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Review Article **Oral Pathology**

A Review of Clinicopathological Variants of Fibro Osseous Lesions of Jaw

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Abstract

Fibro osseous lesions are poorly defined group of lesions, characterized by replacement of normal bone by a benign connective tissue matrix. Fibro osseous lesions of jaws such as Fibrous Dysplasia, Osseous Dysplasia and Ossifying Fibroma. Theses lesions have common characteristics that include common clinical, radiographic and microscopic features. Clinicians and pathologist experience difficulty in diagnosis and differentiation due to its significant overlapping of clinical and histological features. Many diagnostic terms have been used for these lesions in the literature. Therefore, proper categorization requires good correlation of the history, clinical findings, radiographic characteristics, operative findings and histologic appearance. Theses lesions have undergone frequent renaming and reclassification due to its varied features. This revies article is an attempt to simplify the understanding of this diverse group of lesions.

Keywords: Fibro osseous lesions, confusing, diagnostic challenges, fibrous dysplasia, osseous dysplasia and ossifying fibroma, key features of clinicopathological variants of fibro osseous lesions.

INTRODUCTION

Fibro-Osseous lesions [FOL] are group of lesions that affects the jaws and the craniofacial bones and are considered as confusing area in diagnostic pathology [1]. It comprises of diverse, interesting, and challenging group of conditions that pose difficulties in classification and treatment [2]. The term fibro-osseous lesion is a generic designation referred to a group of lesions which are characterized by the replacement of bone by a benign connective tissue matrix [3]. A bewildering number of diagnostic terms have been used for these group of lesions in the literature. In order to properly categorize this group of lesions, it requires good correlation of the history, clinical, radiographic and operative findings, and histologic appearances [2]. Clinically, FOL may have cosmetic and functional disturbances or they may be completely asymptomatic localized lesions that are usually identified on routine radiograph. Radiographically, fibro-osseous lesions may manifest as solitary, multifocal, multiquadrant disease, they may be ill or well defined; they may have radiolucent, mixed radiolucent radiopaque, predominantly radiopaque, or ground glass appearance and they may or may not be associated with the root apex [4]. Histologically, FOLs is characterized by replacement of normal bone by a tissue composed of collagen fibers and fibroblasts that contain varying amounts of mineralized substance, which may be bony or cementum-like in appearance or it may contain admixture of these calcifications [2]. Classification and diagnosis of these group lesions is difficult because there is overlap of clinical and histological features [5]. Fibro-Osseous Lesions of the jaw have undergone frequent reclassification and renaming due to its varied features [1].

This article is an attempt to throw a light on this diverse group and simplify the understanding of this lesions.

Classification

The different classifications systems proposed by authors are enumerated as below.

- Classification of the Fibro-Osseous Lesions of jaws by Charles Waldron (1985) [2].

- Mico M. Malek (1987) Working Classification of Fibro-Osseous Lesions [6, 1].
- Classification of FOLs by Peiter J. Slootweg and Hellmuth Muller (1990) [7, 1].
- WHO Classification of FOLs of jaws (1992) [8, 1].
- Modified Classification of Fibro-Osseous Lesions of Jaws by Charles Waldron (1993) [9, 1].
- Classification of Fibro-Osseous Lesions of Jaws by Brannon and Fowler (2001) [10, 1].
- WHO Classification of Fibro-Osseous Lesions of Jaws (2005) [8, 1].
- Classification of Fibro-Osseous Lesions of Jaws by Paul M. Speight and Roman Carlos (2006) [5].
- Classification of Fibro-Osseous Lesions of Jaws by Eversole (2008) [11, 1].

In 2006, Paul M. Speight and Roman Carlos proposed a classification. The purpose of this classification is the concept of a spectrum of clinicopathological entities in which the diagnosis can only be made on the basis of a full consideration of clinical, histological and radiological feature [5].

