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Review Article

Benign Fibro-Osseous Lesions of the Jaws: Review of Literature

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Abstract

Fibro-osseous lesions of the jaws comprise a diverse group of conditions, which are characterized by replacement of normal bone by a benign connective tissue stroma in which the normal architecture of bone is replaced by fibrous tissue containing varying amount of foci of mineralization. Adequate clinical and para clinical observations, imaging characteristics, and histologic findings are necessary to arrive at a confirmatory diagnosis. Guanine nucleotide-binding protein/alpha subunit (GNAS) [1,2] mutations are diagnostic to fibrous dysplasia whereas in ossifying fibroma, mutations are seen in HRPT2 gene that encodes parafibromin protein. The current classification systems include neoplasms, developmental dysplastic lesions and inflammatory/reactive processes [1]. Despite striking similarity in the clinical, radiographic and to some extent the histologic patterns, the biologic behavior varies, so each lesion may require a different treatment approach.

Keywords: Fibro-Osseous Lesions; Fibrous Dysplasia; Jaws; Ossifying Fibroma; Cemento-Osseous Dysplasia; WHO Classification

Abbreviation

BFOL: Benign Fibro-Osseous Lesions; FD: Fibrous Dysplasia; OF: Ossifying Fibromas; JAOF: Juvenile Aggressive Ossifying Fibroma; JTOF: Juvenile Trabecular Ossifying Fibroma; JPOF: Juvenile Psammatoid Ossifying Fibroma; PCOD: Periapical Cementoosseous Dysplasia; FCOD: Focal Cementoosseous Dysplasia; FICOD: Florid Cementoosseous Dysplasia.

Introduction

Benign fibro-osseous lesions (BFOL) of jaws, facial and skull bones are a variant group of intraosseous disease processes that share microscopic features [4]. In 1940s and early 1950s, these lesions were commonly termed as localized osteitis fibrosa, osteofibroma or fibrous osteoma. The lesions in the skull were termed as leontiasis ossea because it produces lion-like appearance. From a clinical standpoint, fibro-osseous lesions may be associated with significant cosmetic and functional disturbances or they may be completely asymptomatic localized lesions that are identified only on routine radiograph [3].

Eversole 2008 Classification [5]

Bone dysplasias

- Fibrous dysplasia
    - Monostotic
    - Polyostotic

- Polyostotic with endocrinopathy (McCune-Albright)
- Osteofibrous dysplasiaa

- Osteitis deformans
- Pagetoid heritable bone dysplasias of childhood
- Segmental odontomaxillary dysplasia

Cemento-osseous dysplasias

- Focal cemento-osseous dysplasia
- Florid cemento-osseous dysplasia

Inflammatory/reactive processes

- a. Focal sclerosing osteomyelitis
- b. Diffuse sclerosing osteomyelitis
- c. Proliferative periostitis

Metabolic Disease: hyperparathyroidism

Neoplastic lesions (Ossifying fibromas)

- Ossifying fibroma
- Hyperparathyroidism jaw lesion syndrome
- Juvenile ossifying fibroma
    - Trabecular type
    - Psammomatoid type
- Gigantiformcementomas

Discussion

Fibrous dysplasia

Fibrous dysplasia (FD) is an uncommon bone disease that has very low potential for malignant transformation. Diagnostic difficulty is encountered when the clinical, radiological and histological features do not correlate [1].

The true incidence and prevalence are reported to represent approximately 5% to 7% of benign lesions of bone [3,4]. Fibrous dysplasia can present in one bone (monostotic) or multiple bones (polyostotic) and can be associated with other constitutional conditions [2,3].

Etiology and pathophysiology

Fibrous dysplasia is assumed to occur as a result of a developmental failure in completion of the remodeling process of primitive bone to mature lamellar bone which does not mineralize normally& leaves a mass of immature trabeculae entangled in dysplastic fibrous tissue [6]. These phenomena result in significant loss of mechanical strength, leading to the development of pain, deformity, and pathologic fractures [1].

