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University of Baghdad
College of Dentistry

Fibro-osseous Lesions

A Project Submitted to

The College of Dentistry, University of Baghdad, Department of Oral Diagnosis in Partial Fulfillment for the Bachelor of Dental Surgery

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(Oral Pathology)

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بسم الله الرحمن الرحيم

(يُؤْتِى ٱلْحِكْمَةَ مَن يَشَآءُ ۚ وَمَن يُؤْتَ ٱلْحِكْمَةَ فَقَدْ أُوتِىَ خَيْرًا كَثِيرًا ۗ وَمَا يَذَّكَّرُ إِلَّآ أُو۟لُوا۟ ٱلْأَلْبَٰبِ)

صدق الله العظيم

(البقرة:269)

Supervisor's Declaration

This is to certify that the organization and preparation of this graduation project have been made by the undergraduate student: **Sara Jdeea** under my supervision in the College of Dentistry, University of Baghdad / Department of oral diagnosis.

Signature

Assist Prof. Dr . Layla Sabri

Date: / / 2022

We, the members of the discussion committee, certify that we have read and examined this graduation project and that in our opinion it meets the standard of a graduation project.

Signature Signature Signature

A proved by the head of oral diagnosis department at the college of dentistry, university of Baghdad.

Signature

Dedication

This work is dedicated to my beloved parents
who have been my source of
inspiration and gave me strength when
I thought of giving up, who
continually provide moral, spiritual and
emotional support.

To my sisters and my brothers and
my faithful friends
who always gave me the words of advice
and encouragement to finish this work.

Acknowledgment

My faithful thanks to ALLAH for granting me the will and strength with which this research was accomplished.

I wish to express my sincere appreciation to my supervisor, Dr. Layla sabri , who has guided and encouraged me to be professional and do the right thing. Without her persistent help, patience and motivation the goal of this project would not have been realized.

I wish to show my gratitude to the person who taught me the meaning of life my beloved and supportive mother.

To the one I miss with all my heart, to the one who left our world, and the sound of his advice still guides me. To the symbol of devotion and sincerity, which fate did not allow to see me as he dreamed and died in the prime of youth .... my father

To the one who gave my happiness and comfort over her happiness.. to the one who taught me to give, and showered me with her tenderness and generosity.. to the source of goodness, sacrifice and altruism... my mother.

To the example of giving, pride and sacrifice, my support in this life... my brothers

To those who rely on them in every small and large, who have good and pure hearts... my sisters

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Introduction

Fibro-osseous lesions are a diverse group of processes that are characterized by replacement of normal bone by fibrous tissue containing a newly formed mineralized product. The term fibro-osseous lesion is descriptive and does not constitute a specific diagnosis. Lesions belonging to this category may be developmental (hamartomatous), reactive, dysplastic, or neoplastic **(de Noronha Santos Netto J *et al.*,2013).**

Fibro-osseous lesions of the jaws include the following:

• Fibrous dysplasia

• Cemento-osseous dysplasia

(a) Focal cemento-osseous dysplasia

(b) Periapical cemento-osseous dysplasia

(c) Florid cemento-osseous dysplasia

• Ossifying fibroma **(Eversole R *et al*., 2008)**

Although these conditions differ in etiology, they may exhibit very similar histopathologic features. Therefore, correlation of the histopathologic findings with the clinical and radiographic features typically is essential for establishing a specific diagnosis. (However, in some cases of cementoosseous dysplasia, a presumptive diagnosis may be made based on the clinical and radiographic findings.) A specific diagnosis is critical because the treatment, biologic behavior, and prognosis of these pathoses vary greatly. Some fibroosseous lesions only require monitoring, whereas others necessitate surgical recontouring or complete removal **(Worawongvasu R and Songkampol K,2010).**

Fibrous dysplasia

Fibrous dysplasia is a developmental tumorlike condition, characterized by replacement of normal bone by a proliferation of cellular fibrous connective tissue with irregular bony trabeculae. This sporadic condition results from postzygotic, activating mutations in the GNAS gene, which encodes the alpha subunit of a stimulatory G protein. Such mutations have not been detected in ossifying fibroma or cemento-osseous dysplasia **(Akintoye SO *et al*.,2013).**

Clinically, fibrous dysplasia may involve one bone or multiple bones; in some cases, involvement of multiple bones may occur in conjunction with cutaneous and endocrine abnormalities. The extent of disease depends on when the GNAS mutation occurs. During early embryonic development, mutation of a pluripotent stem cell can cause abnormalities in multiple cell types, including osteoblasts, melanocytes, and endocrine cells **(Chapurlat RD and Orcel P,2008).**

In contrast, if the mutation occurs in a skeletal progenitor cell in a later stage of embryonic development, then only osteoblasts will be affected. Alternatively, if the mutation occurs during postnatal life, then osteoblasts in only a single bone will be affected. Furthermore, the parental origin of the mutated GNAS allele may affect the phenotype, because in certain cell types (such as pituitary somatotrophs) genomic imprinting results in expression of only the maternal allele **(Cheng J *et al*.,2013).**

Clinical and Radiographic Features of fibrous dysplasia

Monostotic Fibrous Dysplasia: About 70% to 85% of patients with fibrous dysplasia have disease limited to a single bone (monostotic fibrous dysplasia). Monostotic fibrous dysplasia is diagnosed most often during the second and third decades of life. Males and females are affected with about equal frequency. Commonly involved sites include the craniofacial bones, ribs, femur, and tibia **(Zillo Martini M *et al*.,2010).**

Among cases involving the jaws, the maxilla is affected more often than the mandible. There is a predilection for the posterior region. Although mandibular lesions are truly monostotic, maxillary lesions often extend to involve adjacent bones (e.g., zygoma, sphenoid, ethmoid, frontal bone, temporal bone, occiput) in which case the term craniofacial fibrous dysplasia is appropriate. Painless, unilateral swelling is the most common clinical finding (Fig.1) . Growth is generally slow, and it is common for the patient to be aware of the condition for several years before seeking professional evaluation. Occasionally, however, the growth may be fairly rapid. Adjacent teeth may be displaced by the bony mass but usually remain firm **(Summerlin DJ and Tomich CE,1994).**

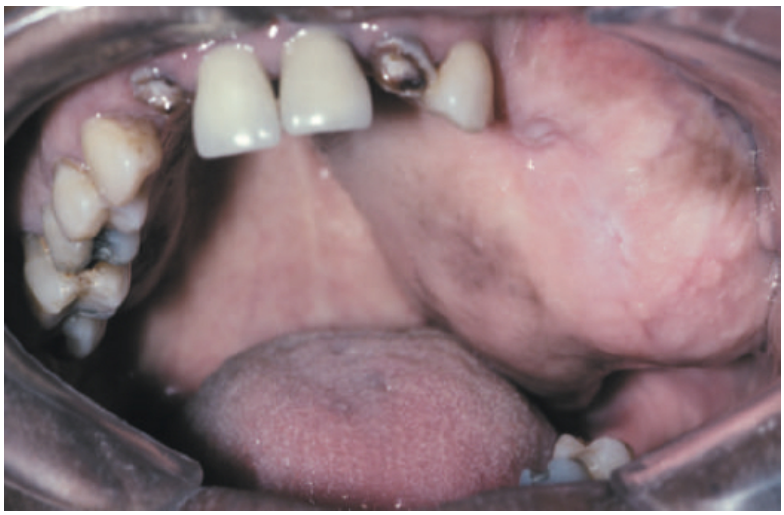


Fig (1) Fibrous Dysplasia. Expansile mass of the left maxilla in a 45-year-old woman. This lesion was known to have been present for at least 20 years **(Chapurlat RD and Orcel P,2008).**

The classic radiographic finding is a fine “ground-glass” opacification with poorly defined margins (Figs. 2 through 4). However, some lesions may appear radiolucent or mixed radiolucent-radiopaque. Mandibular lesions often exhibit buccolingual expansion and bulging of the inferior border. There may be superior displacement of the inferior alveolar canal. Periapical radiographs of the adjacent dentition may demonstrate narrowing of the periodontal ligament space and an ill-defined lamina dura that blends with the abnormal bone. Maxillary lesions often cause superior displacement of the sinus floor and obliteration of the antrum. In addition, extensive skull involvement may be evident (Fig. 5). Bone scintigraphy may aid in determining the extent of involvement and ruling out polyostotic disease **(Zillo Martini M *et al*.,2010).**