Classification of fibro osseous lesions of maxillofacial region by Paul M. Speight and Roman Carlos (2006).
Fibrous Dysplasia. Monostotic fibrous dysplasia Polystotic fibrous dysplasia Craniofacial fibrous dysplasia
Osseous Dysplasia Periapical osseous dysplasia Focal osseous dysplasia Florid osseous dysplasia Familial Gigantiform cementoma
Ossifying Fibroma Convential ossifying fibroma Juvenile trabecular ossifying fibroma Juvenial psammomatoid ossifying fibroma

Fibrous Dysplasia

Fibrous dysplasia (FD) is a benign osseous disease which is characterised by the replacement of normal bone by excessive proliferation of cellular fibrous connective tissue which is slowly replaced by bone, osteoid or cementum like material [12]. Originally introduced by Lichtenstein in 1938 and by Lichtenstein and Jaffe in 1942. It can present in one bone (monostotic form) or multiple bones (polyostotic form) and can be associated with other conditions [13]. Fibrous dysplasia occurs in a monostotic form (70%) and in polyostotic form (30%) of cases [14]. It is usually caused by a mutation in the GNAS1 gene (20q13.2) (guanine nucleotide binding protein, stimulating activity polypeptide) [15]. The etiology has been associated with a mutation in the Gsα gene that occurs after fertilization in somatic cells and is located at chromosome 20q13.2-13.3 [13].

Monostotic fibrous dysplasia

Is more frequent, with approximately equal frequency in males and females in their first or second decades of life. It is insidious in onset and manifests clinically as a slow growing, painless expansion of the involved bone. It commonly affects rib (24%), femur (17%), tibia (13%), mandible (12%), and maxilla (12%). Maxillary involvement, particularly posterior maxilla is more common than mandibular. Cases of FD which affects the maxilla or facial bones and give the patient a leonine appearance is termed as “leontiasis ossea” [4].

Polystotic fibrous dysplasia

Is 6 times less frequent than the monostotic type [16]. It involves two or more bones. Mostly diagnosed before 10 years of age, and there is a female predilection. Involvement of skull and jaw may result in facial asymmetry. It may be association with the following syndromes.

- Jaffe- Lichtenstein syndrome- characterized by café au lait (coffee with milk) pigmentations and polystotic fibrous dysplasia
- McCune-Albright’s syndrome-characterized by café au lait pigmentation and multiple endocrinopathies and polystotic fibrous dysplasia.
- Mazabraud syndrome- characterized by intramuscular myxomas and fibrous dysplasia [17].

Craniofacial FD

The term “craniofacial fibrous dysplasia” (CFD) describes fibrous dysplasia where the lesions are confined to contiguous bones of the craniofacial skeleton. Craniofacial fibrous dysplasia cannot be truly categorized as monostotic because of the involvement of multiple adjacent bones of the craniofacial skeleton and neither it can be truly categorized as polyostotic as bones outside the craniofacial complex are spared. It has a slight female predilection. Detected in first 3 decades of life and usually stabilize when the patient reaches skeletal maturity [18]. The main presentation of Craniofacial FD is a diffuse swelling of affected region, it affects the calvaria, the skull base, the zygoma, the maxilla and the mandible. Maxillary involvement is more often than mandible. It may cause aesthetic

impairment and deformities with clinical symptoms such as visual disturbances, proptosis, orbital dystopia, dental problems and sensory disturbances in the affected regions [19].

FD has three different radiographic patterns which is cystic (radiolucent or lytic), sclerotic, and mixed (radiolucent/radiopaque). It presents with asymmetric homogeneous "ground-glass" appearances that blend into normal bone, thin cortices, and bone expansion are the main characteristics of FD [20]. Malformation of proximal femur known as coxa vera, shepherd's crook deformity in polyostotic FD [17].

Histological examination of FD shows bony trabeculae which are immature, delicate, curvilinear and has variable degree of mineralization with classic "Chinese character" pattern with minimal or no osteoblastic rimming within a vascularized fibrous stroma of variable cellularity. Initially osteogenesis may be evident by the presence of thin anastomosing woven bone trabeculae rimmed by osteoblasts. The stroma is hypercellular and active and lacks pleomorphism. Later, woven bone is replaced by lamellar bone and resting and reversal lines may result from the extensive remodelling (Figure 1- a,b,c) [21].