The etiology of fibrous dysplasia has been linked with a mutation in the Gsαgene comprises of the substitution of cysteine (R201C) or histidine (R201H) by arginine at position 201 [7] that occurs after fertilization in somatic cells and is located at chromosome 20q13.2-13.3. All cells that are derived from these mutated cells appear to show the dysplastic features [4,5].

The G-proteins begin a cascade of events that finally leads to activation of the enzyme adenylyl cyclase that produces cAMP.

One mechanism involves c-fos, a protein that forms heterodimers with proteins of the jun family, forming a complex that binds to specific sequences in the promoter region of several genes. Studies done by Sassone-Corsi., *et al*. 1988 have shown that the c-fos promoter contains a cAMP responsive element, and is therefore a potential target for Gsα mutations in fibrous dysplasia [7]. Indeed, Gsα-activating mutations induce c-fos overexpression in vitro (Sassone-Corsi., *et al*. 1988).

In McCune-Albright Syndrome (MAS), a rare disorder that comprises of polyostotic fibrous dysplasia, skin pigmentation (Cafe-au-lait spots), and one or several endocrinopathies [8]. So, overproduction of the cAMP leads to increased amounts of activity that would in turn affect each tissue differently based on its designated function. Cafe-au-lait spots are from overproduction of the enzyme tyrosinase, which is the rate-limiting step in melanin production.

	Bone Involvement		Café au Lait	Endocrine	Soft-Tissue
	Single	**Multiple**	**Spots**	**Disorders**	**Masses**
Monostotic	+				
Polyostotic		+			
McCune-Albright Syndrome		+	+	+	
Mazabraud disease		+			+

Table 1

Clinical presentation

The first symptom is pain in the involved limb, spontaneous fracture, or both [9]. Femoral neck is commonly affected. The age

group ranged from six to fifty-six years [1]. In female patients change in level of estrogen hormone during pregnancy and menstrual cycle results in increase in pain [10]. "Shepherd's crook" deformity is the classic deformity of polyostotic fibrous dysplasia, i.e., curvature of the femoral neck and proximal shaft which results in a coxavara deformity resembling lateral bowing of the proximal part of the thigh, widening of the hip region, and shortening of the limb and is appreciated radiographically.

Study done by Leet., *et al*. showed that lesions of the spine may cause scoliosis [11]. Study by Frodel., *et al* [12]. reported that local expansion of fibrous dysplasia in craniofacial bones like maxilla, zygomatic, or ethmoid bones can produce significant functional and cosmetic deformity.

In McCune-Albright syndrome, a triad of clinical features, i.e., bony lesions, endocrine abnormalities, and café au lait spots are seen. Café au lait spots of skin pigmentation are found around the trunk and the proximal parts of the extremities. They have a discontinuous irregular border that mimics the coast of Maine as compared to the smooth-bordered (coast-of-California) characteristic of neurofibromatosis or von Recklinghausen disease of skin. The incidence of sarcomas has been increased in fibrous dysplasia in recent years. Another type of fibrous dysplasia is Mazabraud Syndrome where there is development of benign intramuscular myxomas along with skeletal lesions [13]. The great majority of patients were reported to have multiple tumors in polyostotic disease.

Microscopic pathology

The key histologic features for diagnosis of fibrous dysplasia are the delicate trabeculae of immature, disconnected dysplastic bone, with no osteoblastic rimming, entangled within a bland fibro cellular stroma of dysplastic spindle shaped cells without any cellular atypia [Figure 1-3].

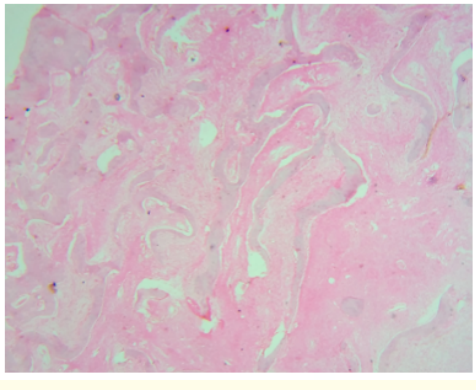

Figure 1: Showing trabeculae of immature, disconnected dysplastic bone, with no osteoblastic rimming.

