Imaging of Fibro-osseous Lesions and Other Bone Conditions of the Jaws

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KEYWORDS

• Fibro-osseous • Fibrous dysplasia • Cemento-osseous dysplasia
• Ossifying fibroma • Central giant cell granuloma • Aneurysmal bone cyst
• Langerhans cell histiocytosis • Paget's disease

KEY POINTS

- Pathophysiology of fibro-osseous and other bone lesions affecting the jaws can be dysplastic, reactive, or neoplastic growths.
- The lesions discussed show common histology that can be insufficient for a diagnosis.
- Specific radiographic presentations can be depicted in advanced imaging and contribute to adequate diagnosis and management.

INTRODUCTION

The term "fibro-osseous lesions" is an umbrella term that is used to describe the histopathological picture of certain lesions where normal bone is replaced by fibrous tissue and immature bone or cementum-like structures. The term was employed as the histopathological features of these entities are indistinguishable.[1] However, as per the definition, several other inflammatory, reactive, benign, and malignant lesions would fit the description. In an effort to understand these entities, several classifications and reclassifications have been introduced in the World Health Organization (WHO), textbooks, and published articles.[2]

A key player to reaching the diagnosis is the oral and maxillofacial radiologist (OMFR). Despite attempts by OMFRs to publish work on classification and diagnosis, knowledge gap remains and is evidenced by continued confusion and published case reports with unwarranted management or intervention.[3,4]

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Consistency in nomenclature and understanding of disease mechanisms among the dental specialists are crucial to ensure proper diagnosis and management.

Fibro-osseous lesions and other bone lesions affecting the jaws are a group of diverse conditions that can be partially similar in clinical, radiographic, or histopathologic presentations. This review aims to discuss these lesions with focus on radiographic features to avoid diagnostic pitfalls.

CLASSIFICATION

Fibro-osseous Lesions

Fibrous dysplasia

Fibrous dysplasia (FD) is a benign skeletal disorder where normal bone tissue is substituted with fibrous tissue and disorganized woven bone. FD is somatic in nature and can be attributed to mutation of the GNAS1 gene.[5] FD can be monostotic (80%–85%) or polyostotic, typically below the age of 30.[6,7] The jaw bones, frontal, sphenoid, and zygoma are the principal bones involved in the skull and facial region and the maxilla is the most affected gnathic bone.

Polyostotic FD is linked with McCune-Albright syndrome, a condition that is almost exclusive to females.[8] McCune-Albright syndrome exhibits café au lait skin pigmentation, precocious puberty, hyperthyroidism, and excessive secretion of growth hormone. Mazabraud syndrome is a rarer syndrome characterized by the co-occurrence of polyostotic FD and intramuscular myxomas.[9]

FD in the craniofacial region is generally regarded as monostotic, even if multiple bones are affected, and is referred to as "craniofacial fibrous dysplasia." This is because FD is contiguous within the craniofacial bones affected and can cross the midline (**Fig. 1**A and B).[10] The incidence of monostotic craniofacial FD ranges from 10% to 25%, while craniofacial involvement in cases of polyostotic FD can reach rates as high as 90%.[11]

Most patients present with painless swelling and facial asymmetry; however, pain, pathologic fracture, and visual impairment have been reported.[7] Approximately 1% of

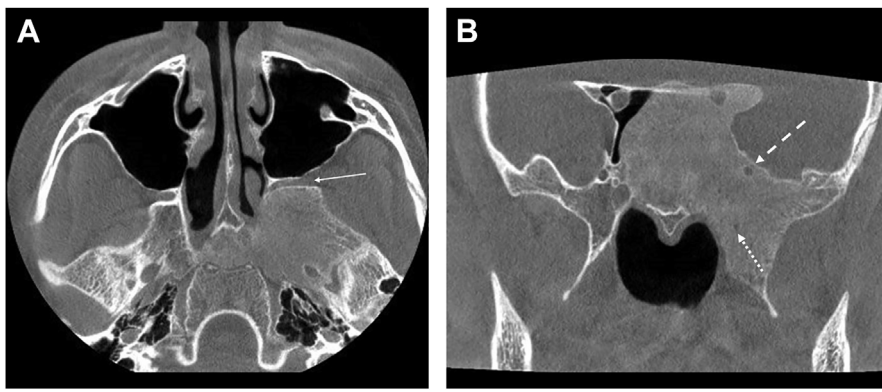

Fig. 1. Craniofacial FD. (*A*) Axial CBCT section and (*B*) coronal CBCT sections reveal FD in the body, pterygoid process, lesser and greater wings of the left sphenoid. Mild narrowing of the left pterygomaxillary fissure and pterygopalatine fossa (*arrow*) and vidian canal (*dotted arrow*). The distance between the left vidian canal and foramen rotundum (*dashed arrow*) is larger than on the left side. Note the enlargement but maintenance of the anatomic shape of the sphenoid bone and obliterated sphenoid sinus. CBCT, cone beam computed tomography; FD, fibrous dysplasia.

FD progresses into malignant sarcomatous transformation.[12] Significant risk factors for this transformation include exposure to radiation and the presence of McCune-Albright syndrome.[12]

Radiographically, FD typically demonstrates abnormal granular trabecular pattern classically described as "ground glass" (Fig. 2A–D). The borders are usually diffuse with a wide zone of transition between abnormal and normal bone; an important feature that is crucial in differentiating FD from cemento-ossifying fibroma (COF). Early lesions, however, can manifest with well-defined margins with mixed to low density.[13] Unlike benign neoplastic lesions, FD tends to cause bony expansion while maintaining the original anatomic shape of the involved bone. Loss of lamina dura and periodontal ligament space can be seen. Displacement of teeth and other adjacent structures (eg, sinonasal boundaries, orbital floor) can be observed.[14] The inferior alveolar canal may be displaced superiorly, in contrast to cystic lesions and benign tumors affecting the mandible (see Fig. 2A–D). This finding is considered by some authors as pathognomonic feature of FD.[15]

Concurrent presentation of simple bone cysts (SBCs) or aneurysmal bone cysts (ABCs) with fibro-osseous lesions has been reported.[16,17] Radiographic features of SBC of radiolucent spaces and scalloping can be seen within FD (Fig. 3A–C).