Central ossifying fibroma has a radiographic and histological appearance similar to that of fibrous dysplasia. Central ossifying fibroma on radiographic examination shows well-defined margin, whereas the margins of fibrous dysplasia tend to merge with the surrounding normal bone. Fibrous dysplasia is mostly discovered in the second decade whereas Central ossifying fibroma occurs mostly in the third and fourth decades of life. Fibrous dysplasia may be confused with Paget's disease of bone. The main differentiating characteristic of Paget's disease from fibrous dysplasia is that the former tends to occur bilaterally in the jaws, whereas the latter affects only one side and histologically, Paget's disease is characterized by presence of many osseous trabeculae with prominent reversal lines showing simultaneous osteoblastic and osteoclastic activity [22].

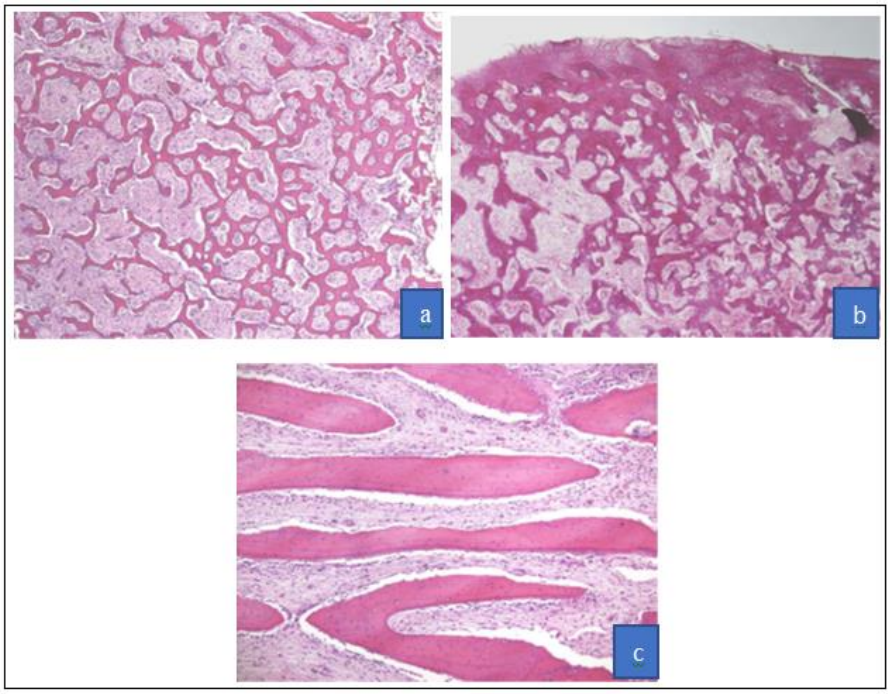

Figure 1: a-Fibrous dysplasia, fine branching trabeculae of woven bone "Chinese character." b- bone blends imperceptibly with the overlying cortical bone. c -A mature lesion lesion Lamellar bone is arranged in parallel arrays. The stroma is typically moderately cellular with sparse collagen production [5]

Osseous Dysplasia

Osseous dysplasia is a reactional and non-neoplastic process that developes in periapical tooth area and characterized by normal bone replacement by fibrous tissue and metaplastic bone. These pathological changes can assume several clinical forms and therefore receive different denominations [23].

It can be further divided into three groups: periapical, focal and florid osseous dysplasia, on the basis of its clinical and radiographic features [24].

Periapical osseous dysplasia are probably the most common fibro-osseous lesions found in clinical practice. Its pathogenesis is not known, although it can represent a reactive or dysplastic process. The key points in diagnosis of this disease, according to Brannon & Fowler are:

- Predilection for mid-age Black women
- One or more circumscribed lesions (approximately 0.5 cm or shorter) at the periapical area of vital teeth.
- It is Painless non-expansive lesion located usually at mandible's anterior area.
- Radiographically it presents as radiolucency of mixed density (radiolucent with opacities), or opaque with a narrow radiolucent margin.
- Cellular fibrous stroma with lamellar osseous tissue and/or oval calcification [23].