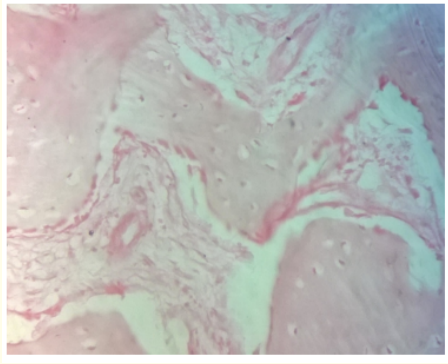

Figure 2: Ossifying Fibroma.

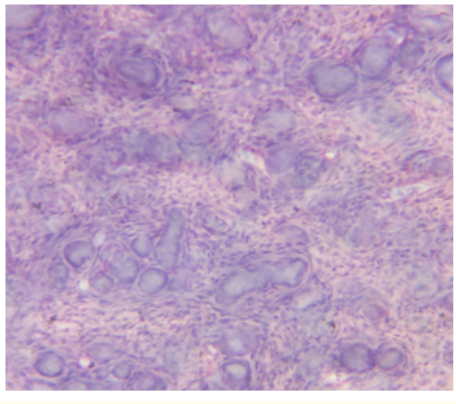

Figure 3: Juvenile Ossifying Fibroma.

The ratio of this fibrous tissue to bony trabeculae ranges from fields that are totally fibrous to those completely filled with dysplastic trabeculae, which is arranged in an alphabetical pattern commonly referred to as resembling "Chinese characters" [14].

These trabeculae, rarely contain reversal lines. Multiple capillaries are found throughout the lesion and, when injured, they incite a giant-cell reactive process. Other secondary changes, like metaplastic chondroid component or aneurysmal bone cyst like changes, which can include cystic degeneration can also occur [9]. However, these findings may make the diagnosis a bit challenging; but the overall histological features are bland and lack cytological atypia, and in most of the cases, usually a classic appearing area of fibrous dysplasia is present for definitive diagnosis.

Immunohistochemistry

Immunohistochemistry is done to rule out the possibility of a malignancy. The cytogenetic changes of FD are structural rearrangements involving 12p13 and trisomy in a small number of re-

ported cases. But still, there are some inconsistent chromosomal findings in FD, and the significance of that is not clear yet.

Laboratory findings

The levels of alkaline phosphatase is increased. Urinary hydroxyproline, an older marker of bone resorption, may also be elevated. In some cases, increased collagen metabolites may be demonstrated (N-telopeptide) [15]. In McCune-Albright syndrome, high levels of growth hormone prolactin, thyroid hormones and, less commonly, testosterone, adrenocorticosteroids, and parathyroid hormone have been reported.

Imaging findings

The radiographic features depend upon the ratio of the mineralized bone to fibrous tissue [18]. Early lesions are radiolucent with ill-defined or well-defined borders, and may be unilocular or multilocular. As the lesions mature, the bony defects acquire a mixed radiolucent/radiopaque appearance, and established fibrous dysplasia exhibits mottled radiopaque patterns, often described as resembling ground glass, orange peel, or fingerprints, with ill-defined borders blending into the normal adjacent bone [16,17].

CT scan is the study of choice for diagnosis and follow-up of fibrous dysplasia because of its superior bony detail and accurate assessment of the extent of the lesion [19].

Magnetic resonance imaging is a sensitive means of establishing the size, shape, content, and the extent of the lesion. It provides complementary information when performed in conjunction with CT imaging.

Differential diagnosis

- Osteofibrous dysplasia
- Simple bone cyst.
- Paget's disease.
- low-grade central osteosarcoma

Management

The management of FD is often cosmetic. Fibrous dysplasia's that are not symptomatic and progressive should simply be monitored. For relief of bone pain and reduction of osteoclastic activity intravenous bisphosphonate therapy can be given [20]. Gene therapy targeted to the abnormal cell populations for skeletal as well as extra skeletal lesions would be futuristic and ambitious treatment modality.

Ossifying fibroma

Ossifying fibromas (OF) of the craniofacial skeleton, as described in WHO classification of odontogenic tumors (2005) (Barnes L 2005), are benign fibro-osseous neoplasms characterized by the replacement of normal bone by a fibrous cellular stroma containing foci of mineralized bone trabeculae and cementum-like material that vary in amount and appearance [21].