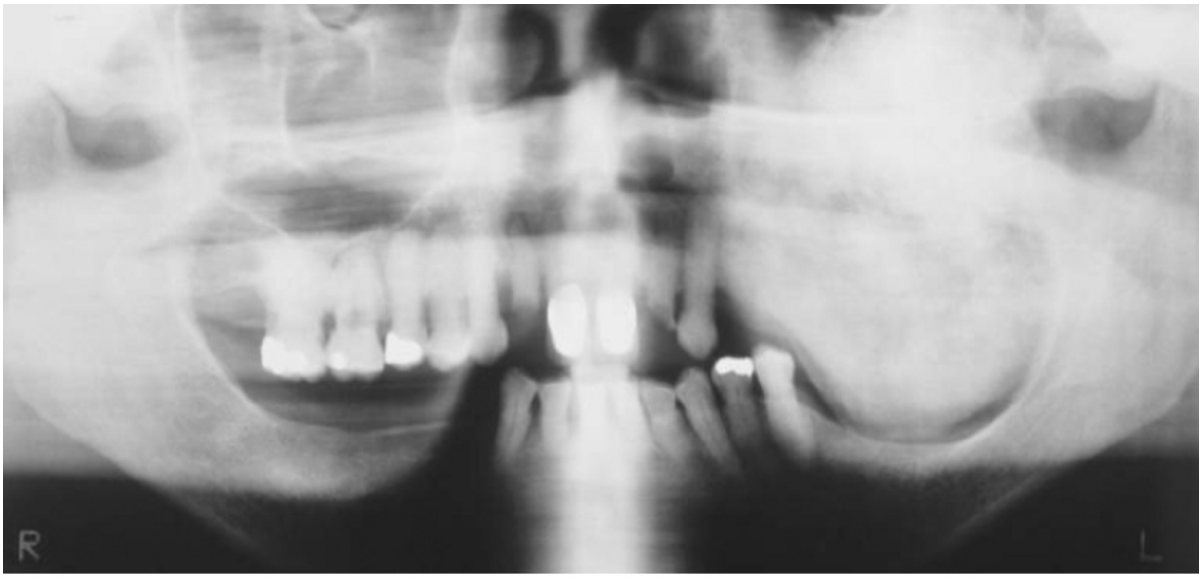


Fig (2) Fibrous Dysplasia. Panoramic radiograph of the patient shown in Fig (1) A diffuse “ground-glass” radiopacity is evident **(Chapurlat RD and Orcel P,2008).**

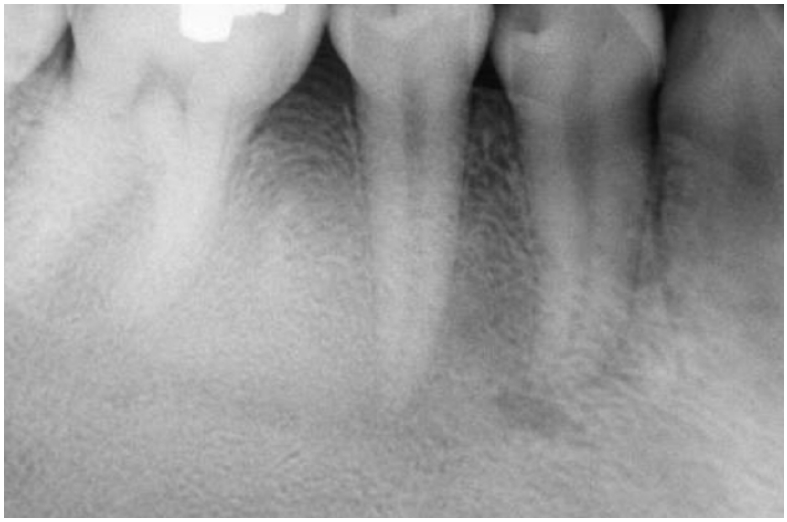


Fig (3) Fibrous Dysplasia. Periapical radiograph showing a diffuse “ground-glass” radiographic appearance **(Zillo Martini M *et al*.,2010).**

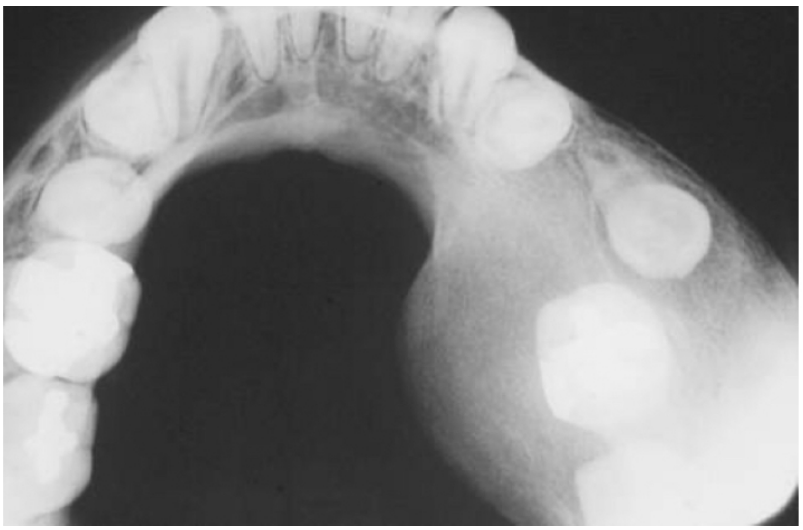


Fig (4) Fibrous Dysplasia. Occlusal radiograph showing localized expansion of the mandible and the “ground-glass” radiographic appearance. The margins of the lesion are not well defined and blend into the adjacent bone **(Waldron CA and Giansanti JS,1973).**

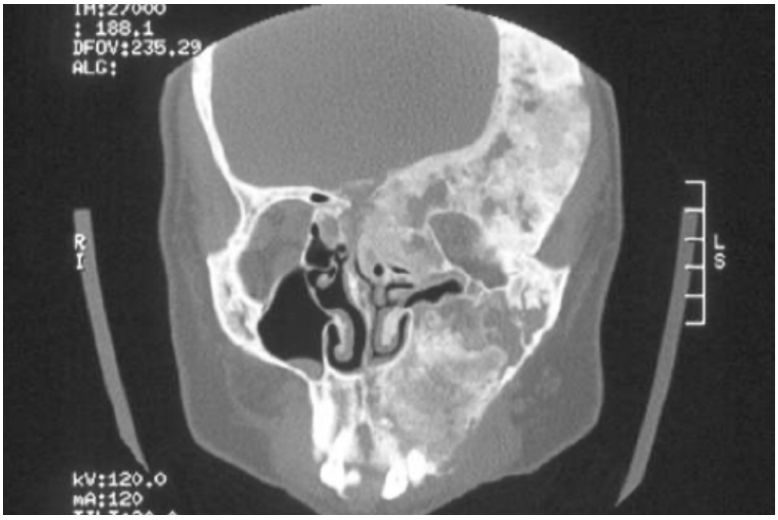


Fig (5) Fibrous Dysplasia. Computed tomography (CT) image showing extensive involvement of the maxilla and skull **(Zillo Martini M *et al*.,2010).**

Polyostotic Fibrous Dysplasia; Jaffe-Lichtenstein Syndrome; MCCune-Albright Syndrome

A minority of patients with fibrous dysplasia exhibits involvement of two or more bones (polyostotic fibrous dysplasia). Most patients with polyostotic disease are diagnosed before 10 years of age, and there is a female predilection. The number of involved bones varies from a few to 75% of the entire skeleton **(Chapurlat RD and Orcel P,2008).**

Presenting signs and symptoms typically are related to long bone involvement and include pain, pathologic fracture, limping, leg length discrepancy, and bowing deformity. Radiographic examination may reveal malformation of the proximal femur (known as coxa vara, shepherd's crook deformity, or hockey stick deformity). Involvement of the skull and jaws may result in facial asymmetry (Fig.6).

