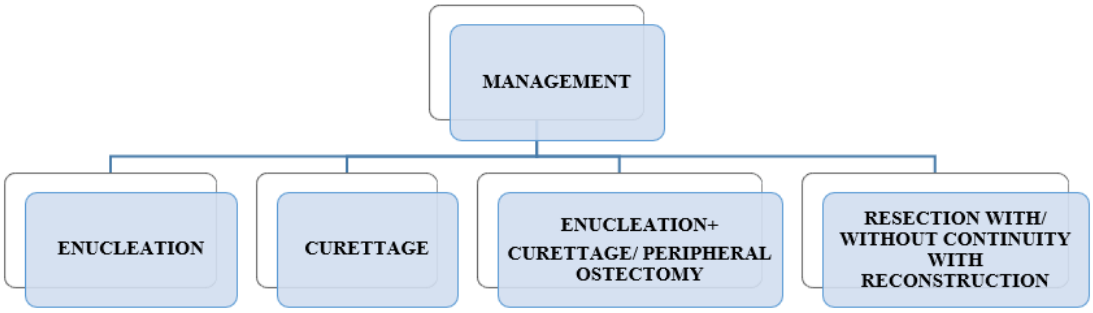

FIG.3 Management strategies of ossifying fibroma

1. Enucleation

2. Curettage

3. Enucleation +curettage/ peripheral ostectomy

4. Resection with/ without continuity with reconstruction

Enucleation is performed when the lesion is well-defined, encapsulated, and has not grown to a significant size. Curettage is performed when the lesion has a soft consistency that merges with the surrounding normal bone during surgery, or when there is no apparent radiolucency [14]. An extensive systematic review indicated that enucleation and curettage had a higher recurrence rate, regardless of the lesion's location or type (JTOF/JPOF). Enucleation with curettage followed by peripheral osteotomy is the preferred therapeutic option to reduce the

recurrence and also to avoid the disfigurement usually associated with surgical resection, in both JOF variants. Moreover, it has been recommended that resection should be considered in cases where there is recurrence, invasion of adjacent bones, or where preservation of the inferior border is not possible [22]. Resection with continuity defect is performed in cases involving the inferior border of the mandible or its proximity to it. In the maxilla, if the tumour extends into the maxillary sinus and nasal cavities, having diffuse/ill-defined margins, it is also subject to resection. Studies have reported that the primary reasons for recurrence are often due to incomplete excision of the lesion, which is frequently caused by the infiltrative nature of the tumor [8,22]. According to Chrcanovic BR et al, recurrence rates of JPOF are Curettage (46.2%), Enucleation (37.5%), Enucleation & curettage/PO (12.5%), & Marginal/Segmental resection (0%). Similarly, for JTOF, the procedures are Curettage (62.5%), Enucleation (45.5%), Enucleation & curettage/PO (33.3%), Marginal resection (10%) & Segmental resection (0%) [8].

Conclusion

Ossifying fibroma is a distinct, benign fibro-osseous neoplasm of the jaws characterized by well-demarcated growth, progressive expansion, and a variable histopathological spectrum. Although often asymptomatic in the early stages, its potential for significant deformity, tooth displacement, and recurrence in specific aggressive variants underscores the importance of early diagnosis and appropriate management. Compared to other odontogenic lesions, these are easier to treat since they are usually well defined and rarely penetrate the surrounding bone. Curettage and enucleation alone did not effectively eliminate JOF lesions, leading to high recurrence rates. Enucleation followed by peripheral osteotomy/curettage is the preferred treatment for both JOF variants to avoid the disfigurement associated with surgical resection, although surgical resection has a low recurrence rate.

References

1) MacDonald DS. Classification and nomenclature of fibro-osseous lesions. Oral Surgery, Oral Medicine, Oral Pathology and Oral Radiology. 2021 Apr 1;131(4):385-9.
2) Okwuosa CU, Nwoga MC, Adisa AO. Ossifying fibroma of the jaws: Review of 57 cases in Enugu and of global literature Niger J Med 2022;31:535-9.
3) Haefliger S, Baumhoer D. Die neue WHO-Klassifikation der Kiefertumoren [The new WHO classification of jaw tumours]. Pathologie (Heidelb). 2023 Jul;44(4):240-249. German. doi: 10.1007/s00292-023-01195-4. Epub 2023 May 13. PMID: 37179260; PMCID: PMC10322770.