These are neoplasms showing progressive proliferative capabilities with bony expansion and, well defined radiological margins [11]. It accounts for only 0.1% of the bony lesions [22]. The tumor is a well demarcated lesion which is occasionally capsulated consisting of fibrous tissue containing variable amounts of mineralized material resembling bone and/or cementum or both [22]. The term ossifying fibroma is used if the predominant component is bone, while cementifying fibroma is defined by the presence of presence of curvilinear trabecular structures or spherical calcifications [23] The lesions characterized by the presence of bone and cementum are referred to as cemento-ossifying fibroma [23]. The term "cement-ossifying fibroma" is not used now as the cementum-like material thought to be of dental origin was found in fibromas occurring in extra-gnathic sites. Thus, excluding the dental origin of these tumors [24].

Ossifying fibromas are subdivided into conventional and juvenile clinicopathologic subtypes [25,26]. Cemento ossifying fibroma, is an odontogenic neoplasm arising from the periodontal ligament and affects the tooth-bearing areas of the jaws, mandible, and the maxilla with cementicles as the characteristic histopathologic feature [21].

Clinical and imagiological features

Ossifying fibroma most commonly occurs in the 2nd to 4th decades and shows a predilection for females [21], Juvenile aggressive ossifying fibroma (JAOF) may present has two histologic variants: Juvenile psammomatoid ossifying fibroma (JPOF) and juvenile trabecular ossifying fibroma (JTOF) [24] JOF needs to be distinguished from malignant bone tumors in that there is a similarity of clinical manifestations, but it can be easily excluded on the routine histological examination [27], juvenile ossifying fibroma are of two types-juvenile trabecular ossifying fibroma and juvenile pssammatoid ossifying fibroma. The mean age of the histological subtypes varies with JTOF showing mean age range of 8.5-12 years whereas patients with JPOF it is about 20 years [21].

Juvenile aggressive ossifying fibroma (JAOF) is a relatively rare fibro-osseous lesion of the jaws characterized by the early age of onset (usually less than 15), tumor location, radiological appearance, and high recurrent potentials [24]. The lesion is known by various name such as juvenile ossifying fibroma, active juvenile ossifying fibroma, aggressive ossifying fibroma, reticular desmo-osteoblastoma or active fibrous dysplasia [28]. The juvenile variants of ossifying fibromas share many similarities, but they differ on the basis of their histopathological features, site, and age of recurrence [29] (Shields, Peyster., *et al*. 1985; Noudel, Chauvet., *et al*. 2009; Smith, Newman., *et al*. 2009). In conclusion, the term "Juvenile aggressive ossifying fibroma" is a misnomer as it can occur in adults as well [30].

JTOF also known as trabecular desmo-osteoblastoma [34,35]. Clinically, it is presented as an asymptomatic lesion with progressive expansion. Radiographically JPOF shows a round, well defined, sometimes corticated osteolytic lesion with a cystic appearance [31].

The preferred site of occurrence is reported to be mandible varying from70%-89% of cases and maxilla in 11%-26% with affinity for premolar &molar area, although posterior mandibular lesions may extend upward into the ascending ramus Goaz and White [22] reported that when OF occurs in the maxilla, it is most commonly located in the canine fossae and zygomatic arch. The maxillary lesions were found to be more aggressive. Lesion produces a notable swelling and mild deformity and facial asymmetry with or without displacement of teeth. When rapid growth does occur, the symptoms are related to the lesion site and may include painless cheek swelling, unilateral proptosis, diplopia and epistaxis [22]. Death is a rare occurrence secondary to intracranial extension (Table 2).

Etiopathogenesis

Odontogenic, developmental and traumatic origins of ossifying fibroma have been suggested [21]. It has been hypothesized that JPOF originates from overproduction of the myxofibrous cellular stroma normally involved in the growth of the septa in the paranasal sinuses as they enlarge and pneumatize. These stromal cells secrete hyaline material that ossifies and connective tissue mucin that initiates the cystic areas [21].