Craniofacial involvement may cause vision changes, hearing impairment, sinonasal congestion, and airway obstruction. Hypophosphatemia caused by renal phosphate wasting is a fairly common finding, which appears to be related to the release of fibroblast growth factor 23 (FGF23) by the affected bones **(Akintoye SO *et al*.,2013).**

A small subset of patients may exhibit polyostotic fibrous dysplasia in association with the following syndromes:

• Jaffe-Lichtenstein syndrome, characterized by polyostotic fibrous dysplasia and café au lait (coffee with milk) pigmentation

• McCune-Albright syndrome, characterized by polyostotic fibrous dysplasia, café au lait pigmentation, and multiple endocrinopathies

• Mazabraud syndrome, characterized by fibrous dysplasia and intramuscular myxomas **(Zillo Martini M *et al*.,2010).**

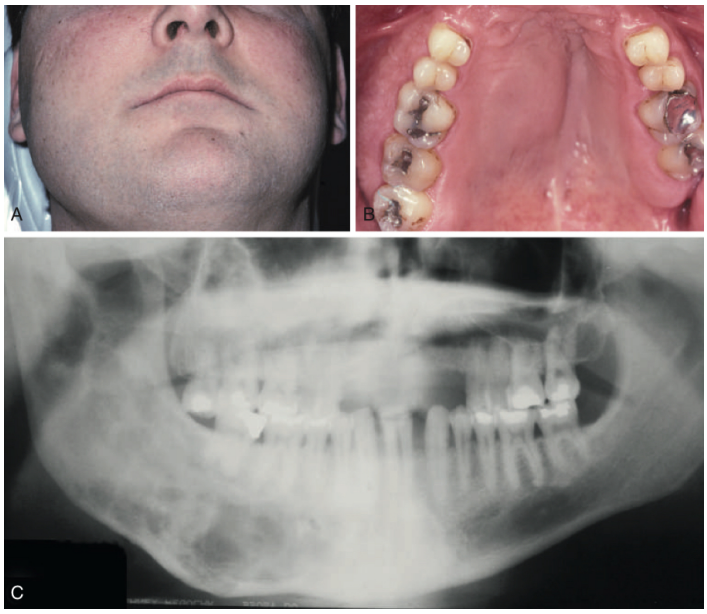


Fig (6) Polyostotic Fibrous Dysplasia. Jaffe-Lichtenstein syndrome. A, Young man exhibiting enlargement of the right maxilla and mandible. B, Intraoral photograph showing unilateral maxillary expansion. C, Panoramic radiograph showing ill-defined lesions of the right side of both jaws **(Zillo Martini M *et al*.,2010).**

Histopathologic Features of fibrous dysplasia

Fibrous dysplasia consists of a slight to moderate cellular fibrous connective tissue stroma that contains foci of irregularly shaped trabeculae of immature bone (fig.7). The bone trabeculae assume irregular shapes linked to Chinese characters and they do not display any functional orientation, without osteoblastic activity at the bone trabeculae margins **(Odell, Edward W,2015).**

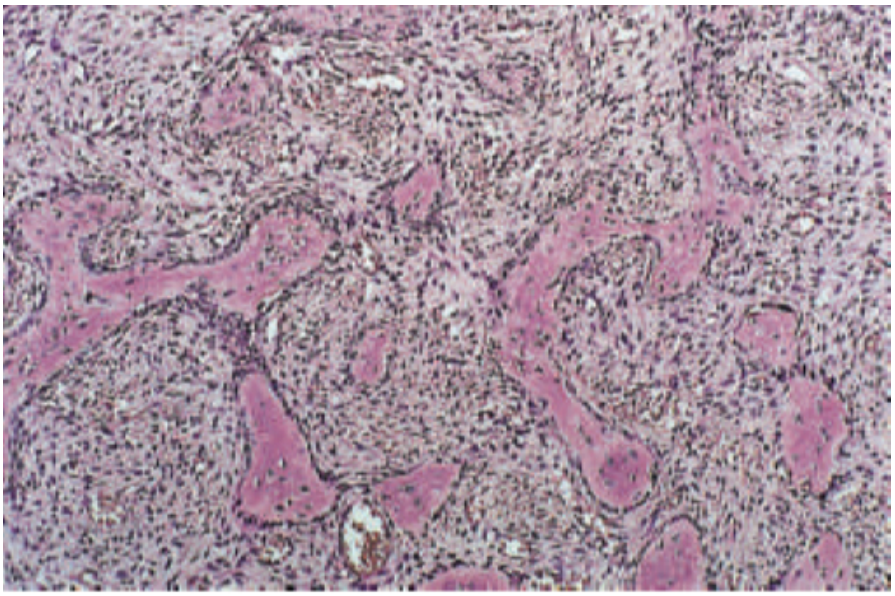


Fig (7) Fibrous dysplasia. Slender trabeculae of woven bone, said to resemble Chinese characters in shape, lying in a very cellular fibrous tissue. With maturation there is progressively more bone formation **(Odell, Edward W,2015).**

Treatment and Prognosis of fibrous dysplasia

Fibrous dysplasia tends to stabilize upon skeletal maturation, and spontaneous regression even has been reported in a few cases. Therefore, conservative management is preferred. Some lesions, nevertheless, exhibit continued growth into adulthood. The risk for severe deformity and complications is particularly elevated among patients with widespread polyostotic fibrous dysplasia—especially in the setting of McCune-Albright syndrome with uncontrolled growth hormone excess **(Akintoye SO *et al*.,2013).**

Patients with minimal cosmetic and functional disturbances may not require surgical treatment. For young patients with significant problems due to large or extensive lesions, surgical contouring, shaving, or other debulking procedures may be performed. However, subsequent regrowth may require additional surgery. Approximately 20% to 50% of patients show some regrowth after surgical debulking, and the risk for regrowth is greater among younger than older patients. Therefore, if possible, many authorities prefer to delay surgery until the disease is quiescent. Some investigators have proposed that serum alkaline phosphatase levels after incomplete surgical removal may be predictive of disease progression, although validation studies are needed **(Chapurlat RD and Orcel P,2008).**

Alternatively, complete surgical removal may be considered in some cases, such as monostotic lesions, very aggressive lesions, or lesions refractory to repeated debulking. Combined orthodontic treatment and orthognathic surgery may be performed to correct malocclusion. Successful placement of dental implants has been reported in a few cases, but additional studies are needed. Several reports suggest that bisphosphonates (e.g., IV pamidronate, oral alendronate) may help to relieve bone pain in fibrous dysplasia. However, well-designed studies are needed to confirm these findings, to assess the potential for inducing disease stabilization, and to evaluate the long-term safety of such treatment in young patients **(Neville, Brad W,2009).**

CEMENTO-OSSEOUS DYSPLASIAS (OSSEOUS DYSPLASIA)

Cemento-osseous dysplasia occurs in the tooth-bearing areas of the jaws and is probably the most common fibro-osseous lesion encountered in clinical practice. Because the histopathologic features share many similarities with fibrous dysplasia and ossifying fibroma, correct diagnosis can be problematic but is critical for appropriate management **(Alsufyani NA and Lam EWN,2011).**

Some investigators have suggested that cemento-osseous dysplasia originates from the periodontal ligament, because of microscopic similarity and lesion proximity to this structure. Others believe this condition represents a defect in extraligamentary bone remodeling that may be triggered by local injury or, possibly, an underlying hormonal imbalance **(Groot RH *et al*.,1996).**

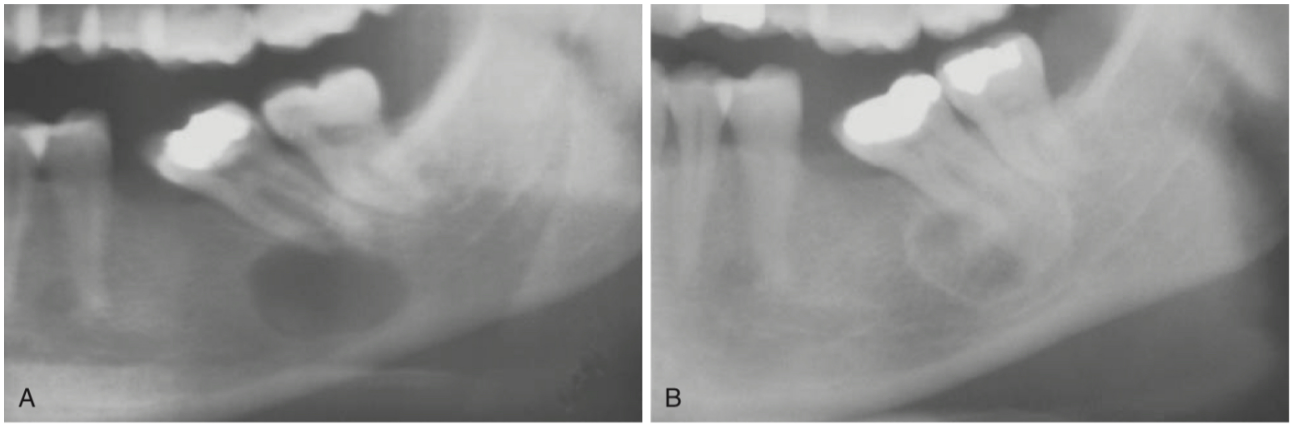
Clinical and Radiographic Features of cemento-osseous dysplasia