The diagnosis is mostly made with a routine radiographic examination. Periapical osseous dysplasia is described as progressing through three radiographically distinct stages (Sapp *et al*., 2002)

(i) Osteolytic stage: is characterized by well-defined radiolucencies at the apex of one or more teeth. The radiolucencies that surrounds the root apex are usually indistinguishable from inflammatory periapical lesions of pulpal origin.

(ii) Cementoblastic stage is characterized by radiolucencies with nodular radiopaque deposits.

(iii) Mature stage characteristic feature is a well-defined, dense radiopacities mostly surrounded by a radiolucent rim, periodontal ligament separates the lesion from the root [25].

Florid Osseous Dysplasia is a rare condition. It characteristically affects the jaws of middle aged women. Melrose *et al*., in 1976 was the first to described Florid osseous dysplasia. This condition has been considered as a developmental anomaly or dysplastic lesion arising in tooth-bearing areas [26].

It has a tendency towards bilateral, often quite symmetrical location, and it is quiet usual to find extensive lesions in all 4 posterior (molar-premolar region) quadrants of the jaws. Clinically, these lesions are mostly asymptomatic and may present as incidental radiological findings. Symptoms like dull pain/drainage are usually associated to exposure of the sclerotic calcified masses in the oral cavity. This may be because of alveolar atrophy under a denture or after extraction of teeth in the affected area involved. On radiographic examination it shows lobulated, dense masses, often symmetrically located in various regions of the jaws [27]. According to the review literature in 2006 only five patients from India have been reported, which makes the occurrence of Florid osseous dysplasia a relatively rare phenomenon [26].

Focal Osseous Dysplasia: Summerlin and Tomich were the first to suggest this lesion, primarily based on the location of dysplastic area of the bone (i.e in the tooth bearing area of posterior jaw or at the site of extraction). The dysplasitic lesions were identified as periapical osseous dysplasia or Focal osseous dysplasia on the basis of their location only (anterior or posterior) because two type of lesion share same clinical radiographic and histological features. The lesion is termed as "focal osseous dysplasia" only when it is not associated with tooth apex [28]. Focal osseous dysplasia is predominantly noted in African-American females, with a peak incidence in the fourth and fifth decades [29].

Radiographically Focal Osseous Dysplasia has three specific features:

- **Osteolytic stage**: shows well defined radiolucent area with loss of periodontal ligament and lamina dura.
- **Cementoblastic stage**: small opacitiesis noted in the radiolucent are, which display both radiolucent and radiopaque architecture. This is due to the deposition of cementum like droplets in the fibrous tissue. At this stage, the lesion can be misdiagnosed histopathologically as cemento ossifying fibroma.
- **Osteosclerotic and inactive stage** presents with definite radiopacity in major part of lesion [28].

Histologically all the three patterns of osseous dysplasia show similar histopathological features, such as cellular fibrovascular connective tissue with scattered hemorrhage and a variable mixture of woven bone, lamellar bone and cementum like particals. As the lesion matures, the ratio of fibrous connective tissue to mineralized material is reduced. Over time, the bony trabeculae become thick and curvellinear, with shapes similar to ginger roots. In the final radiopaque stage, fusion of individual trabeculae takes place to form sheetlike or globular masses of sclerotic, disorganized cemento osseous material (Figure 2- a & b) [30].

It is difficult to distinguish Focal Osseous Dysplasia and ossified fibroma on the basis of clinical and histopathological features. Ossified fibromas is well-demarcated which has radiolucent feature with small radio opaque calcifications. However, Focal osseous dysplasia is usually radiopaque. Unlike Focal osseous dysplasia, Ossified fibromas can be excised as one segment or easily separated from surrounding tissues because of its well-demarcated border [29].