Cemento-osseous dysplasia

This is a non-neoplastic change affecting the tooth-bearing bone where normal bone is replaced by fibrous tissue and varying amounts of calcified material that increase as the lesion matures (Fig. 4A and B). Cemento-osseous dysplasia (COD) is common in

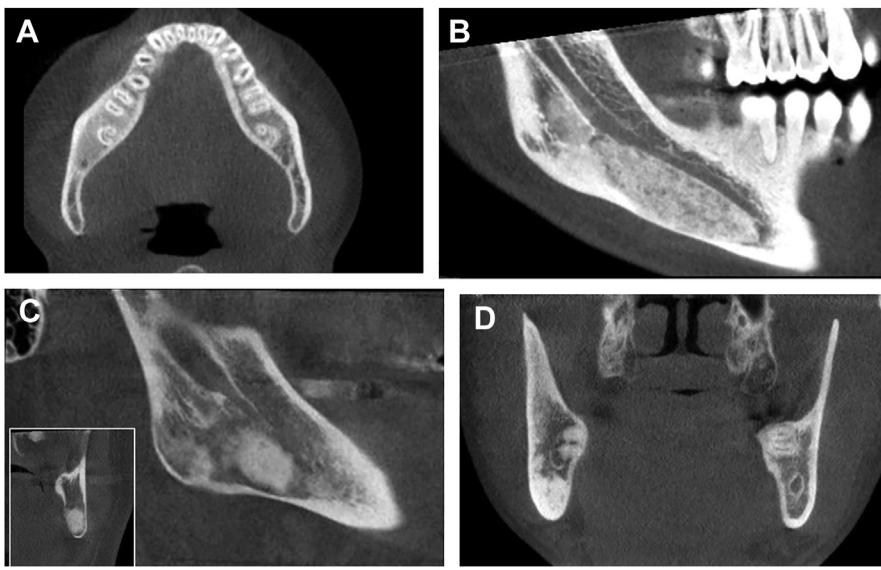

Fig. 2. Radiographic presentation of FD. CBCT images (*A*) Axial section, fine-granular bone pattern of FD in the right posterior mandibular molar area. (*B*) Corrected sagittal section reveals superior displacement of the mandibular canal. (*C*) Corrected sagittal section with inset coronal section, FD with cotton-wool density. (*D*) Coronal section, FD with a density similar to that of a dense bone island. Note the overall enlargement of the right mandibular body and ramus with maintenance of anatomic shape. FD, fibrous dysplasia; CBCT, cone beam computed tomography.

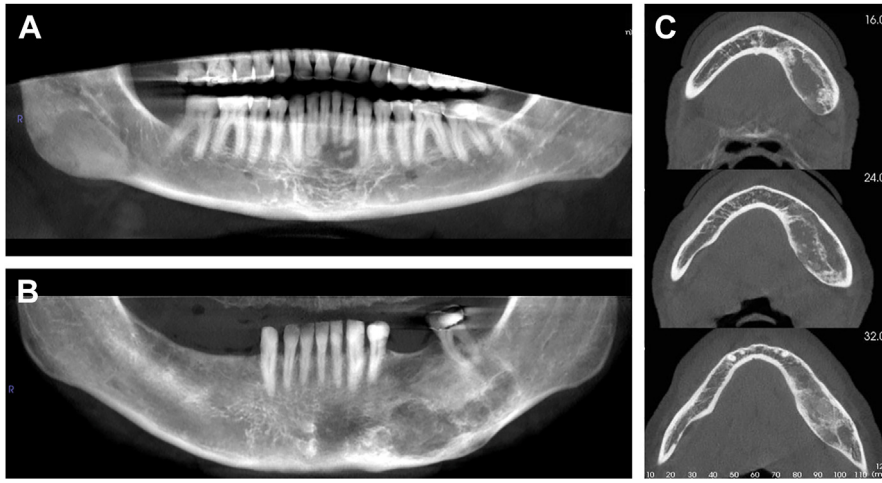

Fig. 3. Lesions existing with FD. Panoramic reconstruction from CBCT images (*A*) FD in the right mandibular angle and periapical cemento-osseous dysplasia (mixed stage) in the mandibular incisor region, (*B*) FD with simple bone cyst in the left mandibular body. (*C*) Axial CBCT images of the same case in (*B*) showing granular bone pattern and the large radiolucent component with expansion and thinning of the lingual cortex of mandible. FD, fibrous dysplasia; CBCT, cone beam computed tomography.

black and Asian females but can occur in any ethnicity and gender. The associated teeth are typically vital.

The term "cemento-" has been added and removed several times because the nature of calcified material cannot be confirmed to be cementum, only cementum-like.[1,2]

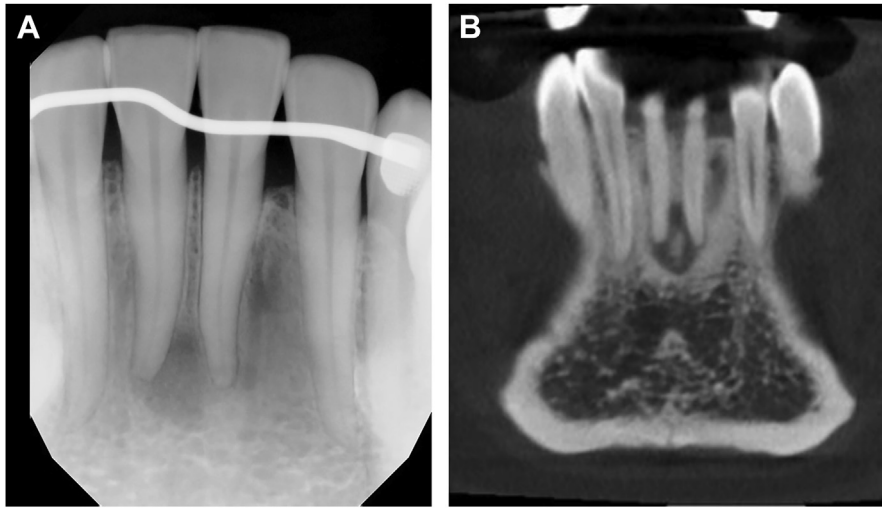

Fig. 4. Maturation of periapical cemento-osseous dysplasia. (*A*) Periapical radiograph of periapical radiolucency at mandibular incisors with intact PDL spaces. (*B*) Three years later, cementum-like calcifications are noted in the coronal CBCT section. CBCT, cone beam computed tomography; PDL, periodontal ligament.

There are 3 subjective presentations of this entity.

- Periapical COD: it affects the anterior teeth (can be bilateral).
- Focal COD: it affects the posterior teeth (can be bilateral).
- Florid COD: it shows extensive involvement of one or both jaws.