Based on clinical and radiographic features, cementoosseous dysplasia includes the following variants: (1) focal, (2) periapical, and (3) florid **(MacDonald-Jankowski DS,2003).**

Focal Cemento-Osseous Dysplasia

Focal cemento-osseous dysplasia involves a single site. Before the concept of focal cemento-osseous dysplasia was clarified in the mid-1990s, most cases were misdiagnosed as a variant of ossifying fibroma. About 90% of cases of focal cemento-osseous dysplasia occur in females, with an approximate mean age of 41 years and a predilection for the third to sixth decades. The lesion has been reported across ethnic groups—most often American blacks followed by East Asians and whites. In contrast to the periapical and florid variants, the focal variant seems to affect a greater proportion of whites, although this finding may be due to study population bias **(MacDonald-Jankowski DS,2008).**

Focal cemento-osseous dysplasia most commonly involves the posterior mandible. The disease typically is asymptomatic and is detected incidentally by radiographic examination. Most lesions are smaller than 1.5 cm in diameter. Radiographically, the lesion varies from completely radiolucent to densely radiopaque with a thin peripheral radiolucent rim. Most commonly, however, there is a mixed radiolucent and radiopaque pattern (Fig.8). The borders tend to be well defined but slightly irregular. The lesions typically occur around tooth apices or in extraction sites. A focal lesion occasionally may represent an early stage in the transition to multifocal involvement, especially in black females **(Mahomed F *et al*.,2005).**



- Fig (8) Focal Cemento-Osseous Dysplasia. A, A radiolucent area involves the edentulous first molar area and the apical area of the second molar. B, Radiograph of the same patient taken 9 years later showing a mixed radiolucent and radiopaque pattern **(Mahomed F *et al*.,2005).**

Periapical Cemento-Osseous Dysplasia (Osseous Dysplasia; Periapical Cemental Dysplasia; Periapical Cementoma)

Periapical cemento-osseous dysplasia predominantly involves the periapical region of the anterior mandible. Solitary lesions may occur, but multiple foci typically are present. There is a marked female predilection (female-to male ratio ranging from 10 : 1 to 14 : 1), and approximately 70% of cases affect blacks. Most patients are diagnosed initially between 30 and 50 years of age, with the diagnosis almost never made in individuals younger than 20 years. The associated teeth are usually vital and seldom have restorations **(MacDonald-Jankowski DS,2008).**

Periapical cemento-osseous dysplasia is an asymptomatic condition that often is discovered when radiographs are taken for other purposes. Early lesions appear as circumscribed periapical radiolucencies, similar to periapical granulomas or periapical cysts (Fig.9). Adjacent lesions may fuse to form a linear radiolucency that envelops the apices of several teeth (Fig.10). Over time, the lesions tend to “mature” and become mixed radiolucent-radiopaque (Fig.11). In the end stage, the lesions appear as circumscribed, dense radiopacities surrounded by narrow radiolucent rims. The periodontal ligament space usually appears intact, and fusion to the tooth is rare.

Most lesions are nonexpansile with self-limiting growth; individual lesions seldom exceed 1.0 cm in diameter **(MacDonald-Jankowski DS,2003).**

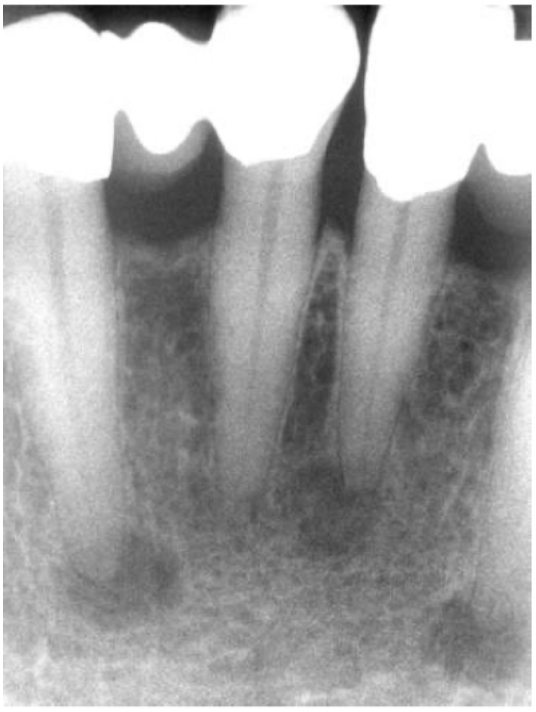


Fig (9) Periapical Cemento-Osseous Dysplasia. Periapical radiograph showing multiple radiolucent lesions at the apices of the anterior mandibular teeth **(MacDonald-Jankowski DS,2003).**

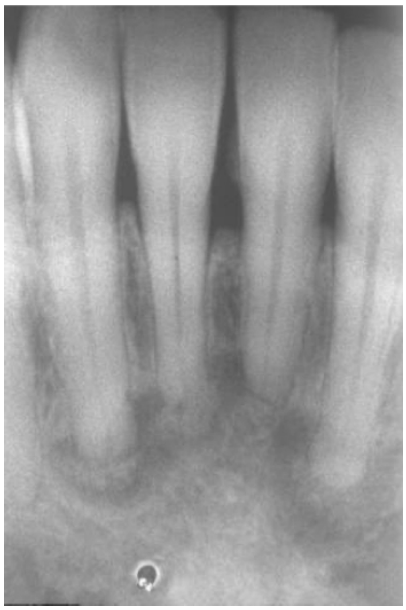


Fig (10) Periapical Cemento-Osseous Dysplasia. Later stage lesions exhibiting significant mineralization **(Neville, Brad W,2009).**

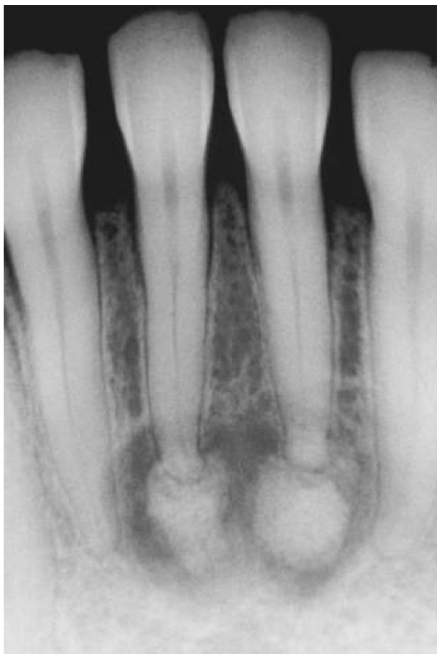


Fig (11) Periapical Cemento-Osseous Dysplasia. Later stage lesions exhibiting significant mineralization **(Neville, Brad W,2009).**

Florid Cemento-Osseous Dysplasia

Florid cemento-osseous dysplasia exhibits multifocal involvement not limited to the anterior mandible. Although many cases affect only the posterior portions of the jaws, synchronous involvement of the anterior mandible may be observed as well (Fig.12). Like the periapical pattern, this form predominantly affects black females (in some series, more than 90% of patients), with a marked predilection for middle-aged to older adults. An intermediate frequency among East Asian populations also has been described **(Schneider LC and Mesa ML,1990).**

The lesions show a tendency for bilateral and fairly symmetrical involvement of the mandible, and occasionally there may be extensive involvement in all four quadrants. At times the disease may be asymptomatic and discovered only when radiographs are taken for unrelated reasons. In other cases, patients may have dull pain, alveolar sinus tracts, and exposure of yellowish, avascular bone to the oral cavity (Fig. 13). Although rarely prominent, some jaw expansion may be evident **(Su L *et al*.,1997).**

Radiographically, the lesions demonstrate a maturation pattern similar to that noted in the other forms of cemento-osseous dysplasia. Initially, the lesions are predominantly radiolucent but with time become mixed, then predominantly radiopaque with only a thin radiolucent rim (Fig. 14). On occasion, a lesion can become almost totally radiopaque and blend with the adjacent normal appearing bone. Typically, the radiopacities remain separated from adjacent teeth with an intervening, intact periodontal ligament space. However, in some end-stage lesions, the cemento-osseous material may fuse with the tooth root surface to produce thickened root apices surrounded by radiolucency (or a “hypercementosis-like” appearance).

