REVIEW ARTICLE

BENIGN FIBRO-OSSEOUS LESIONS OF JAWS- A REVIEW

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Abstract
Benign Fibro-osseous Lesions is a group of lesions in which normal bone is replaced initially by fibrous connective tissue and over a period of time, the lesion is infiltrated by osteoid and cementoid tissue. This is a benign and idiopathic process. Fibro-osseous lesions of the maxillofacial bones comprise a diverse group of pathologic conditions that include developmental lesions, reactive or dysplastic diseases, and neoplasms. The concept of fibro-osseous lesions has evolved over the last several decades and now includes two major entities: fibrous dysplasia and ossifying fibroma. The less common lesions include florid osseous dysplasia, periapical dysplasia, focal sclerosing osteomyelitis, proliferative periostitis of Garre, and osteitis deformans. It represents a diverse group of pathological conditions that includes developmental lesions, reactive or dysplastic diseases, and neoplasms. Owing to substantial overlap of the histopathologic findings, sub classification of Benign Fibro-osseous Lesions may be problematic. Despite the advances in the understanding of these conditions, fibro-osseous lesions continue to present problems in classification, diagnosis, and management due to multiple histological and radiographic similarities. The objective of this article is to review the most current clinicopathologic, radiographic, and molecular studies of Benign Fibro-osseous Lesions to aid the surgical pathologist in the recognition and diagnosis of this diverse group of maxillofacial lesions.
Introduction

The fibro-osseous lesions represent a large group of disorders that may have common characteristics including clinical, radiographic and microscopic features. Although most are of unknown etiology, some are believed to be neoplastic and others are related to metabolic imbalances. It is not unusual to see these lesions presenting with a range of radiographic appearances, causing considerable diagnostic confusion. Benign fibro-osseous lesion is a well-known, descriptive term that encompasses a wide range of conditions, the diagnoses of which may be challenging. In part, the challenge arises because the histopathological appearances of all fibro-osseous lesions are very similar, if not identical, making clinical diagnosis difficult based on microscopic features alone. The maxillofacial fibro-osseous lesions are the lesions that are different (except fibrous dysplasia) to those found in the rest of the skeleton.

Charles Waldron wrote “In absence of good clinical and radiologic information a pathologist can only state that a given biopsy is consistent with fibro-osseous lesions. With adequate clinical & radiologic information, most lesions can be assigned with reasonable certainty into one of the several categories owing to their similar histology. Radiographically, fibro-osseous lesions vary considerably from a simple radiolucent lesion to mixed radiolucent/radiopaque or radiopaque lesion. These can be well defined or ill-defined blending imperceptibly into the surrounding bone. There may or may not be expansion of bone, with or without displacement of tooth. Histologically, the fibro-osseous lesions mainly consist of two components - hard tissue and soft tissue component. The treatment of fibro-osseous lesions varies depending on the nature of the lesion. It may vary from simple surgical excision or curettage in cemento ossifying fibroma to a surgical excision and resection of the involved jaw in cases of juvenile ossifying fibroma, osteogenic sarcoma and chondrosarcoma.

Classification

Charles A Waldron in 1985 classified fibro-osseous lesions into main groups on the basis of clinical behavior, histopathology and radiographic findings:

1) Fibrous Dysplasia
2) Reactive (dysplastic) lesions arising in the tooth bearing area: Periapical cemento osseous dysplasia
   Focal cemento osseous dysplasia
   Florid cemento osseous dysplasia
3) Fibro – osseous neoplasms
   Cementifying fibroma
   Ossifying fibroma
   Cemento ossifying fibroma
FIBROUS DYSPLASIA
It is one of the most perplexing diseases of osseous tissues & has been described as a lesion of unknown etiology, uncertain pathogenesis and diverse histopathology. It is a congenital, metabolic, non-familial disturbance that produces 2.5% of all bony tumors and over 7% of all non-malignant tumors of bone. It is a benign fibro-osseous lesion characterized by formation of fibrous connective tissue within the spongiosa of the affected bone and often by the painless expansion of that bone to cause deformity. There is the replacement of normal bony architecture with fibrous and osteoid tissue. It may also contain islands of calcified tissue, the appearance of which is dependent on the age of the lesion. There is proliferation of fibroblast-like cells that have features of osteoblasts in some areas and those of chondroblasts in others. It occurs because of maturation arrest of bone formation at the stage of woven/fibre bone. The resultant fibro-osseous tissue is poorly formed, elastic and structurally inadequate. It can impair cosmetic & structural function of bone leading to osteolytic lesions, fractures, & deformations. It may involve one or more bones of the cranial or extra cranial skeleton. It has two basic clinical forms: monostotic and polyostotic. It may also be associated with endocrine dysfunction, abnormal pigmentation, and precocious puberty in girls.