Radiographically, COD is usually well defined, with or without cortical outline, at the apical one-third of the root, and has internal calcifications similar to cortical bone or cementum.[18,19] As the lesion matures, a thin radiolucent rim, independent of the periodontal ligament space, is noted; however, it can be indiscernible in mature lesions (**Fig. 5**A–D). The cortical plates are usually unaffected but mild thinning, undulating endosteal surface, or nonconcentric expansion can be observed.[1,18]

Concomitant presentation of SBC with COD has been reported (12.7% prevalence).[16] The radiolucent background extends beyond the cementum-like calcifications and shows scalloping between the roots (**Fig. 6**A and B). Compared with solitary SBC, COD-associated SBC showed higher female predilection, presented in older age groups (fifth decade vs second), and were more likely to cause cortical expansion, root scalloping, and loss of lamina dura.[16] It is hypothesized that the empty cavity in bone forms due to the low or inadequate osteoblast numbers in the bones of middle-aged women or because of the changing biomechanical properties of adolescent bone during growth.[16]

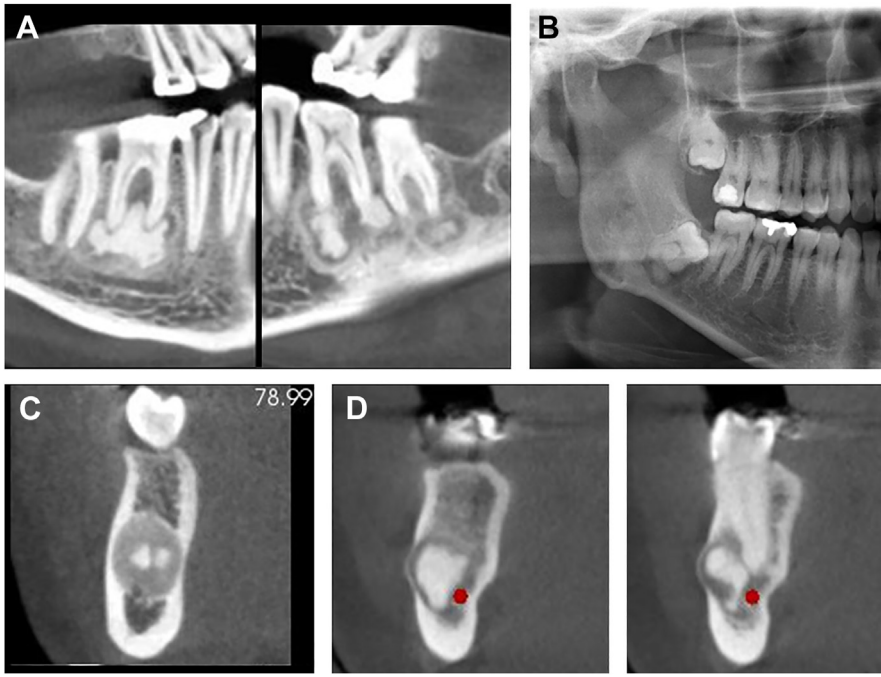

Fig. 5. Radiographic presentation of periapical/focal cemento-osseous dysplasia. (*A*) Corrected sagittal CBCT images showing bilateral, mixed lesions at the roots of mandibular molars. (*B*) Cropped panoramic radiograph showing similar lesion in an impacted third molar. CBCT bucco-lingual cross-sections of the right mandibular premolar area in (*C*) a combination of granular and cementum-like opacities and (*D*) irregular, nonconcentric cortical expansion, and no displacement of the mandibular canal. CBCT, cone beam computed tomography.

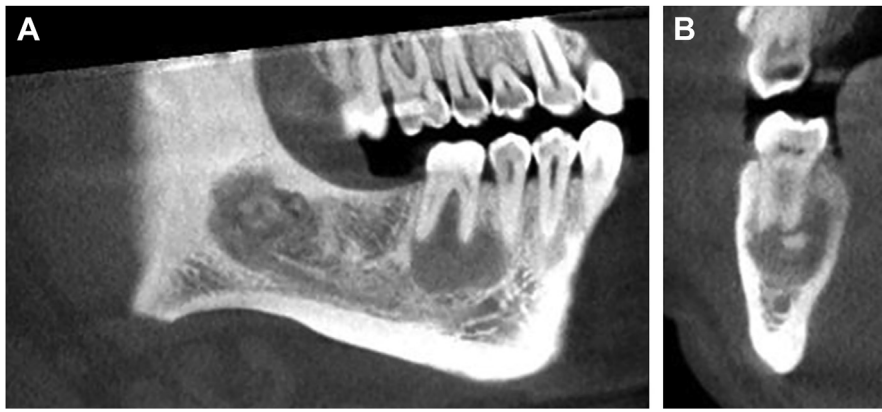

Fig. 6. Concomitant SBC with COD. CBCT images (*A*) corrected sagittal section, SBC scalloping between the roots of the right mandibular first molar, (*B*) cropped coronal section of the same lesion showing the cementum-like calcification. Note a second COD lesion in the third molar area. CBCT, cone beam computed tomography; COD, cemento-osseous dysplasia ; SBC, simple bone cyst.

Rarely, COD lesions can exist in ABC and cases of neurofibromatosis type 1 (NF1).[20,21] The cause is unknown, but theories favor altered local hemodynamics in ABC and gene alteration in NF1 affecting bone organization and reorganization (**Fig. 7**).[20,21]

A subcategory of COD that remains an enigma is "*familial gigantiform cementoma (FGC)*, aka familial florid COD or familial ossifying fibroma." This is a rare autosomal dominant condition with multiple, rapidly growing COD jaw lesions causing disfiguring

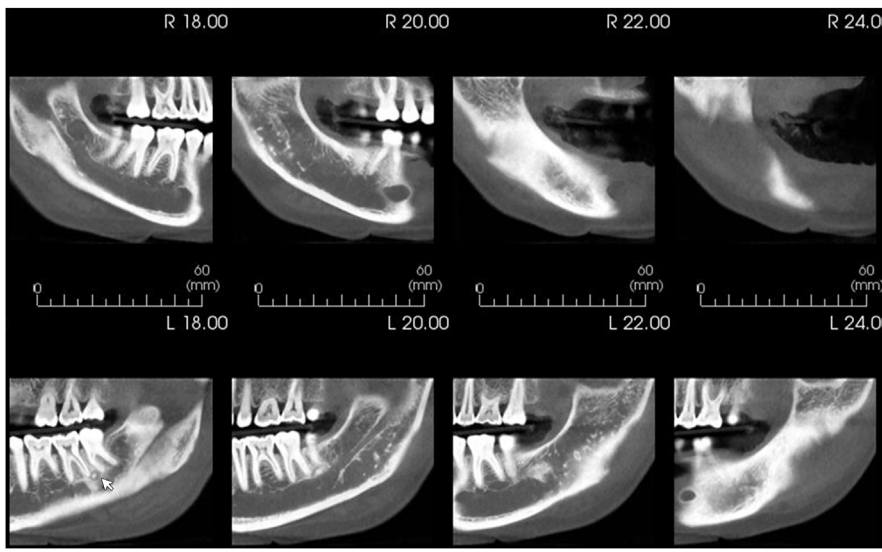

Fig. 7. COD with neurofibromatosis. Corrected sagittal CBCT images of bilateral wide mandibular canals and mental foramina. Note the mixed COD between the root apices of the left second and third molars (*arrow*). CBCT, cone beam computed tomography; COD, cemento-osseous dysplasia.