Both dentulous and edentulous areas may be affected, and involvement appears to be unrelated to the presence or absence of teeth. Sharply defined radiolucent areas, which on surgical exploration prove to be simple bone cysts , may be intermixed with the other lesional elements. Investigators have suggested that these simple bone cysts

may result from interstitial fluid obstruction by the fibro-osseous proliferation **(Larrea-Oyarbide N *et al*.,2008).**

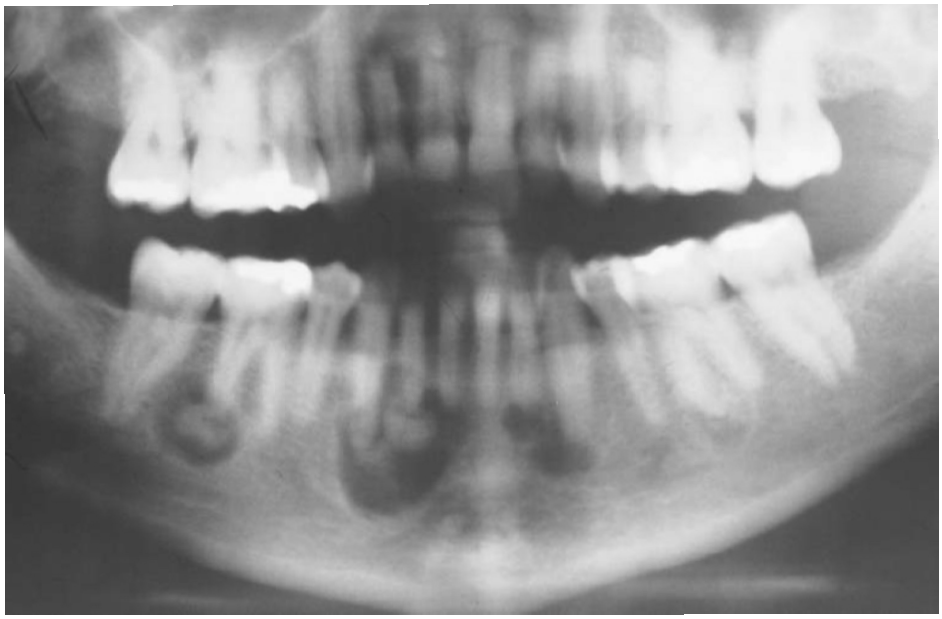


Fig (12) Florid Cemento-Osseous Dysplasia. Multiple mixed radiolucent and radiopaque lesions involving the anterior and posterior regions of the mandible **(Larrea-Oyarbide N *et al*.,2008).**

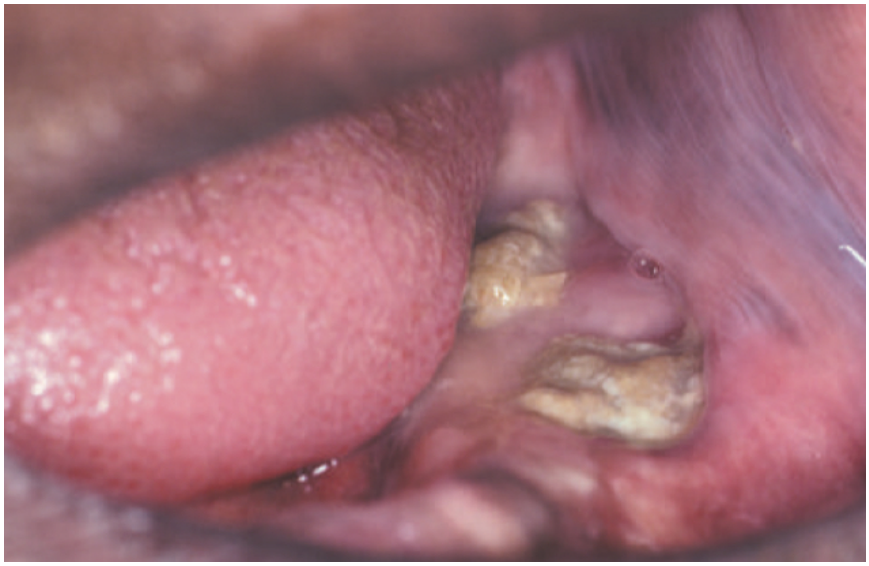


Fig (13) Florid Cemento-Osseous Dysplasia. Yellowish, avascular cementum-like material is beginning to exfoliate through the oral mucosa **(Neville, Brad W,2009).**

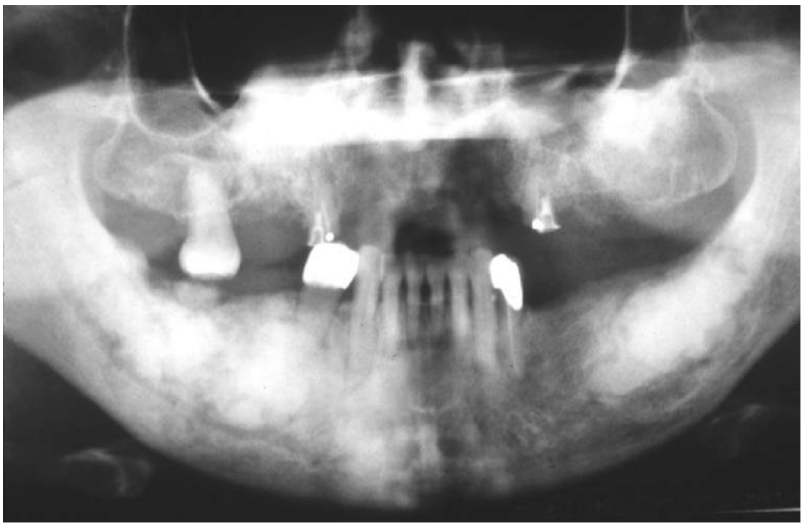


Fig (14) Florid Cemento-Osseous Dysplasia. Multifocal radiopaque lesions of the posterior areas of the jaws **(Neville, Brad W, 2009).**

Histopathologic Features of cemento-osseous dysplasia

All three patterns of cemento-osseous dysplasia demonstrate similar histopathologic features. There are typically fragments of cellular fibrovascular connective tissue with scattered hemorrhage and a variable mixture of woven bone, lamellar bone, and cementum-like particles (Figs. 15 and 16). As the lesions mature, the ratio of fibrous connective tissue to mineralized material decreases **(Melrose RJ, 1997).** Over time, the bony trabeculae become thick and curvilinear, with shapes likened to ginger roots. In the final radiopaque stage, both lesion types demonstrate a mixture of bone and cementum-like particles, although subtle histopathologic differences may be appreciated. The bony trabeculae in ossifying fibroma tend to be more delicate and show more prominent osteoblastic rimming compared to those in cemento-osseous dysplasia. Also, the cementum-like particles in cemento-osseous dysplasia are irregularly shaped and often exhibit retraction from the adjacent stroma, whereas those in ossifying fibroma are more ovoid and often demonstrate brush borders in intimate association with the adjacent stroma. Although ossifying fibroma can exhibit peripheral hemorrhage, cemento-osseous dysplasia typically reveals hemorrhage throughout the lesion and sinusoidal vascularity in close association with the bony trabeculae **(Noffke CE and Raubenheimer EJ, 2011).**

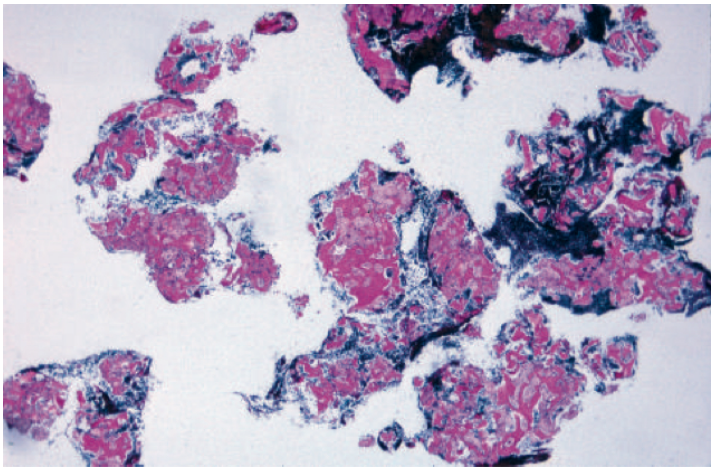


Fig (15) Cemento-Osseous Dysplasia. Low-power photomicrograph showing fragments of cellular fibrous connective tissue containing scattered trabeculae of bone **(Neville, Brad W, 2009).**