Familial gigantiform cementoma (FGC) is a rare and distinct subtype of Osseous dysplasia. It is regarded as an odontogenic lesion that shares a same periodontal ligament origin with focal, periapical, and florid Osseous Dysplasia. FGC is histologically similar with the other three osseous dysplasia but it is still a quiet distinct clinical entity. The term "familial" in FGC is warranted because of its autosomal dominant transmission feature. It has a tendency towards more exuberant growth and multi quadrant jaw involvement [31].

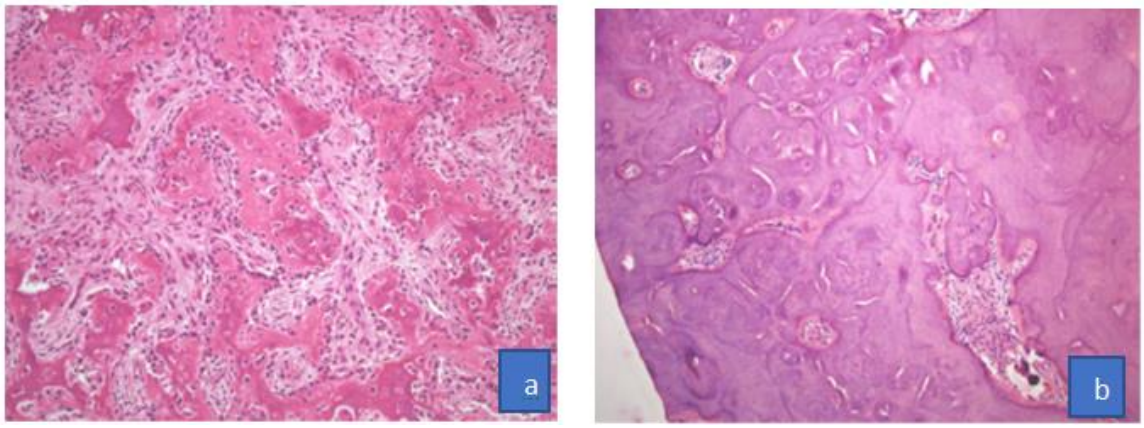

Figure 2: a-Osseous dysplasia. a- Cellular fibrous tissue contains irregular trabeculae of woven bone. Osteoblasts are prominent. b-the calcified material forms 'cementum-like' spherules that fuse to form large globular masses [5]

Ossifying Fibroma

Ossifying fibromas (OF) of the craniofacial skeleton, as described in WHO classification of odontogenic tumors (Barnes L 2005), are benign fibro osseous neoplasms which is characterized by the replacement of normal bone by a fibrous cellular stroma consisting foci of mineralized bone trabeculae and cementum-like material that vary in amount and appearance [32].

The widely used term cement ossifying fibroma was based upon the fact that most lesions may be associated with the teeth or may contain cementum-like spherical calcifications. But lesions that are not associated with the jaws, such as in the sinonasal regions mostly contain these types of calcifications and it is now considered that cementum and bone are essentially the same tissue. The term ossifying fibroma is now considered as more appropriate [5].

OF is further divided into:

- Conventional
- Juvenile (JOF)

Histologically both fibrous tissue and bony material may vary in wide range. Conventional OF may present with Woven bone and/or cementoid and /or lamellar bone and the stroma can vary from being highly cellular to prominently vascular. Juvenile ossifying fibroma has two well defined subtypes based on the histological findings.

- Trabecular (JTOF) and
- Psammomatoid (JPOF)

JPOF is characterized by "psammomatoid bodies" which is spherical calcified ossicles containing osteocytes, with basophilic centre distributed in dense cellular fibrous stroma. The JTOF forms immature irregular trabeculae with osteoidseams [33].

Cemento Ossifying Fibroma (COF)

The term "Cemento-ossifying fibroma" is referred to all the fibro-osseous lesions that were before classified as ossifying and/or cementifying fibromas as they fall within the spectrum of the same disease entity. Waldron and Giansanti suggested that the presence and ratio of the osteoid and cementum reflected this spectrum of the disease. Cementum-like substances of these lesions have also been found in other facial bones such as the maxillary antrum, sphenoid, orbitofrontal and temporal bone away from the tooth-bearing mandible. Eversole *et al*., suggested that the production of these cementum-like structures may be associated with membranous bone and may not only be related to cementogenesis [34]. In new WHO classification (2005), "cemento-ossifying fibroma" this terminology has been reduced to "ossifying fibroma" (OF) [35].