Etiology and Pathogenesis

Fibrous dysplasia is postulated to occur as a result of a lack of stress alignment and insufficient mineralization results in substantial loss of mechanical strength, leading to the development of pain, deformity, and pathologic fractures. Marie et al showed that an activating mutation of Gsα in osteoblastic cells of patients with McCune-Albright syndrome and monostotic disease leads to constitutive activation of adenylate cyclase, increased cell proliferation, and inappropriate cell differentiation, resulting in overproduction of a disorganized fibrotic bone matrix in polyostotic and monostotic fibrous dysplasia. Pregnancy has been implicated in exacerbation of fibrous dysplasia perhaps because of estrogen receptors in the fibrous tissue.

Clinical Features

It occurs most commonly in second or third decade of life. The average age of occurrence is ten years. Some studies revealed no gender predilection. Male to female ratio in some studies is 2:1. Some studies also show that sex predilection is almost equal. Among the jaw bones, maxilla is more commonly affected than the mandible.

The most common sign is painless expansion of the affected area and deformity of the affected site. The foramina of cranial nerves, may be encroached upon producing nerve palsies, the disfigurement may be
extreme justifying the term "leontiasis ossea". Diffuse polyostotic lesions in large weight-bearing bones are prone to lead to bowing deformities that increase with age and skeletal growth. Unlike deformities in patients with monostotic disease, deformities in patients with polyostotic disease may continue to progress after skeletal maturity.

**Oral Manifestations**

Pain or paraesthesia is an unusual complaint. Displacement of the teeth with resultant malocclusion and interference with normal eruption patterns may occur. In children teeth in the affected part may fail to erupt. Dental dysplasia is a disorder that occurs in patients with inherited fibrous dysplasia. Pathologically, fibrous tissue that is firm, rubbery, and gritty.

Histologically, fibrous dysplasia consists of varying amounts of spindle cell bundles and trabeculae of immature woven bone. The fibrous tissue in fibrous dysplasia is well vascularized and often show numerous small vessels in the centre and large peripheral sinusoids. Three site specific patterns of histopathology have been identified. Chinese writing type; sclerotic/pagetoid type; and sclerotic/ hypercellular type.

**Radiographic Picture**

The lesions of fibrous dysplasia are usually poorly circumscribed, with the lesions demonstrating a blending margin and are radiopaque (ground glass appearance) although early lesions may be largely radiolucent. According to Akintoye, it can present as a spectrum of four patterns in a panoramic radiograph: ground glass (condensed/granular), radiolucent (lytic), mixed radiolucent/radiopaque (mixed density) and radiopaque (sclerotic). Variations in the cortical thickness are caused by slow resorption of the endosteal surface, commonly referred to as "endosteal scalloping." The peristeal surface remains smooth.

Three varieties of appearances are seen on CT scan: ground glass pattern, homogeneously dense pattern, and cystic variety. Magnetic resonance imaging in addition can help distinguish fibrous dysplasia from meningioma, osteoma, or mucocele and define the extent of soft tissue involvement, particularly if central nervous system structures are impinged.

Single photon emission computed tomography has been reported to be more sensitive in detecting the areas involved in cases of fibrous dysplasia. A slight elevation of serum alkaline phosphatase may be seen in some cases but may not always be raised. Calcium, phosphate and various other hormones are seen in normal range.

I) Polyostotic Fibrous Dysplasia

Involvement of two or more bones is called as polyostotic fibrous dysplasia. Two apparent types of polyostotic fibrous dysplasia are described:
a) Jaffe – Lichtenstein syndrome
b) Mc Cune –Albright syndrome

It most commonly occurs in childhood. Median age of onset of symptoms is 8-10 years, with most occurring before the age of ten\textsuperscript{11}. The disease apparently has a distinct tendency to occur in women with a male: female ratio of 1:3\textsuperscript{11}. Long bones of extremity are most often affected in following order of frequency: femur, tibia, humerus & radius. Next in order of frequency are bones of the skull (cranial vault & jaw bones)\textsuperscript{11}.