expansion in young subjects. Jaw lesions are excised to avoid excessive growth. Affected subjects may develop osteopenia and sustain long bone fractures. Diagnosing FGC is challenging because the COL1A2 mutation is not consistently found in FGC, sporadic gigantiform cementomas are reported (ie, lacking familial history), and its confusing heterogeneity with gnathodiaphyseal dysplasia, an autosomal dominant disorder in the ANO5 gene that features disfiguring fibro-osseous jaw lesions, long bone bowing with bone fragility, and recurrent fractures.[22–25]

Because of the abnormal bone metabolism, bone with COD is hypovascular and as such is prone to osteomyelitis (11% prevalence) if injury or infection is introduced by biopsy, dental disease, or treatment.[1,18,26] Alveolar ridge atrophy and presence of pulpal or periodontal disease increase the chances of secondary infection and the cementum-like calcifications would act as sequestra (**Fig. 8**A–C).[27] Maintaining good oral hygiene, arriving at proper diagnosis, and avoiding unnecessary intervention are key to avoid complications.

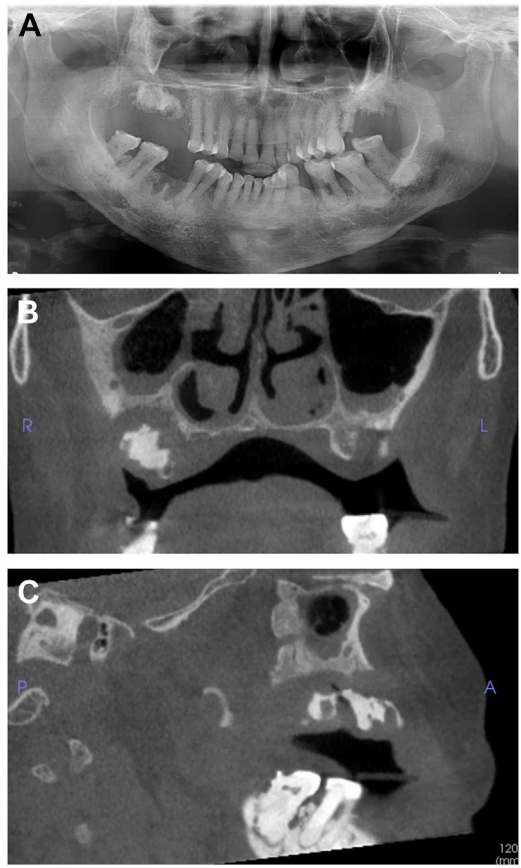

Fig. 8. Florid COD with secondary osteomyelitis. (*A*) Panoramic radiograph showing multifocal, mixed lesions periapical to the mandibular incisors and all molar teeth. CBCT images (*B*) Coronal section reveals "sequestrum" COD in the right posterior maxilla, resorption of alveolar crest, buccal and palatal plates of maxilla, sclerosis and thickening of the right maxillary sinus, (*C*) Sagittal section shows dense opacities periapical to the right mandibular molars. CBCT, cone beam computed tomography; COD, cemento-osseous dysplasia.

Cemento-ossifying/ossifying fibroma

COF is currently classified as a benign mesenchymal odontogenic tumor.[28] It is a thinly encapsulated benign neoplasm composed of dense fibrocellular tissue with irregular bony trabeculae or cementum-like material. The WHO previously grouped ossifying fibroma (OF) with COF but recently was reclassified as nonodontogenic origin under "benign fibro-osseous and chondro-osseous lesions."[29]

Juvenile OF is an uncommon variant presenting in middle childhood to adolescence and exhibits aggressive behavior with a higher likelihood of recurrence.[30] Multiple juvenile OFs can present in hyperparathyroidism-jaw tumor syndrome, which is an inherited disorder caused by a mutation in the tumor suppressor gene *CDC73*.[31]

Presence of multiple COF-like lesions warrants further investigations to rule out FGC, gnathodiaphyseal dysplasia, or hyperparathyroidism-jaw tumor syndrome.

COF is commonly observed in females during their third and fourth decades of life whereas males are slightly more affected by juvenile OF.[29,32] COF is mostly seen in the posterior mandible. The majority of cases are incidental or present as painless swelling and only 16% have experienced pain.[29]

Radiographically, COFs are well-defined, expansile lesions, with a thin border of radiolucent soft-tissue encapsulation. The lesions exhibit progressive calcification, initially manifesting as low-density areas that gradually become more radiopaque (**Fig. 9**A–C). Internally, the majority of lesions (58%) exhibit a ground-glass appearance, 26% of lesions were radiolucent, and 16% were sclerotic.[29]

COF may mimic FD radiographically. Unlike FD, which exhibits a tendency to merge with the adjacent bone, COF maintains well-defined boundaries with a distinct radiolucent band that separates it from the adjacent bone.[33] In addition, COF exhibits predominantly concentric expansion, which aligns with the anticipated pattern of benign

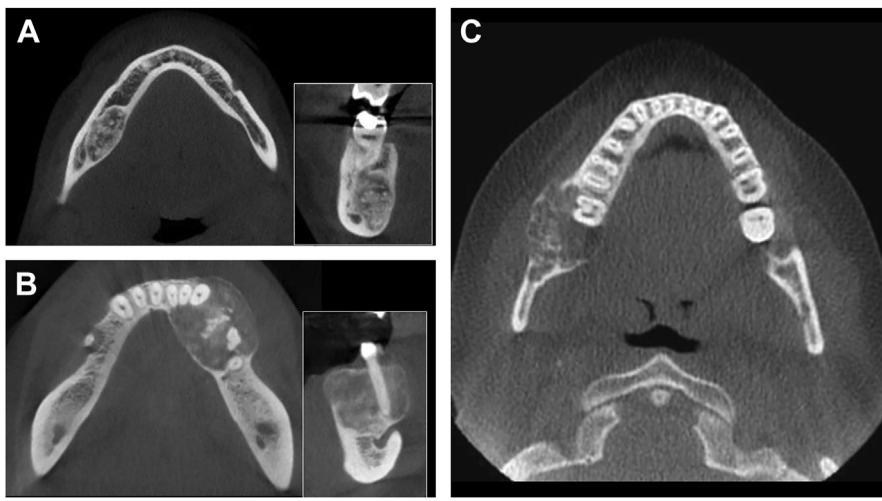

Fig. 9. COF in the mandible. CBCT images (*A*) Axial and coronal section (inset) showing mature COF with sclerotic border, a mixture of granular and cementum-like calcifications, and causing inferior displacement of the mandibular canal, (*B*) Axial and coronal section (inset) showing concentric expansion of COF with mesial displacement of the mandibular anterior teeth and narrowing of the mental foramen, (*C*) Axial section of COF presenting with scattered, wispy trabecular pattern. CBCT, cone beam computed tomography; COF, cemento-ossifying fibroma. (Images B and C are courtesy of Dr. Marcel Noujeim, DDS, MS, Oral and maxillofacial Radiologist, San Antonio, Texas, USA.).