JTOF	JPOF
Mainly affects children and adolescents	Young adults.
Affect maxilla as well as mandible.	Affects extragnathic bones particularly centered on the periorbital, frontal, and ethmoid bones [21].
Radiographically, JTOF is an expansive lesion and may be fairly well demarcated, with cortical thinning and perforation [21]. The lesion shows mixed radioopaque radiolucency depending on the amount of calcified tissue produced [32], sometimes Ground-glass as well as a multilocular honeycomb appearance has been described	Radiographically JPOF shows a round, well defined, sometimes corticated osteolytic lesion with a cystic appearance [31].
Cystic degeneration and aneurysmal bone cyst formation are few complications.	Orbital extension of sinonasal tumors may result in proptosis, and visual complaints including blindness, nasal obstruction, ptosis, papilledema, and disturbances in ocular mobility [33].
	The psammomatoid type of juvenile ossifying fibroma, is more aggressive, and it has a strong tendency to recur
JTOF showed osteoid and immature woven bone trabeculae. Osteoblastic rimming was commonly observed among JTOF but absent in JPOF [30].	All cases of JPOF showed presence of psammoma bodies which can be considered as a main diagnostic feature [30].

Table 2

Genomic alterations

Cytogenetic analysis was done in only a few cases of ossifying fibroma. In one case of Cemento ossifying fibroma of the mandible, deletions were detected in 2q31-32 q35-36 [33]. A study of 3 cases of JPOF of the orbit demonstrated nonrandom chromosome break points atXq26 and 2q33 resulting in (X; 2) translocations [21]. Regarding OF ossifying fibroma not otherwise specified NOS there

are reports that identify mutations in HRPT2 a gene that encodes parafibrominprotein [21].

Microscopy

Microscopically ossifying fibroma shows fibrous and osseous tissues with the former tissue predominating. A fibrous capsule may be seen in some cases the fibrous stroma is highly cellular, with spindle-shaped fibroblastic cells. The osseous tissue consists of rounded or lobulated basophilic masses (cementum-like), trabeculae of osteoid, woven or lamellar bone, with or without osteoblastic riming [36]. Sometimes focal clusters of giant cells (osteoclasts) can be seen to be arranged haphazardly or adjacent to the mineralized material [37].

Juvenile ossifying fibromas

Two distinct clinicopathologic entities are known

- Microscopic features of Psammomatoid variant *of* juvenile ossifying fibroma shows multiple small acellular calcified structures, round and uniform and with concentric lamellar calcification, called ossicles/psammomatoid bodies which are homogenously distributed in a relatively cellular stroma that may have whorled appearance, composed of uniform, stellate, and spindle shaped cells. The psammomatoid bodies are basophilic and bear superficial resemblance to dental cementum, but may have an osteoid rim [21].
- Microscopic features of trabecular variant of juvenile ossifying fibroma shows a well-defined but unencapsulated lesion that infiltrates surrounding bone, composed of a cell-rich fibrous stroma containing bundles of cellular osteoid and bone trabeculae without osteoblastic rimming. Anastomosing trabeculae of osteoid is seen in a pattern that resembles 'paintbrush' strokes [38,39].

Differential diagnosis of ossifying fibroma includes

- Focal cementoosseous dysplasia
- Fibrous dysplasia.

The ossifying fibroma is found to be relatively hypovascular and well demarcated from the surrounding tissue, which is not seen in fibrous dysplasia.

- JPOF might be easily mistaken for psamomatous meningioma. The diagnosis should be based on morphological, clinical and radiographic findings [21,40].

Treatment

Complete excision of a OF lesion is the treatment of choice and it can be curative Radiotherapy is generally contraindicated because of the risk of malignant transformation and the potentially harmful late effects in children. Prognosis is excellent and recurrence after removal is seldom seen.