Clinical Presentation

A limp, pain in leg and fracture is the initial symptom. It pursues a protracted clinical course characterized by pain, deformity & a tendency to pathological fracture of the affected bones\textsuperscript{11}. Leg length discrepancy is very common as a result of involvement of the upper portion of the femur\textsuperscript{12}. Frequently identified deformities include coxa vara, the shepherd’s crook deformity, bowing of the tibia etc\textsuperscript{10}. The objective features seen in roentgenograms of bones affected by polyostotic fibrous dysplasia include: broadening or expansion of bone, thinning of cortex, characteristic rarefied & apparently trabeculated appearance, secondary deformities of affected bones\textsuperscript{11}. Premature secretion of pituitary follicle stimulating hormone has been reported as well as moderately elevated basal metabolic rate. Most surgical tissue is obtained by curettage. The specimen has a distinct gritty feeling reflecting the osteoid trabeculae inherent in the lesion.

The typical microscopic findings of fibrous dysplasia show irregularly shaped trabeculae of immature bone in a cellular, loosely arranged fibrous stroma. The bony trabeculae are not connected to each other\textsuperscript{12}. Stellate osteoblasts are seen particularly in active lesions and appear to arise from fibroblasts.

II) Monostotic Fibrous Dysplasia

It is more common than the polyostotic type. It most commonly occurs at the age of 20 to 30 years with some cases becoming dormant by the third decade and hormonal changes like in pregnancy reactivating a dormant lesion\textsuperscript{7}. It can also occur in infancy\textsuperscript{6}, occurs with apparently equal predilection for males and females. Ribs and craniofacial bones are most commonly affected\textsuperscript{7}. Other bones affected include, clavicle, tibia, femur etc. The patient may be asymptomatic and lesion discovered incidentally or patient may present with a painless swelling caused by a slow growing lesion causing expansion of the jaw and producing a non tender facial asymmetry\textsuperscript{13}. In children the teeth may fail to erupt\textsuperscript{13}. Fibrous dysplasia of the maxilla is an especially serious form of the disease since it has a marked predilection for occurrence in children. Severe malocclusion and bulging of the canine fossa, or extreme prominence of the zygomatic process, producing a marked facial deformity, are typical sequelae of this disease in maxilla.

Serum alkaline phosphatase and urinary hydroxypoline are examples of
useful markers and are used to monitor response in the non surgical treatment of disease rather than for diagnosis\textsuperscript{13}. A ground glass or orange peel appearance is seen when there are areas of condensation interspersed with areas of radiolucency. The lesion causes resorption of roots of erupted teeth\textsuperscript{13}. It may show focus of gritty tissue in the bone\textsuperscript{8}.

The trabeculae may be devoid of osteoblastic rimming, thereby appearing to be formed by fibroblastic osseous metaplasia\textsuperscript{13}.

**Syndromes Associated with Fibrous Dysplasia**

McCune-Albright syndrome is an endocrinopathy occurring mainly in girls, consisting of the triad of precocious puberty, polyostotic fibrous dysplasia and characteristic cutaneous pigmentation. The cutaneous lesions are flat pigmented macules, often referred to as "café au lait" spots. Mazabraud syndrome is the rare combination of fibrous dysplasia and soft-tissue myxomas. There are three modes of treatment i.e. observation, medical therapy & surgical treatment. Cortisone has been reported to produce some relief in pain of bone lesions. Important line of medical treatment is with bisphosphonates which inhibit osteoclastic activity\textsuperscript{6}. Young patients receiving pamidronate should be monitored with serial radiographs to check for a transient mineralization defect, which presents as increased growth plate thickness, thickening of cortices and/or ossification of radiolucent areas\textsuperscript{10}.

**Surgical Treatment**

According to El Deeb M, the treatment of choice is surgical, depending upon the size of the lesion as ascertained by the radiographic picture and by biopsy. In the osteolytic type radical curettage is indicated, whereas in the more mature, solid type surgical shaving and recounting is indicated. Fibrous dysplasia is treated by curettage and packing with cancellous chip grafts, by subperiosteal excision and cancellous bone graft, by extraperiosteal excision and cancellous bone graft, cortical graft or both. In maxillofacial area, a common procedure is to delay surgery until bone growth ceases and to contour the bulged portion of the bone for an esthetic appearance. In case of visual disturbance caused by compression of optic nerve, immediate surgery is needed\textsuperscript{5}. According to Edgerton, the surgical techniques used are:

1) Simple bone contouring

2) Resection and acrylic implant

3) Resection, remodeling and replantation

Recurrence of fibrous dysplasia following curettage is more common in children than in adults. This “remove, reshape, and replant” technique has excellent bone healing, good postoperative contours, and no clinical evidence of recurrence of bone enlargement. Malignant transformation of fibrous dysplasia occurs very infrequently, with reported prevalence’s ranging from 0.4% to 4% with average incidence being 1%\textsuperscript{10}. The most common
malignant tumors were osteosarcoma, fibrosarcoma, and chondrosarcoma.