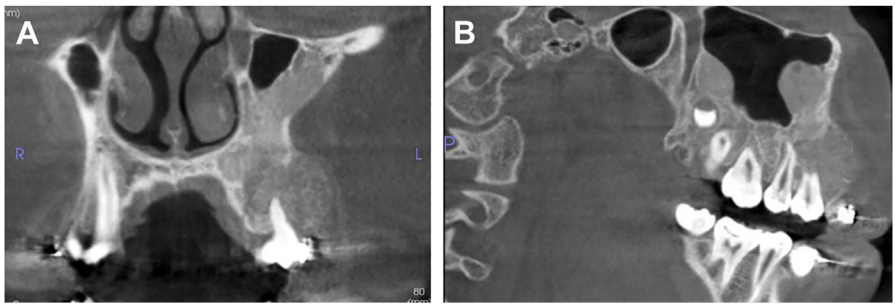

Fig. 10. COF of the maxilla. CBCT images A) Coronal section of COF in the left maxilla extending to the floor of maxillary sinus. Note the 2 bone patterns; fine-granular similar to "fibrous dysplasia" superiorly, fine-wispy and expansile at the alveolar process. B) Sagittal section showing concentric expansion affecting the buccal cortex of maxilla and anterior wall of maxillary sinus. Note apical root resorption of the maxillary left first premolar. CBCT, cone beam computed tomography; COF, cemento-ossifying fibroma. (Images are courtesy of Dr. Marcel Noujeim, DDS, MS, Oral and maxillofacial Radiologist, San Antonio, Texas, USA.).

neoplasms. Teeth displacement can occur as well as loss of lamina dura and root resorption. Maxillary lesions have the potential to cause significant displacement of the maxillary sinus, occasionally taking up nearly the entire volume of the sinus (Fig. 10A and B).

Giant Cell Lesions

Central giant cell granuloma

These lesions grow from bone cells with abundant giant cells and mostly affect the anterior mandible of patients younger than 30 years. There is a debate whether central giant cell granuloma (CGCG) is a benign neoplasm or reactive of unknown stimulus. However, due to its unpredictable and sometimes aggressive nature, classifying it as a benign neoplasm is preferable. Although efforts were done to identify mutations and immunohistochemical markers to differentiate CGCG of the jaws from giant cell tumor of bone, these are not conclusive due to small sample size and variability in data.[34,35]

Radiographically, CGCG is usually well defined, thinly corticated, unilocular or multilocular with fine-wispy septae (Fig. 11A–C). Dependent on aggressive behavior, teeth displacement and resorption, cortical thinning and expansion can vary (Fig. 12A–D). The presence of CGCG in adults warrants biochemical tests to assess hypercalcemia, hypophosphatasia, and increased PTH (parathyroid hormone) to rule-out brown tumor of hyperparathyroidism.

CGCG can exist with other lesions and become a hybrid CGCG. Common coexiting lesions are central odontogenic fibroma (35.9%), central OF (28.2%), and FD (17.9%).[36] Radiographically, hybrid CGCG tends to blend with the coexisting lesion except for the radiopaque lesions of FD, melorheostosis, and Paget's disease of bone (PD), where CGCG features are clearly evident.[36]

Cherubism

Cherubism is an autosomal dominant condition due to mutations in the SH3BP2 gene where bilateral CGCG present in the posterior jaws. Although thought to be familial, hereditary and sporadic cases have been reported. It is a disease of childhood where CGCG causes significant bilateral expansion, giving rise to the facial appearance of

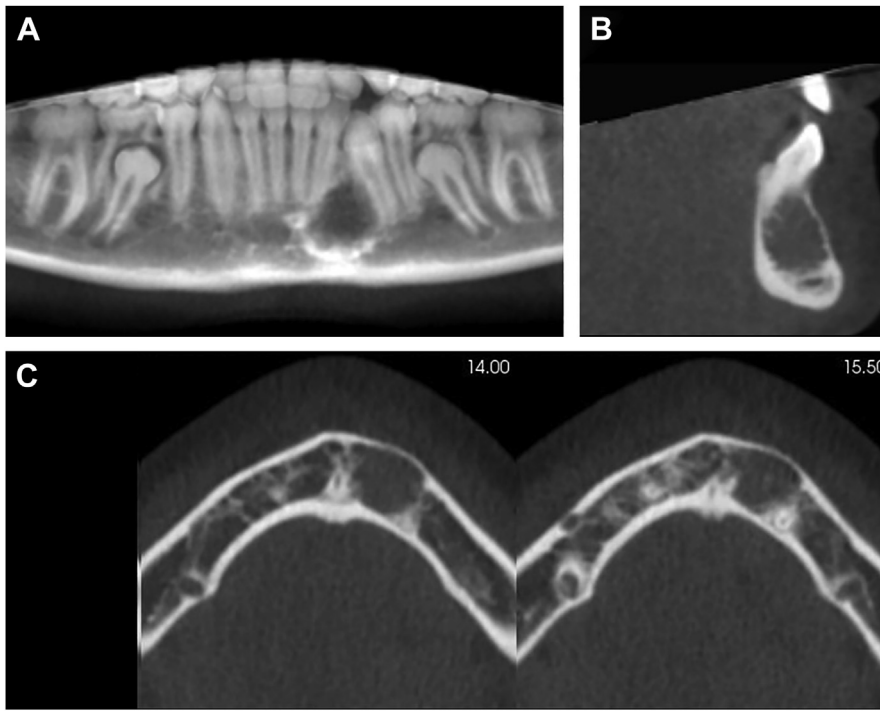

Fig. 11. CGCG. CBCT images (*A*) Cropped panoramic reconstruction of a 14-year-old female with CGCG in the left mandible causing displacement and delayed eruption of the left canine. (*B*) sagittal and (*C*) axial sections showing subtle wisps of fine-granular bone, undulating periphery, mild thinning, and expansion of the buccal cortex of mandible. CBCT, cone beam computed tomography; CGCG, central giant cell granuloma.