Menzel in 1872 gave the description of COF as a variant of Ossifying fibroma. Etiology of COF is unknown. Bernier believed that COF in the bone may be caused due to irritant stimulus (such as tooth extraction) which may activate the production of new tissue from the remaining periodontal membrane. Periodontal membrane posses multipotential cells which are capable of forming cementum, lamellar bone and fibrous tissue. It is reported by Cakir and Karadayi that nasopharyngeal COF originating from embryologic nest.

COF usually occurs in young and middle aged adults. There is a marked predelection, for female, with the female:male ratio of 2:1. Premolar and molar region of mandible is more commonly involved than maxilla. The lesion is usually asymptomatic until the growth produces a noticeable swelling and mild deformity. Displacement of teeth can be noted as an early clinical feature.

Radiographically, it is shows three stages: initial or early, mixed and mature stage. In early stage, it appears as a well-defined radiolucent lesion with no evidence of internal radiopacities. On maturation of the lesion, evidence of calcification is noted and the radiolucent area becomes flecked with opacities until ultimately the lesion appears as an extremely

radiopaque mass in the mature stage. The significant diagnostic point is that COF has centrifugal growth pattern therefore, the lesions grow by expansion equally in all directions and present as a round tumor mass. It has a well defined borders with a thin radiolucent line suggesting a fibrous capsule that separates the lesion from the surrounding bone [36].

Histopathologically cemneto- ossifying fibroma is composed of delicate interlacing collagen fibers, infrequently arranged in discrete bundles, interspersed with large numbers of active, proliferating fibroblasts and cementoblasts sometimes osteoblasts may be seen. Few mitotic figures may be present. On the maturation of lesion, the islands of cementum increase in number, enlarge and ultimately conjoin (Figure 3) [37].

COF is well circumscribed from bone, it should be excised conservatively, but complete resection of the lesion is necessary. As COF is less vascularized and well circumscribed, it is easy to remove from the surrounding bone. The prognosis is known to be fair and recurrence after surgical removal seems to be unusual [36].

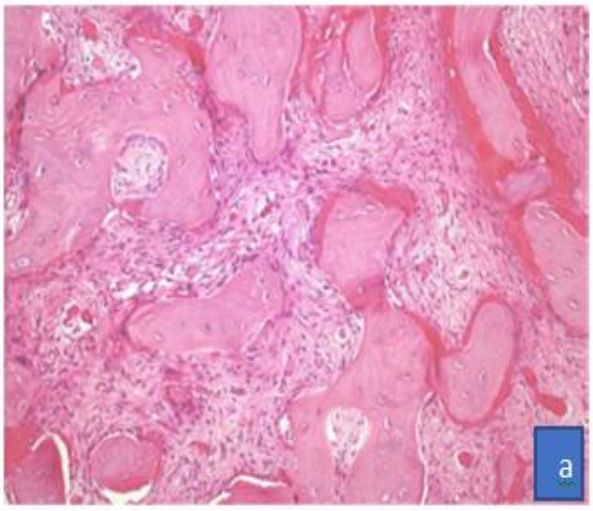

Figure 3: Irregular trabeculae of bone in a cellular fibrous stroma. In this image, few osteoblasts are visible and there is some evidence of lamellar bone formation [5]

Juvenile ossifying fibroma

Juvenile ossifying fibroma was first described as "osteoid fibroma with atypical calcification" by Benjamin in 1938 (Khoury *et al*., 2002). In 1952, Johnson suggested the term "Juvenile active ossifying fibroma" (Neville *et al*., 2002) [38]. The term aggressive is appropriate for most cases of juvenile OF because of their recurrence rate ranging from 30% to 56% and its rapid growth [39].