**Differential Diagnosis**

Other entities which may be confused with fibrous dysplasia are ossifying fibroma, cemento osseous dysplasia, Paget disease, cementoma, cherubism, hyperparathyroidism, chronic sclerosing osteomyelitis, osteogenic sarcoma etc. Lesions that may suggest fibrous dysplasia include simple bone cysts, nonossifying fibromas, osteofibrous dysplasia, adamantinoma, low-grade intramedullary osteosarcoma, and Paget disease.

**CEMENTO-OSSEOUS DYSPLASIA (COD)**

Cemento-osseous dysplasia is the most common fibro-osseous lesion occurring in the tooth bearing areas of the jaws. COD is a group of non neoplastic processes usually confined to the tooth bearing areas of the jaws or edentulous alveolar processes. Many terms have been used to refer to cemento-osseous dysplasia: Periapical cemento-osseous dysplasia (PCD), Florid osseous dysplasia (FOD), Florid cemento osseous dysplasia (FLCOD), Focal cemento osseous dysplasia (FCOD).

Robinson attributed the cause as injured bone reacts in an abnormal way to low-grade or chronic injury by resorbing formed bone trabeculae and replacing it with cellular fibrous connective tissue, in which immature bone and a cementum-like substance are deposited.

**Periapical Cemento-Osseous Dysplasia** also known as cementoma, osseous dysplasia and periapical cemental dysplasia. The first comprehensive clinical, radiographic, and histopathologic study was reported by Stafne in 1933. Blum in 1930 and Thoma in 1937 and 1944 defined its histopathology. PCD is not a true neoplasm but a dysplastic condition in which multiple focal areas of bone and marrow are replaced by cellular connective tissue lesions with limited growth potential. The lesion attains a fixed size and later undergoes a maturation process that culminates in the formation of multiple dense calcified intraosseous nodules. PCD is an asymptomatic lesion often discovered on routine radiographic examination. Multiple lesions are often present. Buccal and lingual expansion of the cortices is often absent.

The age of occurrence has been variably reported by various authors from 3rd to 5th decade with a range of 14-82 years and mean of 42.5 years with cases rarely occurring before 20 years of age. Mandible (68.15%) is more commonly affected than maxilla. The lesion principally involves the apical area of one or more vital mandibular teeth, particularly the incisors. Female to male ratio has been variably reported between 10:1 to 14:1. Most lesions are less than 0.5 cm in size. Maximum size rarely exceeds 1.5 cm. Periapical cemento-osseous dysplasia has been classically described as progressing through 3 radiographic stages.

1) Osteoloytic stage
2) Cementoblastic stage.
3) The third or mature stage
Differential diagnosis

Radiolucent stage

- Apical periodontal granuloma or a radicular cyst
- Primordial odontogenic cyst
- Early phase of ossifying fibroma
- Chronic osteomyelitis (if 4 to 6 anterior teeth are involved)

Mixed stage and radiopaque stage

- Odontoma
- Chronic osteomyelitis
- Ossifying fibroma
- Osteoblastoma

Treatment

Only periodic observation is necessary during which one would expect to see the radiographic changes associated with maturation of the lesion.

Focal Cemento-Osseous Dysplasia

This condition derives histogenetically from elements of the periodontal ligament. Other etiological theories consider it to be a reactive lesion and it is more common in women. Regardless of stage, an important diagnostic feature is its close association with the periapex or previous extraction site. Focal cemento-osseous dysplasia tends to be well demarcated with or without cortication. There is no bowing of inferior mandibular border.

At the time of surgical exploration, the surgeon usually finds gritty hemorrhagic material. These gross findings contrast sharply with those of cemento-ossifying fibromas, which share many features histologically. The latter neoplasms tend to enucleate in one piece and are often white, glistening and homogeneous on cut surface. Radiology was of central importance to the detection of at least 64% of focal cemento-osseous dysplasias found incidentally to radiography. On the basis of histopathologic study, 3 progressive stages can be identified: The early (osteolytic), the intermediary (fibro-osseous), the late (Osteosclerotic).