the chubby-cheeked little angels "cherubs." Jaw enlargement continues to puberty then tends to regress. Cherubism may be associated with neurofibromatosis type 1, fragile X syndrome, and Noonan-like syndrome.[37]

The radiographic appearance is characteristic; bilateral, well-defined, multilocular, and expansile radiolucent lesions. The epicenter of the lesions is in the posterior aspect of the jaws, resulting in anterior displacement of the teeth (**Fig. 13**A–C). The lesions can cause displacement of the maxillary sinuses and may extend to the condylar heads.[38]

Aneurysmal bone cyst

ABC is a benign, non-neoplastic, expansile, osteolytic lesion. It is composed of multiple blood-filled spaces separated by fibrous septa containing fibroblasts, reactive woven bone, and osteoclast-like giant cells.[39] ABCs can arise de novo or in pre-existing lesions such as FD, COD, CGCG, chondrosarcoma, or osteosarcoma.[21]

Most ABCs occur in children and young adults, with no significant gender predilection. Lesions are most common in the posterior mandible. They usually present as a relatively rapidly growing, painful jaw mass.

Radiographically, the lesion presents as a multilocular, or less commonly, unilocular radiolucency associated with marked expansion ("ballooning") and thinning of the outer cortical plates. The margins may be well defined or ill defined. Lesions often

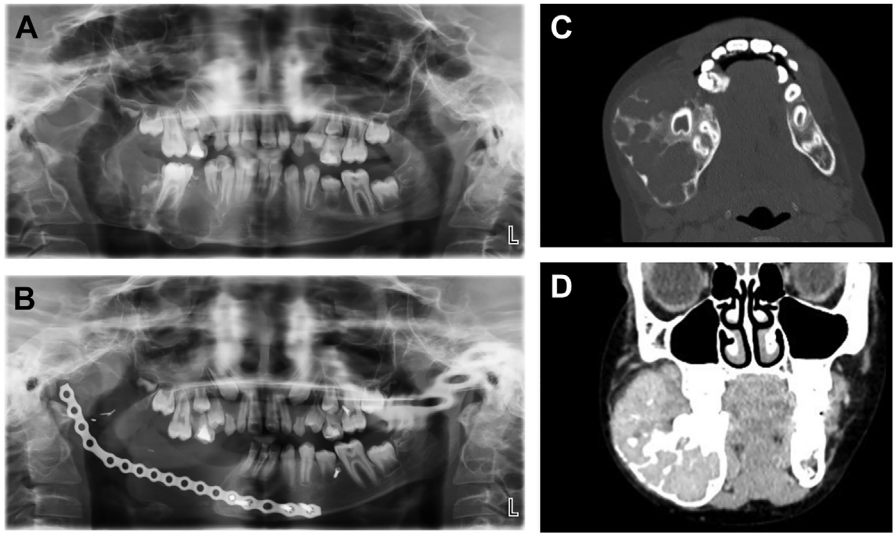

Fig. 12. Aggressive central giant cell granuloma of the mandible. Panoramic radiographs (*A*) of a 10-year-old boy showing multilocular, expansile radiolucency causing teeth displacement, (*B*) Four months postoperative jaw resection. Multidetector computed tomography (*C*) Axial bone window and (*D*) coronal soft tissue window showing a large, expansile, multilocular, low-attenuation lesion with multiple thin septae emanating from periphery to center. Note granular bone matrix surrounding the teeth.

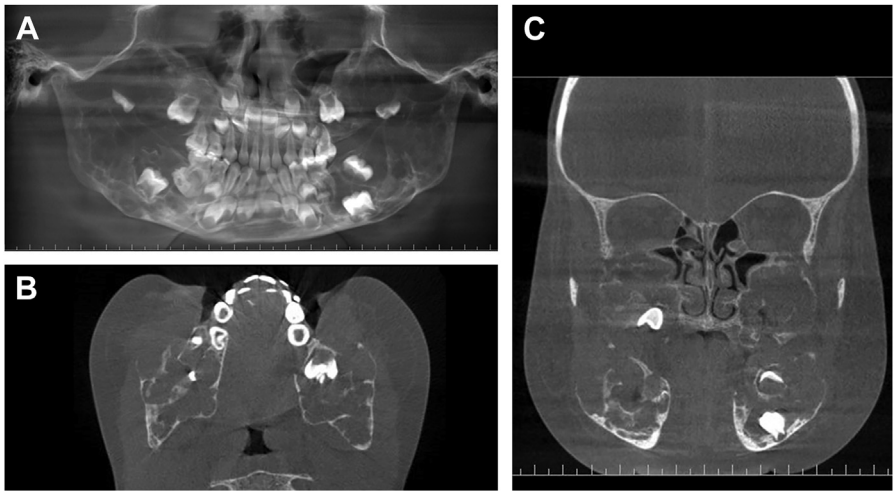

Fig. 13. Cherubism. Cone beam computed tomography images (*A*) Panoramic reconstruction showing bilateral, multilocular, expansile radiolucencies in the posterior maxilla and mandible. The condylar heads are spared. Note the anterior displacement of the unerupted molars bilaterally. (*B*) Axial section showing mandibular lesions with fine septae, some emanating at 90° toward the center of the lesion. (*C*) Coronal image showing the expansile lesions with extra-oral swelling of cheeks and involvement of the maxillary sinuses. (Images are courtesy of Dr. Marcel Noujeim, DDS, MS, Oral and maxillofacial Radiologist, San Antonio, Texas, USA.).

have wispy, ill-defined septa similar to those of a CGCG. Tooth displacement and root resorption may occur.[40] On MRI, typically there are multiple cystic components and fluid-fluid levels (Fig. 14A–D).

Other Osseous Conditions

Paget's disease

PD, also referred to as osteitis deformans, is a chronic disorder characterized by abnormal bone remodeling due to dysregulated osteoclast and osteoblast activity, causing structurally defective bone and marrow fibrosis. PD undergoes 3 temporal phases: lytic, mixed lytic-blastic, and blastic. Although the disease can affect various bones, the involvement of the jaws is relatively rare compared to that of the skull, pelvis, or femur.