According to Johnson *et al*., JPOF originates due to overproduction of the myxofibrous cellular stroma that is normally involved in the growth of the septa in the paranasal sinuses as they enlarge and pneumatize. Sawyer *et al*., suggested the presence of non random chromosome break points at Xq26 and 2q33 resulting in (X; 2) translocation. Mutations of HRPT2 gene in Ossifying Fibroma was identified by Pimenta *et al.,* and postulated that it may arise due to haploinsufficiency of the HRPT2 gene. But this genetic alteration was absent in JOF indicating that it may be distinct clinico-pathological entity and additional studies are needed to confirm this assumption [40].

Juvenils pasmmamatoid ossifying fibroma (JPOF)

Benjamins in 1938 first reported JPOF, who called the lesion "osteoid fibroma with atypical ossification of the frontal sinus." JPOF has a predilection for the sinonasal tract and the orbit particularly periorbital, frontal, and ethmoid bones. In JTOF the average age of occurrence is 8 1/2–12 year, only 20% of the patients are over 15 years of age whereas that of JPOF is 16–33 years although some reports suggest patients age ranged from 3 months to 72 years. The lesion has a potential to proliferate, invade and destroy tissues. Apart from its aggressive behaviour this lesion also has a strong tendency to recur and recurrence rate as high as 30–56% have been reported. Gender predilection is quiet controversial with some authors claiming predilection for either sex whereas Johnson *et al*., found higher incidence in females while El Mofty reported a male predilection. The main and important differentiating feature of JTOF from JPOF, is the site of involvement, with JPOF occur mainly in the paranasal sinuses and JTOF occur mainly in maxilla. Radiographically, it is a round, well-defined, sometimes corticated osteolytic lesion with a cystic appearance. Sclerotic changes may show a ground-glass appearance. Histopathologically, characterized by presence of eosinophilic spherical structures dispersed in a fibrous stroma consisting of uniform, stellate, and spindle shaped cells that are arranged as strands and whorls. Golg was the first to term this spherical structure as Psammoma-Like bodies, they have central basophilic area and peripheral eosinophilic fringe (Figure 4) [41].

Juvenile trabecular Ossifying Fibroma

Is usually asymptomatic and early lesions are usually discovered as incidental radiographic findings. Tooth displacement may be an early sign. No gender predilection noted for either entity. Radiographically, it is mostly unilateral, unilocular mixed radiolucent/radiopaque lesions but may present as completely radiolucent lesions with faint internal radiopacities. It expands concentrically from a central point, in outward direction which may result in displacement of teeth and the inferior alveolar nerve canal. It progresses very rapidly mimicing malignancy. Histologically, it is composed of cellular fibroblastic tissue with thin trabeculae of immature bone. This immature bone anastomose to form a lattice. It is well demarcated but unencapsulated. Plump osteoblastic rimming of bone is quiet common feature. JPOF is

different from JTOF because of absence of the thin trabeculae of immature bone as seen in JTOF. If left untreated JTOF continues to enlarge and therefore excision of early lesions is mandated to prevent further expansion. The prognosis of JTOF varies and recurrence after the removal is not uncommon [42].

The first line of management of JOF to minimize morbidity is curettage with peripheral ostectomy and resection should be reserved for very extensive and recurrent neoplasms [43].

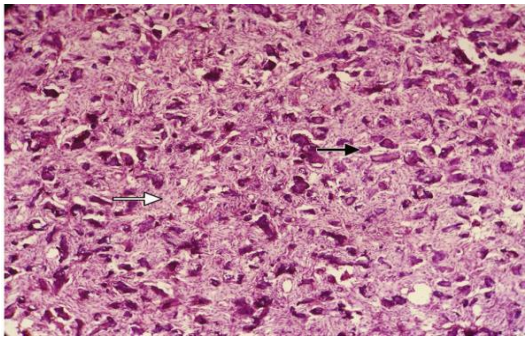

Figure 4: Numerous homogeneously distributed psammomatoid ossicles (black arrow head) with a smooth contour and a thin fringe of collagen fibers set in cellular fibroblastic stroma (white arrow head) [40]