Because focal cemento-osseous dysplasia generally exhibits little or no tendency to enlarge even after partial removal of the lesion, these lesions do not require any treatment.

Florid Cemento-Osseous Dysplasia

Cemento-osseous dysplasia has a pattern of expression that is often multifocal and commonly affects all
quadrants of the maxilla and mandible. This multifocal expression is known as florid cemento-osseous dysplasia. It is clinically the most extensive form of cemento-osseous dysplasia and hence the term florid. Melrose et al initially reported FLCOD as florid osseous dysplasia. The disease appears to have a familial distribution; it is more common in women\(^2\). The disorder is strictly localized to the tooth bearing areas and not associated with any other skeletal deformity. When the lesions are large, jaw expansion may be noted, particularly of the mandible leading sometimes to facial deformity, symptoms such as dull pain, discharging sinuses or sequestrations. Occasionally, patients without signs of infection complain of an intermittent, dull, aching sensation in the mandibular molar area. All Teeth have normal spontaneous pain and are vital. It is seen more commonly in females\(^3\). They have striking tendency towards bilateral, often quite symmetrical, location, and it is not unusual to find extensive lesions in all four quadrants, particularly the posterior region (molar-premolar region). They affect only the alveolar processes and seem to be independent of teeth. Lesions have been found more commonly in mandible and sometimes in the maxilla.

Radiographically, a wide spectrum is seen. Radiographs usually display diffuse distribution of lobular, irregularly shaped radiopacities throughout the alveolar process. The lobular densities are often enmeshed in poorly defined areas of decreased radiodensity, often having a ground-glass appearance. The lesions appear as multiple sclerotic masses, located in two or more quadrants usually in the tooth bearing areas. Biopsy is not necessary.

Management of FLCOD is often difficult and not very satisfactory. In the asymptomatic patient, it is probably wise to keep the patient under observation without surgical intervention because the radiologic features are diagnostic. Management of the symptomatic patient is more difficult. Sequestration of the cementum-like masses will occur slowly and healing will follow this. Saucerization or surgical excision of the sclerotic masses is often not successful and may make matters worse\(^1\).

**Differential Diagnosis**

These include chronic diffuse sclerosing osteomyelitis, Paget’s disease of bone, the osteomas of Gardner’s syndrome, Gigantiform cementoma, osteogenesis imperfecta and polyostotic fibrous dysplasia.

**Malignant Potential**

Development of malignant spindle cell tumor has been reported in a patient with FLCOD but it is a rare occurrence.

**Hereditary Cemento Osseous Dysplasia/Gigantiform Cementoma**

In 1953, Agazzi & Belloni reported a condition that was clinically and radiographically similar to florid cemento-osseous dysplasia but was inherited as an autosomal dominant trait. They proposed the name Gigantiform cementoma. This condition is rare. The gnathic
enlargement in most patients results in significant facial deformity, as well as impaction, malposition and malocclusion of the involved dentition. If not treated the osseous enlargement eventually ceases during the 5th decade. Usually develop during 1st decade of life and by adolescent typical obvious lesions are noted and followed by a rapid and expansive growth pattern. It demonstrates multifocal involvement of both maxilla and mandible. The initial features resemble those seen in cemento-osseous dysplasia, appearing as multiple radiolucencies, in the periapical regions. With progression, the affected sites expand to replace much of the normal bone within the involved quadrant and develop a mixed radiolucent and radiopaque pattern. With further maturation, the lesion becomes predominantly radiopaque but often maintain a thin radiolucent rim. Extensive resection of the altered bone and reconstruction of the facial skeleton and associated soft tissue is recommended can produce acceptable functional and aesthetic result.

Differential Diagnosis

Osteitis deformans or Paget’s disease of bone, chronic sclerosing osteomyelitis, Sclerotic cemental masses, chronic productive osteitis, osseous dysplasia, multiple enostoses.

OSSIFYING FIBROMA (OF)

Ossifying fibroma is a benign odontogenic tumor of mesenchymal origin. OF behaves like a benign bone neoplasm. The tumor is demarcated and occasionally encapsulated lesion consisting of fibrous tissue containing variable amounts of mineralized material resembling bone and/or cementum.