PD typically affects people between the ages 55 and 75, with a rare incidence among those under the age of 40. Higher prevalence of PD is found in individuals of Anglo-Saxon descent residing in Europe, North America, Australia, and New Zealand,

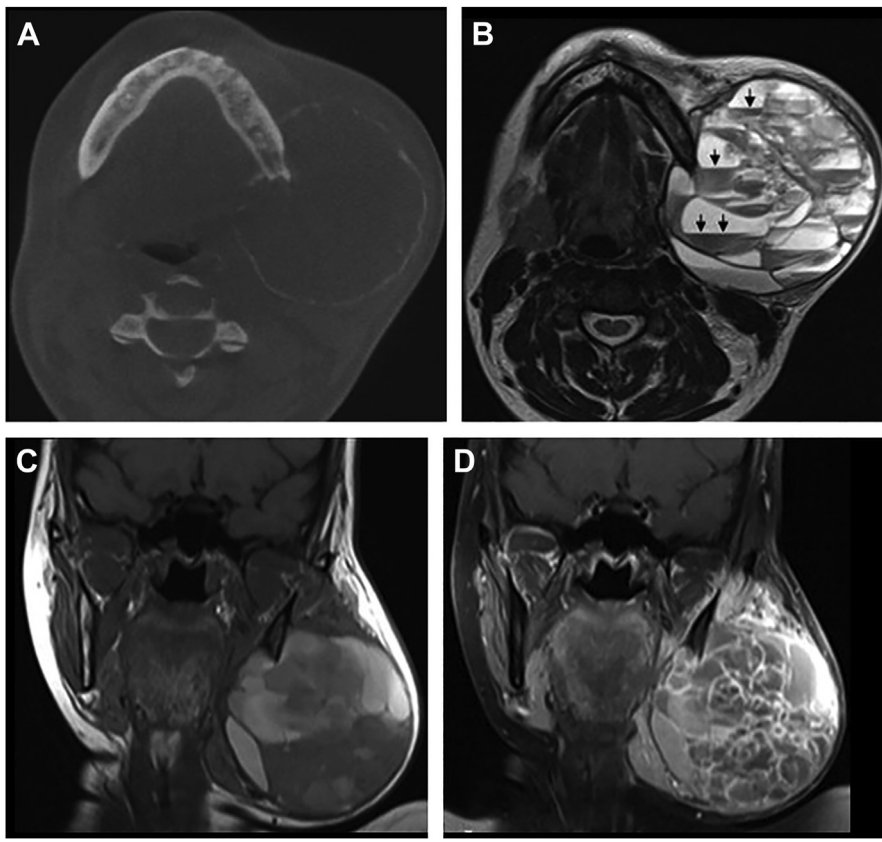

Fig. 14. ABC. (*A*) Axial CBCT image shows expansile, low-density lesion with undulating borders in the left posterior mandible. (*B*) Axial T2-weighted MRI showing hyperintense multicystic appearance with fluid-fluid levels (*arrowheads*) in the ABC. Coronal pregadolinium (*C*) and postgadolinium (*D*) T1-weighted MRI of the ABC showing contrast enhancement. ABC, aneurysmal bone cyst; CBCT, cone beam computed tomography (Reproduced with permission from Elsevier)[40]

while it is notably rare in Asia, Africa, and Scandinavia. Males are slightly more susceptible than females.[41]

Bone pain is a common symptom at diagnosis and blood test reveals elevated alkaline phosphatase levels. Potential complications include deformity and rare sarcomatous degeneration (<0.5%).[42] However, the 5-year survival rate for osteosarcoma arising from PD in the jaw is 21%, significantly lower than primary osteosarcoma of the jaw.[43]

Early lesions may manifest as radiolucencies that progress to a “ground-glass” or "cotton-wool" attenuation pattern and then mature to a uniformly sclerotic appearance with increased bone size. PD jaw lesions often show cortical and trabecular thickening, sclerotic alterations in alveolar bone, hypercementosis, and loss of lamina dura (**Fig. 15**A–C). Extragnathic features of PD include well-defined lytic cranial lesions, referred to as osteoporosis circumscripta, as well as the “tam-o’-shanter”

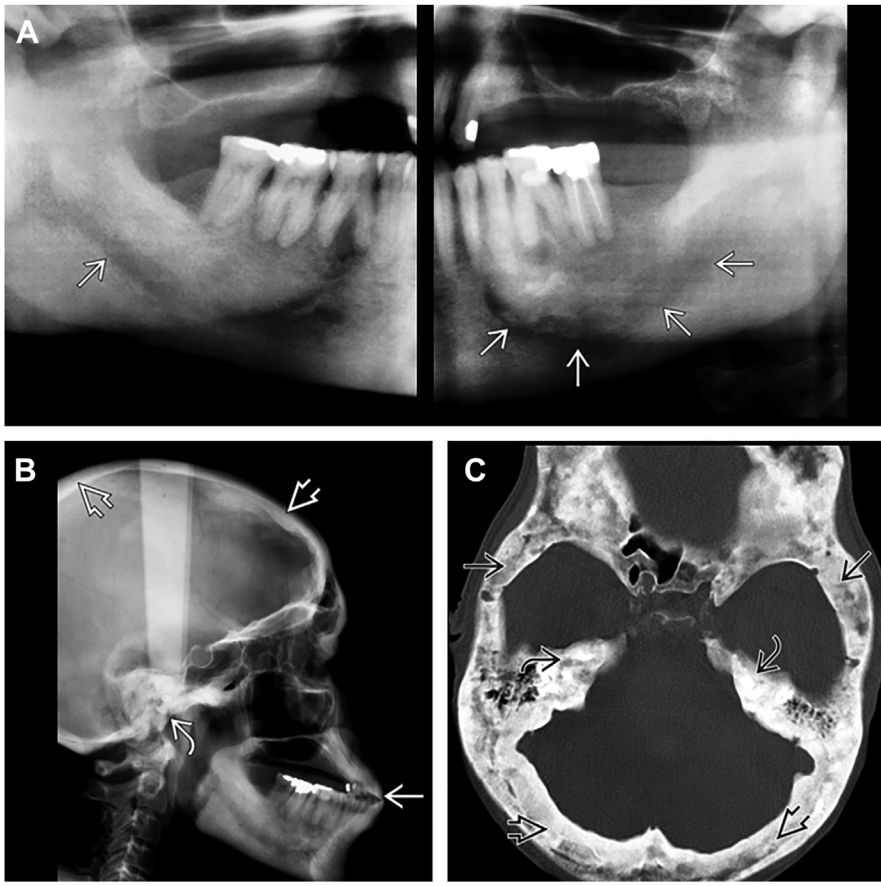

Fig. 15. PD. (*A*) Panoramic radiograph showing late phase of PD in the right mandible and early phase in the left mandible noted by the radiolucent area. Note hypercementosis in the right mandibular molars. (*B*) Lateral skull radiograph showing thickening of the diploic space, sclerosis of skull base, and flaring of the anterior teeth. (*C*) Axial bone MDCT showing mixed phase of PD in calvarium and cranial base with cotton-wool pattern in the squamous temporal and occipital bones. MDCT, multidetector computed tomography; PD, Paget’s disease. (Reproduced with permission from Elsevier.)[45]

sign due to expansion of the diploic space and platybasia caused by the skull base softening. Bone scintigraphy can aid in diagnosing PD by revealing widespread radiotracer uptake in the mandible, creating a "black beard" appearance known as the "Lincoln sign."[13]

Lesions involving the alveolar process can mimic florid COD; however, a distinguishing characteristic is their extension into the basal bone. PD imaging features can resemble FD; however, age of onset (young in FD), bilateral presentation (FD usually is unilateral), and PD changes in the skull can aid differentiation.