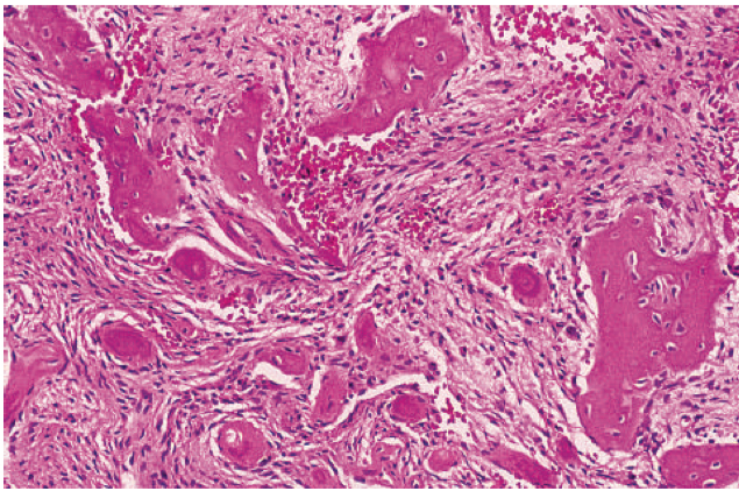


Fig (16) Cemento-Osseous Dysplasia. High-power photomicrograph showing spicules of bone and cementum-like hard tissue within moderately cellular fibrous connective tissue. Note the hemorrhage around the bony trabeculae **(Neville, Brad W,2009).**

Treatment of cemento-osseous dysplasia

Cemento-osseous dysplasia does not appear to be neoplastic and, therefore, generally does not require removal. During the predominantly radiolucent phase, the lesions cause few problems. However, in the sclerotic phase, the lesions tend to be hypovascular and prone to necrosis and secondary infection with minimal provocation. For the asymptomatic patient, the best management consists of regular recall examinations with prophylaxis and oral hygiene reinforcement to control periodontal disease and prevent tooth loss **(MacDonald-Jankowski DS,2003).**

FAMILIAL GIGANTIFORM CEMENTOMA

This extremely rare autosomal dominant condition causes extremely large and disfiguring lesions, usually in all quadrants and presenting in childhood. In at least some cases, the origin appears to be at the tooth roots. Lesions grow progressively and recur after incomplete removal, so that it is unclear whether the condition is one of multiple cemento-ossifying fibromas or a type of cemento-osseous dysplasia. The term gigantiform cementoma must not be used for florid cemento-osseous dysplasia with expansion, and is best reserved for the inherited condition in which the multiple tumours reach 10 or 20 cm in diameter. Conventional ossifying fibromas can be multiple and reach a large size, but not all such cases are gigantiform cementoma **(Cawson, R A, and E W. Odell ,2008).**

Clinical and Radiographic Findings of familial gigantiform cementoma

Unlike florid cemento-osseous dysplasia, familial gigantiform cementoma exhibits neither a predilection for blacks nor a significant gender predilection. Although blacks may be affected, most reported families are white or Asian **(Abdelsayed RA *et al*.,2001).**

Radiographic alterations may begin to develop during the first decade of life. By adolescence, most patients exhibit clinically obvious expansion of the jaws (Fig.17). The lesions affect multiple quadrants, often with simultaneous involvement of the maxilla and mandible. Lesion growth may be rapid or slow. In a few reported cases, especially rapid growth has been noted during pregnancy. Although the course is variable, many patients develop significant facial deformity, s well as impaction, malposition, and malocclusion of the involved dentition. If not treated, then the osseous enlargement eventually ceases during the fifth decade **(Kumar VV *et al*.,2012).**

Radiographically, the lesions initially may appear as multiple periapical radiolucencies, resembling cemento-osseous dysplasia. 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 lesions become predominantly radiopaque but often maintain a thin radiolucent rim. As noted in cemento-osseous dysplasia, the affected bone during the final radiopaque stage is very sensitive to inflammatory stimuli and becomes necrotic with minimal provocation **(Moshref M *et al*.,2008).**

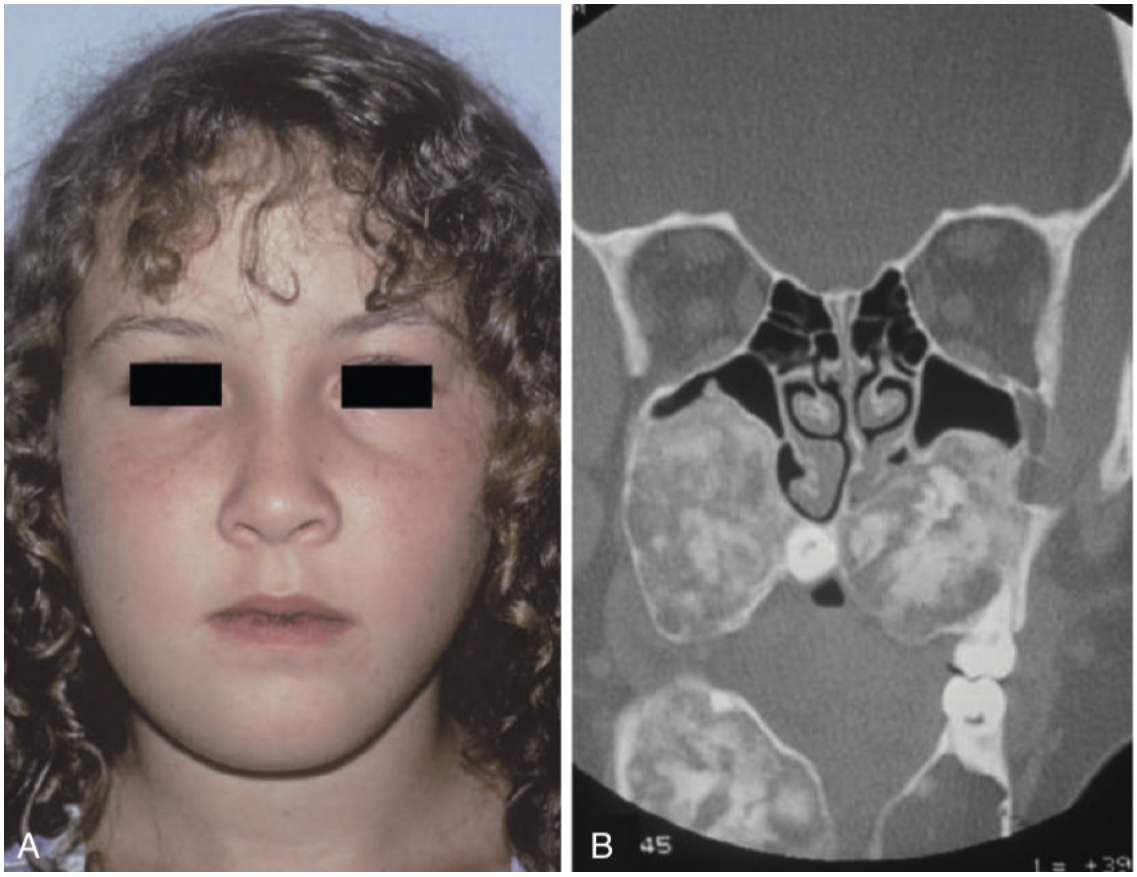


Fig (17) Familial Gigantiform Cementoma. Young woman with massive lesions involving all four quadrants of the jaws **(Abdelsayed RA *et al*.,2001).**

Histopathologic Features of familial gigantiform cementoma

Histopathologically, familial gigantiform cementoma shows the same spectrum of changes seen in florid cementoosseous dysplasia, and the two cannot be distinguished microscopically **(Toffanin A et al.,2000).**

Treatment and Prognosis of familial gigantiform cementoma

Before the final sclerotic stage, shave-down surgical procedures typically are unsuccessful because the dysplastic tissue rapidly regrows. Once the lesions are predominantly radiopaque, partial removal may lead to sequestration of the remaining affected bone. Therefore, if feasible, complete resection and reconstruction are recommended. Because familial gigantiform cementoma might be associated with polypoid adenomas of the uterus, gynecologic evaluation is prudent for female patients—especially those with anemia **(Tsutsumi S *et al*.,2004).**

Ossifying Fibroma

Ossifying fibroma is a benign neoplasm of bone that has the potential for excessive growth, bone destruction & recurrence.