Montgomery was first to coin the term “ossifying fibroma tissue within which the bone is formed. It accounts for only 0.1% of the bony lesions. Ossifying fibroma belongs to the poorly defined group of fibro-osseous lesions involving the jaws and craniofacial bones that result in replacement of the bone by fibrous tissue and subsequent mineralization. The cause of the ossifying fibroma remains unknown. OF usually occurs between the 3rd and 4th decade of life with the average age being 30 yrs. Marked predilection for occurrence is reported to be seen in females with female to male ratio varying from 1.56:1 to 5:1.

Goaz and White reported that when OF occurs in the maxilla, it is most commonly located in the canine fossae and zygomatic arch. It may grow to completely fill the maxillary sinus. It can effect both maxilla and mandible but the preferred site of occurrence is reported to be mandible varying from 70%-26% with affinity for premolar & molar area. The maxillary lesions were found to be more aggressive. Ossifying Fibromas are associated with a slowly progressing enlargement of the affected bone. Lesion is asymptomatic until the growth produces a notable swelling and mild deformity and facial asymmetry. Displacement of teeth is an early clinical feature. When rapid growth does occur, the symptoms are related to the lesion site and may include painless cheek swelling, unilateral proptosis, diplopia and
epistaxis. Death is a rare occurrence secondary to intracranial extension. These lesions may occasionally have ill-defined border, if relatively rapid growth occurs. As the lesion matures, mixed radiolucent and radiopaque appearance may be seen. The characteristic features of OF in radiographs are expansion and lesion margination, demarcation, or cortication. Cortical expansion is present, often with an eggshell-thin cortex. Large ossifying fibromas of mandible often demonstrate a characteristic downward bowing of inferior cortex of mandible.

On surgical exploration, the tumor is found to be relatively hypovascular and well demarcated from the surrounding tissue, permitting relatively easy separation from the surrounding bone. Some lesions will have a definite capsule. This demarcation from the surrounding tissue is an important feature in distinguishing OF from FD.

The vascular spaces resemble arterioles or capillaries displaying a continuous endothelial layer with plump endothelial cells protruding into the capillary lumen. The calcified component consists of rounded or lobulated basophilic cementum-like masses, trabeculae of osteoid or bone or combinations of both, the majority of bony trabeculae in cemento-ossifying fibroma are thin, single, and separate with osteoblastic rimming.

Treatment of ossifying fibroma involves the complete removal of lesion by curettage, enucleation, or excision. Complete excision of the tumor has become a necessity since it is notorious for recurrence.

Juvenile Ossifying Fibroma

Juvenile (aggressive) ossifying fibroma was used in 2nd edition of WHO classification of odontogenic tumor of children to describe a lesion affecting the jaws under the age of 15 years.

Definition

The second edition of the WHO classification of odontogenic tumors defines juvenile (aggressive) ossifying fibroma as an actively growing lesion consisting of cell rich fibrous tissue containing bands of cellular osteoid without osteoblastic rimming together with trabeculae of more typical bone. Giant cells may also be present.

Classification

It is the term used to describe two distinct histopathologic variants of ossifying fibroma of the craniofacial skeleton –

- psammomatoid juvenile ossifying fibroma
- trabecular juvenile ossifying fibroma

Juvenile active ossifying fibroma affects predominantly patients in the first two decades of life. The mean age of occurrence being 3 to 23 yrs. No significant sexual predilection is seen in any of the two forms. Psammomatoid juvenile ossifying fibroma occurs overwhelmingly in the sinonasal and orbital bones of the skull, whereas trabecular juvenile ossifying fibroma is predominantly a gnathic lesion affecting the jaws, with a predilection for maxilla. In the mandible, the tumor occurs more commonly in the ramus than in the
body of mandible. The JOF is often characterized by a progressive and sometimes rapid expansion of the affected area. The clinical diagnostic characteristics suggestive of JOF are the patient's age, rapid increase in lesion size and absence of pain, paresthesia and bruit. When the orbital bones and paranasal sinuses are involved, the patients may develop proptosis, exophthalmos, and bulbar displacement. Rarely, intracranial extension has resulted in meningitis. The lesion exhibits a primary radiolucent quality with varying amounts of internal radiopacity, reflecting degree of mineralization. Some lesions contain numerous uniform, round, often laminated structures described as ossicles or psammoma-like bodies. Foci of multinucleated giant cells may also be present.

References


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