Table 1: Key features of cinicopathological variants of fibrous dysplasia

Lesion	Monostotic	Polyostotic	Craniofacial
Age	2nd and 3rd decade of life.	before 10 years of life	First 3 decades of life
Gender	Male and female equal predeliction.	Female prediction	Female
Site involved	-Rib, femur, tibia craniofacial bone and humerus in decreasing order of frequency. -Maxillary involvement is more frequent than mandibular	Femurs, tibia, pelvis, ribs, skull and facial bone, upper extremities, lumbar spine in decreasing order.	Contiguous bones of craniofacial skeleton Maxilla more commonly involved than mandible
Associated syndromes	Not associated with Endocraniopathies	Albright syndrome Mazabraud's syndroem. Lichtenstein-jaffe (caffe-au lait spot)	May be associate with endocraniopathies.
Radiographic features	Characteristic ground glass Appearance that blends into normal bone.	Malformation of proximal femur known as coxa vera, shepherd's crook deformity. Involvement of skull leads to deformity.	Similar to monostotic and polystotic fibrous dysplasia
Histological features	-Fibrous lesion with proliferating fibroblast. Irregular trabeculae "Chinese character shaped." pattern. - Replacement of woven bone by lamellar bone.	no distinguishing histological features	No distinguishing histological features.

Table 2: Key features of clinicopathological variants of Osseous dysplasia

Disease	Periapical	Florid	Focal
Frequency	Most common	Rare	Less common than periapical
Age	Middle age	Middle age	4th and 5th decade
Gender	Black women	Female	African-American Female
Site	Mandibular anterior Periapical area of vital tooth.	Bilateral, symmetrical, can involve all 4 posterior (molar-premolar region) quadrants of the jaws.	tooth bearing area of posterior jaw or at the site of extraction periapical
Radiographic feature	-Osteolytic phase-well-defined radiolucencies at the apex -Cementoblastic phase- well-defined radiolucencies at the apex. -Mature phase- dense radiopacities usually surrounded by a radiolucent rim.	appear as dense, lobulated masses, often symmetrically	Radiographic features similar to osseous dysplasia. Not associated with tooth apex
Histological feature	Cellular fibrovascular connective tissue with variable mixture of woven bone, lamellar bone and cementum like particals. trabeculae become thick and curvellinear, individual trabeculae fuse to form sheetlike or globular masses of sclerotic, disorganized cemento osseous material	Similar histological features	Similar histological features

Table 3: Key features of clinicopathological variants of ossifying fibroma

Lesion	Ossifying Fibroma	Juvenils pasmmamatoid ossifying fibroma	JTOF:
Age	Middle age	16–33 years	8 1/2–12 year
Gender	Female: male 2:1	no gender predilection	no gender predilection
Site	Premolar and molar region of mandible	Paranasal sinus	Maxilla
Radiological feature	Initial stage: well-defined radiolucencie Later: in mature stage radiopaque mass. It has centrifugal growth pattern. It has well defined borders with a thin radiolucent line suggesting a fibrous capsule	-round, well defined corticated osteolytic lesion. - Sclerotic changes may show a ground-glass appearance.	unilateral, unilocular mixed radiolucent/radiopaque lesions
Histological feature	Cellular fibrous stroma, irregular bone trabeculae. Matures lesion show islands of cementum increase in number, enlarge, and ultimately coalesce.	Psammoma-Like bodies dispersed in a fibrous stroma consisting of uniform, stellate, and spindle shaped cells that are arranged as strands and whorls. These particles show central basophilic area and a peripheral eosinophilic fringe	Cellular fibroblastic tissue with thin trabeculae of immature bone. This immature bone may anastomose to form a lattice. Plump osteoblastic rimming of bone

CONCLUSION

There has been significant progress in recent years in understanding the histogenetic and pathogenetic similarities and differences of the various fibro osseous lesions, thereby enhancing one's ability to differentiate and diagnose accurately. But there is still a need for clarification of many aspects of this perplexing group of lesions [44].

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