Cemento osseous dysplasia

Osseous dysplasias are the most common form of BFOL in the jawbones. The term dysplasia refers to the abnormal production and disordered development of bone and cementum-like material [41]. Their confinement to the alveolar process; in the mandible above the inferior dental canal strongly suggests an odontogenic origin of these lesions [42]. On the basis of their clinical and radiographic features ,they are divided into three groups: periapical, focal and florid cemento-osseousdysplasia [43]. The observation that Periapical cementoosseous dysplasia (PCOD) and Focal cementoosseous dysplasia (FCOD) share the same histopathology and almost similar clinical profiles prompts one to consider that both are the same entity with different locations [5]. Florid cemento-osseous dysplasia (FlCOD) denotes an extensive process with multifocal involvement of the jaws by lesional tissue with the same microscopic appearance as that of PCOD and FCOD. The histologic appearance of osseous dysplasias varies depending on the stage of the lesion [44]. Cemento-osseous dysplasia is a condition that do not require treatment, but periodic observation is advisable.

Periapical cementoosseous dysplasia

Waldron described PCOD as a reactive or dysplastic FOL in the tooth-bearing area, presumably of periodontal ligament origin of unknown aetiology [45]. The World Health Organization (WHO) in their Histological Typing of Odontogenic Tumor's (1992) referred to PCOD as periapical cemental dysplasia (PCD) (Kramer., *et al*. 1992) and classified it as a type of cemento-osseous dysplasia under non neoplastic bone lesions [46].

PCOD is a reasonably well defined clinical-radiological entity, predominantly involving the apical areas of vital mandibular incisors [42]. The WHO definition of periapical cemental dysplasia, is ''a non-neoplastic lesion affecting the periapical tissues of one or more teeth'', which therefore does not subclassify this lesion according to any defined location (i.e.: anterior vs. posterior apical areas of jaws).

Clinically PCOD presents as a well-defined lesion associated with one or more vital and asymptomatic mandibular anterior teeth. It is predominant in black women older than 44 years of age. Solitary or multiple lesions may occur but multiple lesions are present more frequently [43]. The diagnosis is usually made with a routine radiographic examination. Three distinct stages of progression of PCOD is noted radiographically and histologically (Sapp., *et al*. 2002):

(i) **Initial osteolytic stage:** characterized by well-defined radiolucencies at the apex of one or more teeth which are usually indistinguishable from inflammatory periapical lesions of pulpal origin. Histologically, the tissue consists primarily of cellular connective tissue replacing normal trabecular bone with calcified structures of insufficient size to be evident radiographically.

(ii) **Cementoblastic stage:** characterized by the presence of radiolucent areas containing nodular radiopaque deposits. Histologically, there is a mixture of spherical calcifications and irregularly shaped deposits of osteoid and mineralized bone.

(iii) **Mature stage**: characterized by well-defined, dense radiopacities usually surrounded by a radiolucent rim. The periodontal ligament can be seen separating the lesion from the root. Histologically, coalesced spherical calcifications and sclerotic mineralized bone makes up most of the tissue [45].

Focal cementoosseous dysplasia

Since the publication of the second edition of the WHO's histological classification of tumours, focal cemento-osseous dysplasia (FCOD) has been recognized as a form of cementoosseous dysplasia (COD). Previously, Waldron, observing its localized nature, first reported it as the ''localized fibro-osseous-cemental lesion'', which Summerlin and Tomich renamed as focal cementoosseous dysplasia [47]. They further characterized its salient features and compared them with those of the cemento ossifying fibroma (COF), most likely to feature on its differential diagnosis. Waldron suggested that focal osseous dysplasia likely represents the most common BFOL of the jawbones. However, many cases probably are misdiagnosed as OF [48]. Affecting factors including trauma, caries, periodontal disease, infection, hormonal imbalance or systemic diseases have not been clarified. The aetiology and pathogenesis of FCOD remain unknown, and this lesion is considered to be a reactive or dysplastic process in periapical tissues. Almost all FCODs appear above the mandibular canal and are thus confined to the alveolar process, suggesting at least some odontogenic influence on their genes.

FCOD exhibits a single site of involvement and manifest most commonly as a small, solitary, relatively well-demarcated lesion in the posterior mandible, either in close association with the apices of teeth or in areas where a tooth has been extracted previously. The majority of these lesions are asymptomatic with an average size of 1.5 cm and are detected during routine radiologic examinations. The related tooth is vital. Like PCOD, FCOD is much more common in black females and most are recognized during the fourth and fifth decades of life.