Clinically & microscopically similar to cementifying fibroma, it is composed of a fibrous connective tissue stroma in which new bone is formed. Ossifying fibroma is a true neoplasm with a significant growth potential. Recently, mutations in a tumor suppressor gene were identified **(Akcam T *et al*.,2012).**

Clinical Features of ossifying fibroma

The epidemiology of Ossifying fibroma is unclear because many previous diagnosed cases were confused with focal cementoosseous dysplasia (COD). For that reason what was thought to be ossifying fibroma, a common neoplasm, is now considered to be uncommon because most of the cases were in reality focal COD. tends to occur during the 3rd & 4th decades of life, in females more than in males. It is a slow growing asymptomatic & expansile lesion.ossifying fibroma may be seen in the jaw & craniofacial bones. Lesions in the jaw arise in the tooth-bearing region, mostly in the molar & premolar area. The tumor may cause expansion of the buccal and lingual cortical plates (Fig18), however perforation is very rare.Ossifying fibroma is mostly a solitary lesion, although multiple lesions have been reported **(de Mesquita Netto AC *et al*.,2013).**

Radiographic Findings of ossifying fibroma

Well circumscribed, sharply demarcated border is the most common presenting radiographic feature, although ossifying fibroma may present as relatively lucent or opaque depending on the density of the calcification present. Also they may be unilocular or multilocular, mixed radiolucent-radiopaque image may be seen. The roots of the teeth present may be displaced & less commonly resorption is seen **(McHugh JB *et al*.,2009).**

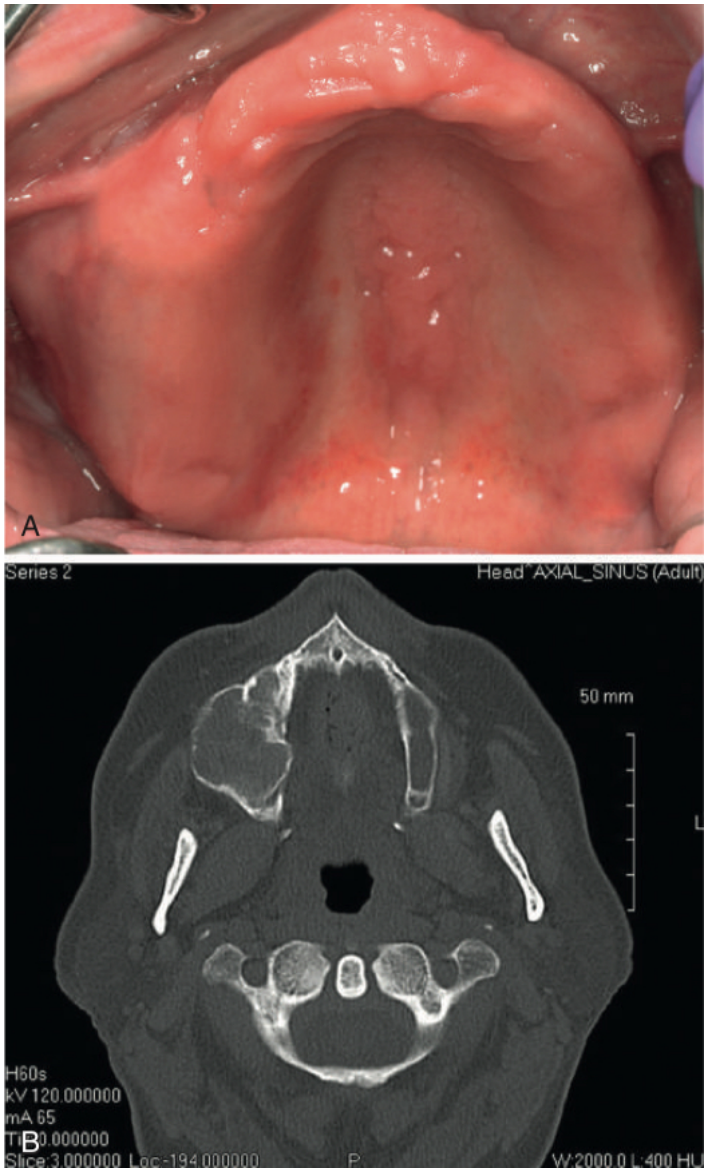


Fig (18) Ossifying Fibroma. Clinical image (A) and computed tomography (CT) scan (B) showing a large, expansile lesion of the posterior maxilla **(de Mesquita Netto AC *et al*.,2013).**

Histopathologic Features of ossifying fibroma

At surgery, the lesion tends to separate easily from the surrounding bone; thus, the tumor usually is submitted as one mass or a few large pieces (Fig. 19). Grossly and microscopically, most lesions are well demarcated but unencapsulated. However, a fibrous capsule may be present in some cases **(Woldenberg Y *et al*.,2005).** Microscopic examination shows cellular fibrous tissue with mineralized product (Fig. 20). The mineralized component may include a variable admixture of osteoid, bone, and basophilic acellular (or "cementum-like") spherules. The bony trabeculae vary in size and frequently demonstrate both woven and lamellar patterns. Peripheral osteoid and osteoblastic rimming are usually present. The cementum-like spherules often demonstrate brush borders that blend into the adjacent connective tissue (Fig. 21). Significant intralesional hemorrhage is unusual **(Akcam T *et al*.,2012).**

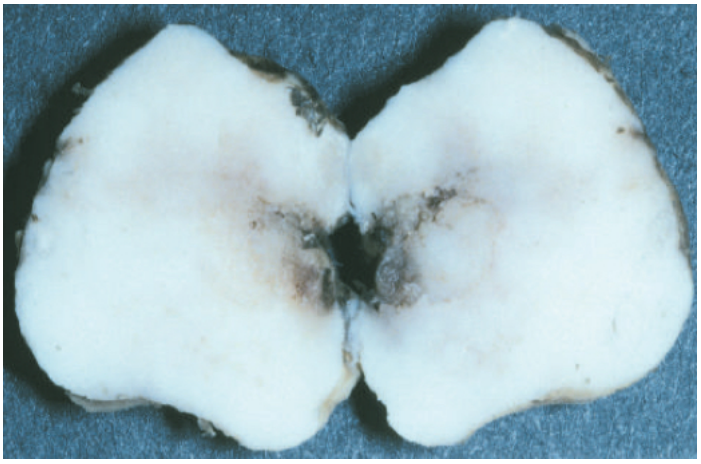


Fig (19) Ossifying Fibroma. Gross specimen showing a well circumscribed tumor that shelled out in one piece and subsequently was hemisected **(Woldenberg Y *et al*.,2005).**

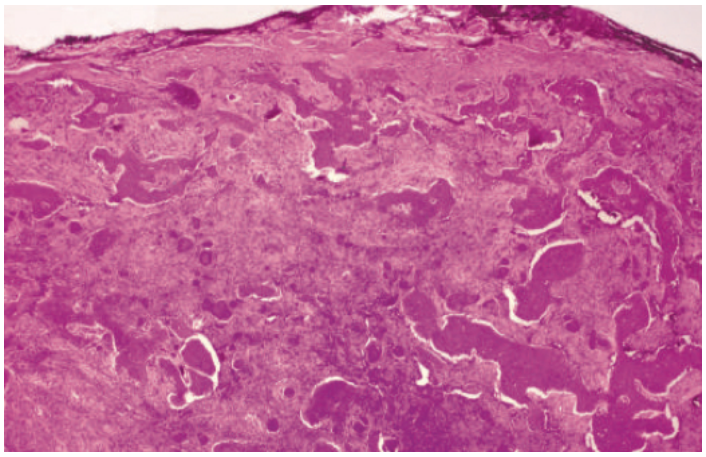


Fig (20) Ossifying Fibroma. This low-magnification photomicrograph shows a well-circumscribed solid tumor mass. Trabeculae of bone and droplets of cementum-like material can be seen forming within a background of cellular fibrous connective tissue **(Akcam T *et al*.,2012).**