4) Verma E, Chakki AB, Nagaral SC, Ganji KK. Peripheral cemento-ossifying fibroma: case series literature review. Case Rep Dent. 2013;2013:930870. doi: 10.1155/2013/930870. Epub 2013 Jan 14. Erratum in: Case Rep Dent. 2013;2013:827247. PMID: 23365762; PMCID: PMC3556846.
5) Gondivkar SM, Gadbail AR, Chole R, Parikh RV, Balsaraf S. Ossifying fibroma of the jaws: report of two cases and literature review. Oral oncology. 2011 Sep 1;47(9):804-9.
6) Kolomvos N, Nadia Theologie-lygidakis N, Christopoulos P, Iatrou I (2013) Benign fibro-osseous lesions of the jaws in children: a 12 year retrospective study. J Craniomaxillofac Surg.
7) Godt A, Gulicher D, Kalwitzki M, Krober SM (2008) Dislocation of an upper third molar by an ossifying fibroma—Case report. J Craniomaxillofac Surg 36(6):360–364
8) Chrcanovic BR, Gomez RS. Juvenile ossifying fibroma of the jaws and paranasal sinuses: a systematic review of the cases reported in the literature, Int J Oral Maxillofac Surg (2019).
9) Kuta AJ, Worley CM, Kaugars GE. Central Cemento-ossifying fibroma of the Maxillary sinus: A review of six cases. *AJNR Am J Neuroradiol* 1995; 16: 1282-1286
10) Bala TK, Soni S, Dayal P, Ghosh I. Cemento-ossifying fibroma of the mandible: A clinicopathological report. Saudi medical journal. 2017 May;38(5):541.
11) Miyashita H, Suzuki H, Matsui K, Sato N, Kitamura J, Kumamoto H, Takahashi T. Pediatric cemento-ossifying fibroma of the anterior mandible: A case report. Journal of Oral and Maxillofacial Surgery, Medicine, and Pathology. 2020 Jul 1;32(4):285-90.
12) Cheng XB, Li YP, Lei DL, Li XD, Tian L. Surgical resection of a huge cemento-ossifying fibroma in skull base by intraoralapproach. J Craniofac Surg 2011;22:594–7.
13) Gunaseelan R, Anantanarayanan P, Ravindramohan E, Ranganathan K. Large cemento-m ossifying fibroma of the maxilla causing proptosis: a case report. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;104:21–5.
14) Mohanty S, Gupta S, Kumar P, Sriram K, Gulati U. Retrospective analysis of ossifying fibroma of jaw bones over a period of 10 years with literature review. Journal of maxillofacial and oral surgery. 2014 Dec;13(4):560-7.
15) Marx RE, Stern D. Oral and maxillofacial pathology. A rationale for diagnosis and treatment. Chicago: Quintessence; 2002. p. 789-95.
16) Triantafillidou K, Venetis G, Karakinaris G, Iordanidis F. Ossifying fibroma of the jaws: a clinical study of 14 cases and review of the literature. Oral surgery, oral medicine, oral pathology, and oral radiology. 2012 Aug 1;114(2):193-9.
17) Slootweg PJ, Panders AK, Koopmans R, Nikkels PG. Juvenile ossifying fibroma. An analysis of 33 cases with emphasis on histological aspects. J Oral Pathol Med 1994;23:385-8.
18) ElMofly S. Psammomatoid and trabecular juvenile ossifying fibroma of the craniofacial skeleton: two distinct clinicopathologic entities. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002;93:296-304.
19) Malathi N, Radhika T, Thamizhchelvan H, Ravindran C, Ramkumar S, Giri GV, Gopal D. Psammomatoid juvenile ossifying fibroma of the jaws. Journal of Oral and Maxillofacial Pathology. 2011 Sep 1;15(3):326-9.
20) Johnson LC, Yousefi M, Vinh TN, Heffner DK, Hyams VJ, Hartman KS. Juvenile active ossifying fibroma. Its nature, dynamics and origin. Acta Otolaryngol Suppl 1991;488:1– 40.

21) Kaplan I, Manor I, Yahalom R, et al. Central giant cell granuloma associated with central ossifying fibroma of the jaws: a clinicopathologic study. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;103:e35-41.
22) Sarode SC, Sarode GS, Waknis P, Patil A, Jashika M. Juvenile psammomatoid ossifying fibroma: a review. Oral Oncol 2011;47:1110–6.
23) Costa-Guda J, Pandya C, Strahl M, Taik P, Sebra R, Chen R, Uzilov AV, Arnold A. Parafibromin Abnormalities in Ossifying Fibroma. J Endocr Soc. 2021 May 8;5(7):bvab087. doi: 10.1210/jendso/bvab087. PMID: 34159287; PMCID: PMC8212678.
24) Sopta J, Dražić R, Tulić G, Mijucić V, Tepavčević Z. Cemento-ossifying fibroma of jaws—correlation of clinical and pathological findings. Clinical oral investigations. 2011 Apr;15(2):201-7.
25) Eversole LR, Leider AS, Nelson K. *Cemento-ossifying fibroma: A clinicopathologic study of sixty-four cases.* Oral Surg Oral Med Oral Pathol. 1985;60(5):505–511