Radiologically, this lesion has three stages of maturation: pure radiolucent, mixed radiolucent/opaque and radiopaque appearances [49]. The early stage classically presents with a well-defined radiolucency at the apices of mandibular teeth which may be misdiagnosed as an endodontic infection. In dentate areas, neither tooth displacement nor root resorption is observed. A pulp vitality test should clarify the clinical confusion. Intermediate stage demonstrate a mixed radiolucent/ opaque pattern, with a well-defined radiolucent rim around the radiopacity [5]. At later stage the lesion demonstrates a diffuse radiopacity, with ill-defined borders and a greater proportion of thick curvilinear, poorly cellular and anastomosing bony trabeculae (the so-called 'ginger root' pattern) [49]. The radio-opaque appearance of FCOD can be cotton wool-like irregular or diffuse. The histopathological appearance consists of bone trabeculae and cementum-like material in a vascular fibrous stroma [50]. FCOD do not require surgical intervention as they are benign and their growth is limited [51]. These lesions should be followed regularly, because they can turn into FLCOD, which is the advanced form of dysplasia, or simple bony cysts can develop within the FCOD areas.

Florid cementoosseous dysplasia

Florid osseous dysplasia first described by Melrose., *et al*. in 1976 is the most clinically extensive form of osseous dysplasia [52]. However, in most cases, it is an innocuous, self-limiting disease [53]. Like the other 2 forms of osseous dysplasia, florid osseous dysplasia arises mainly in black females during the fourth and fifth decades of life, although it also may occur in Caucasians and Asians [54].

Florid osseous dysplasia is a clinical and radiographic diagnosis in which at least 2 quadrants must be involved to make the diagnosis [52]. The disease is limited exclusively to the tooth-bearing areas of the jaws, thus sparing the inferior cortex and ascending ramus of the mandible. Both dentulous and edentulous areas may be affected. It commonly involves the posterior regions of the mandible, manifesting as bilateral, relatively symmetrical lesions [55]. In most cases affected patients are asymptomatic, and the disease is detected only on routine dental radiographs [44]. Exposure of sclerotic calcified masses in the oral cavity results in symptoms such as dull pain or drainage [56]. This may occur as the result of progressive alveolar atrophy under a denture or after extraction of teeth in the affected area. Unlike patients with PCOD and FCOD, patients with FlCOD may have limited bony expansion. Radiographs often demonstrate numerous, irregularly shaped, sclerotic radiopacities admixed with diffuse, ill-defined, radiolucent-radiopaque areas. Anterior mandibular involvement typically manifests radiographically as periapical osseous dysplasia, which reinforces the concept that both the lesions are part of the same disease spectrum [43]. Concomitant multiple traumatic bone cysts commonly develop in association with FlCOD, manifesting as well-delineated, cyst like radiolucencies. In occasional cases, these cysts may represent the initial manifestation of florid osseous dysplasia. Therapeutic management for asymptomatic patients consists of long-term follow-up [57]. In symptomatic patients antibiotic therapy and surgical debridement of the infected sclerotic bone are necessary.

Differential diagnosis

- Periapical granuloma or cyst and chronic osteomyelitis in the osteolytic stage.
- Chronic sclerosing osteomyelitis [58], ossifying/cementifying fibroma [59], osteoblastoma and ameloblastoma in the mixed and radiopaque stages.

Conclusion

Benign fibro-osseous diseases of the maxillofacial bones make up a diverse collection of disorders that include neoplastic and nonneoplastic diseases and hereditary and nonhereditary conditions [60]. Furthermore, a number of other non–fibro-osseous disease processes that develop within the jawbones exhibit findings that may closely mimic those seen in BFOLs. In recent years significant progress has been achieved in understanding the histopathogenic similarities and differences of various fibro osseous lesions. Correlation of the histologic features with the clinical, radiographic, and intraoperative findings along with awide knowledge on the molecular biology of various BFOL is important to ensure early accurate diagnosis and appropriate management of the disease.

Conflict of Interest

No conflict of interest.

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