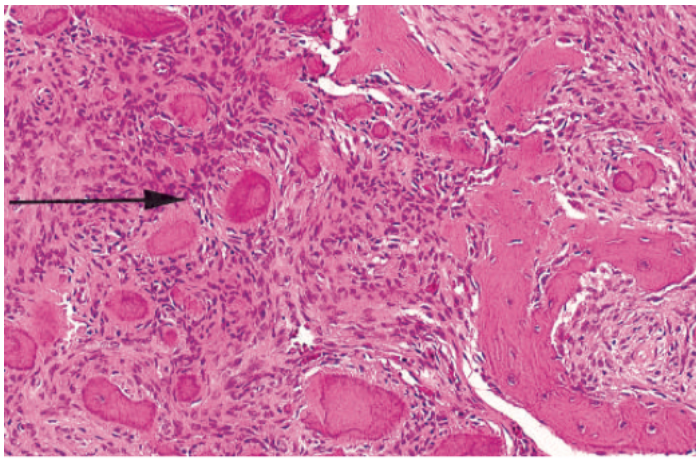


Fig (21) Ossifying Fibroma. High-power photomicrograph showing a mixture of woven bone and cementum-like material. Note the spherules demonstrating peripheral brush borders (arrow), **(Akcam T *et al*.,2012).**

Treatment and Prognosis of ossifying fibroma

The circumscribed nature of the ossifying fibroma generally permits enucleation of the tumor with relative ease. Large lesions that have caused considerable bone destruction may necessitate surgical resection and bone grafting. Recurrence after complete removal is uncommon; one systematic review of the literature has reported a 12% recurrence rate. Overall, the prognosis is very good, and there is no apparent potential for malignant transformation **(McHugh JB *et al*.,2009).**

JUVENILE OSSIFYING FIBROMA (JUVENILE ACTIVE OSSIFYING FIBROMA; JUVENILE AGGRESSIVE OSSIFYING FIBROMA)

Is a well circumscribed rapidly growing neoplasm lack the continuity with adjacent normal bone. Lesions are circumscribed radiolucencies in some cases contain central radio-opacities (Ground glass) opacification may be observed. Those are present within a sinus may appear radio dense and create a clouding that could be confused with sinusitis. Two different neoplasm have been reported: (1) Trabecular and (2) Psammomatoid. The latter neoplasm occur more than the trabecular type in a ratio of approximately 4:1 **(El-Mofty S,2002).**

Clinical and Radiographic Features of juvenile ossifying fibroma

Juvenile ossifying fibromas most often arise in children, adolescents, and young adults; however, a broader age range has been reported for the psammomatoid variant (3 months to 72 years) than the trabecular variant (2 to 33 years). Among various reported case series, the average age at diagnosis is somewhat younger for the trabecular variant (range from 8 2 1 to 12 years) than the psammomatoid variant (range from 16 to 33 years). Most authors report either a slight male predilection or no significant gender predilection **(Sarode SC *et al*.,2011).** The trabecular variant arises primarily in the jaws, whereas the psammomatoid variant predominantly

involves the paranasal sinuses and orbital region. In both variants, gnathic involvement favors the maxilla.

Radiographic examination typically shows a well circumscribed radiolucency or mixed radiolucency and radiopacity (Fig. 22). A sclerotic border may be evident in some cases. “Ground-glass” opacification or a multilocular “honeycomb” pattern also may be observed. Aggressive lesions often cause expansion and cortical thinning or perforation. Similar to conventional ossifying fibromas, juvenile ossifying fibromas may produce downward bowing of the inferior cortex of the mandible. Jaw lesions also can cause tooth displacement, root resorption, and failure of tooth development. Sinus involvement may appear radiographically as cloudy opacification mimicking sinusitis **(Urs AB *et al*.,2013).**

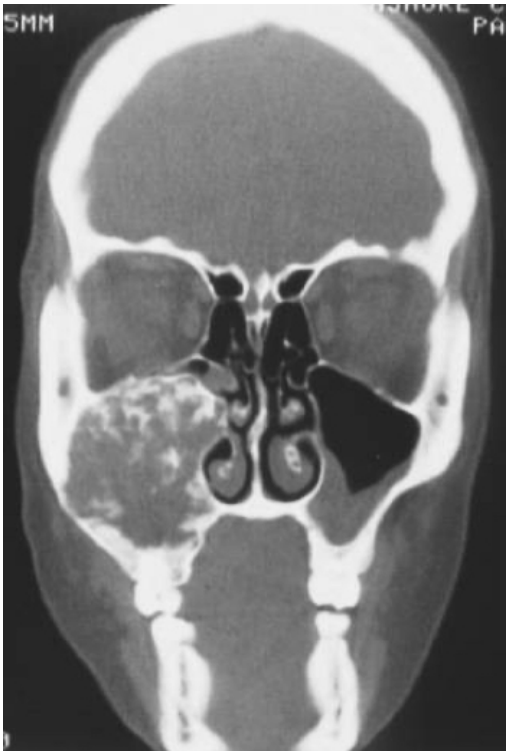


Fig (22) Juvenile Ossifying Fibroma. Computed tomography (CT) scan showing a large tumor involving the left maxilla and maxillary sinus of a 12-year-old girl. Clinically, the tumor was growing rapidly **(Urs AB *et al*.,2013).**

Histopathologic Features of juvenile ossifying fibroma

Both patterns are nonencapsulated but well demarcated from the surrounding bone. Tumors consist of cellular fibrous connective tissue with variants areas of loose and other are so cellular, mitotic figures are found but rare,areas of hemorrhage and small clusters of multinucleated giant cells are usually seen.

The trabecular type shows irregular strands of highly cellular osteoid encasing plump osteocytes (Fig 23). These starnds are lined by plump osteoblast and in other areas by giant cells.

In psammomatoid pattern concentric lamellated and spherical ossicles that have basophilic centers with peripheral eosinophilic osteoid rims **(Williams HK *et al*.,2000).**

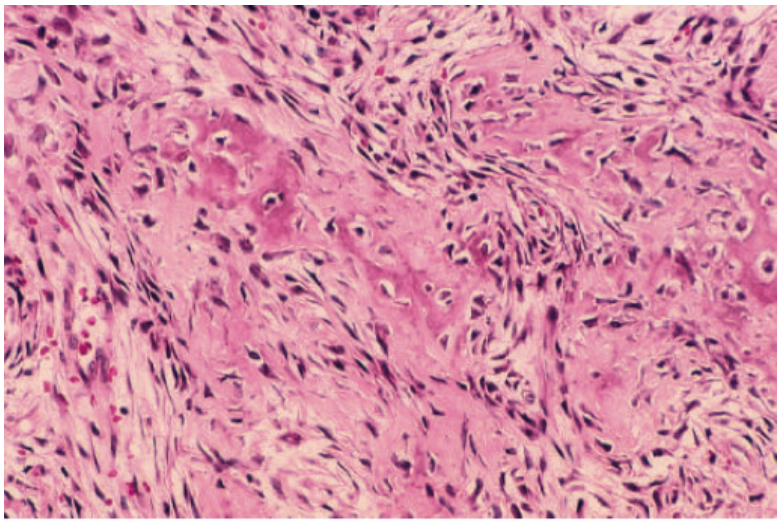


Fig (23) Juvenile Ossifying Fibroma, Trabecular Variant. Trabeculae of cellular woven bone are present in a cellular fibrous stroma **(Williams HK *et al*.,2000).**

Treatment and Prognosis of juvenile ossifying fibroma

For small lesions, complete local excision or thorough curettage appears adequate. For large or aggressive lesions, wider resection may be required.

In contrast to the negligible recurrence rate for conventional ossifying fibromas, recurrence rates of 30% to 58% have been reported for juvenile ossifying fibromas. Many reported recurrences actually may represent tumor persistence after incomplete surgical removal. Malignant transformation has not been documented. Tumor-related deaths are extremely rare and primarily result from complications caused by direct intracranial extension **(Slootweg PJ,2012).**

Conclusions

Fibro-osseous lesions are a poorly defined group of lesions affecting the jaws and craniofacial bones. All are characterized by the replacement of bone by cellular fibrous tissue containing foci of mineralization that vary in amount and appearance. Classification and, therefore, diagnosis of these lesions is difficult because there is significant overlap of clinical and histological features.

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