Langerhans cell histiocytosis

Formerly known as histiocytosis X, Langerhans cell histiocytosis (LCH) includes Letterer-Siwe disease, Hand-Schuller-Christian disease, and eosinophilic granuloma. This rare spectrum is characterized by the proliferation of Langerhans cells, dendritic immune cells formed in the bone marrow. LCH can present as intraosseous lesions or involve multiple organs. The latter presentation tends to be aggressive in behavior and requires radiation or chemotherapy.[44]

The average age of LCH is 14 years (range: 11 months–59 years) and although some studies report male predilection, others show the opposite.[44] LCH of the bone targets vertebrae, long bones, the mandible in children, the cranium and ribs in adults. Patients with jaw lesions may present with pain, tooth mobility, gingival bleeding, ulcerations, or swelling.

Most gnathic lesions present in the mandible as “scooped-out” bony destruction, with periosteal reaction parallel to the original cortex. There may be peripheral bone

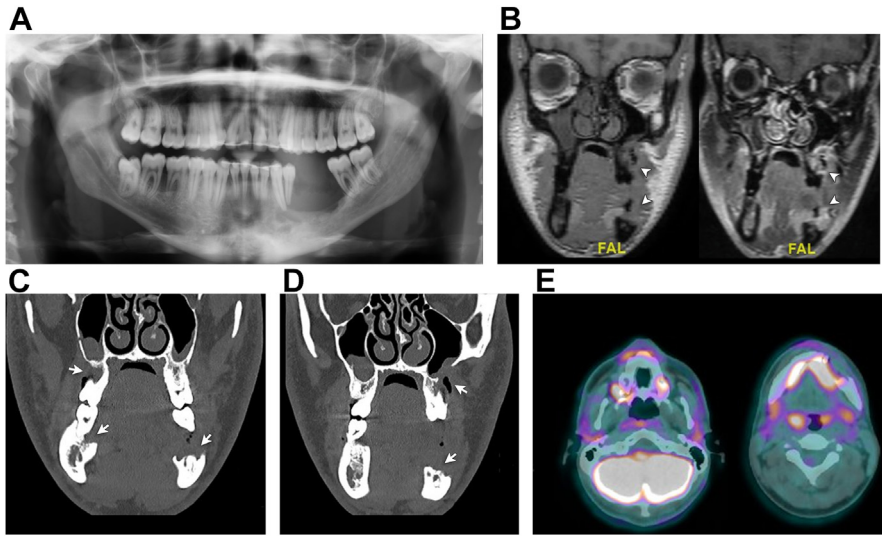

Fig. 16. LCH. (*A*) Panoramic radiograph of a 23-year-old male shows large radiolucent defect in the left mandibular alveolar process with loss of the first molar and second premolar. Note the saucer shape of the bone destruction. A similar but smaller lesion is noted on the contralateral side. Bilateral, ill-defined radiolucencies are noted in the maxillary premolar-molar area. Coronal MR images (*B*) T1WI and T1+C show isointense signal similar to muscle with mild enhancement (*arrowheads*). Coronal MDCT bone-windows (*C*) and (*D*) showing multifocal “punched-out” osteolytic lesions in the maxilla and mandible (*arrows*). (*E*) Nuclear medicine images (PET-FDG) reveal high uptake in the LCH lesions. FDG, fluorodeoxyglucose; LCH, Langerhan’s cell histiocytosis; MDCT, multidetector computed tomography.

Table 1
Summary of common radiographic features of fibro-osseous and other bone lesions affecting the jaws

	Location	Periphery	Internal Structure	Effects on Surrounding Structures	Key Points
FD	Monostotic: Maxilla>mandible Unilateral Polyostotic: Multiple bones Craniofacial: Multiple cranial bones	Ill-defined, blending Early lesions are well defined	Granular (ground-glass) appearance Can be mixed with sclerotic areas and cystic cavities	Nonconcentric expansion (maintain the overall shape) +/− displacement of teeth, sinonasal structures, orbit, IANC Loss of lamina dura and thin PDL space	Polyostotic FD is associated with McCune-Albright syndrome Superior displacement of the IANC is pathognomonic
COD (Periapical, focal, and florid)	Mandible>maxilla Unilateral or bilateral Solitary or multiple Florid type: extensive involvement of one or both jaws	Well defined +/− cortical boundary	3 stages: RL, mixed, RO Radiopacities similar to cortical bone or cementum Calcified material can have swirling or fine-granular pattern	Minimal, nonconcentric cortical expansion Normal PDL space	Generally maintains radiolucent rim Can coexist with simple bone cyst Risk of secondary osteomyelitis Epicenter above IANC
COF	Mandible>maxilla	Well defined with thin radiolucent rim	Variable degree of RO depending on lesion maturity Can be granular, resembling fibrous dysplasia	Concentric expansion Displacement of teeth, maxillary sinus, IANC Root resorption	Juvenile subtypes exhibit aggressive behavior Well-defined margin helps to differentiate COF from FD Multiple lesions→ consider hyperparathyroidism-jaw tumor syndrome
Central giant cell granuloma	Mandible>maxilla Unilateral or bilateral (brown tumors)	Well defined +/− thin cortical boundary	Unilocular or multilocular RL (fine septae, ± emanate from peripheral boundary to center at right angle)	Variable cortical expansion +/− teeth displacement or resorption	Can present with radiopacities when hybrid with bone dysplasia

(*continued on next page*)

Table 1
(*continued*)

	Location	Periphery	Internal Structure	Effects on Surrounding Structures	Key Points
Cherubism	Mandible>maxilla Bilateral Epicenter: posterior	Well defined +/− thin cortical boundary	Unilocular RL (rare) Multilocular RL: multiple, fine-granular septae, overall sclerotic or "fibro-osseous matrix" appearance	Expansile Anterior displacement of teeth	Regress in adulthood
Aneurysmal bone cyst	Mandible>maxilla Solitary	Well defined +/− cortical boundary	Multilocular RL (fine septae) Vascular, solid, and mixed types	Very expansile Teeth displacement or resorption	Can coexist with bone dysplasia and other benign tumors Angiography needed to verify feeding vessels
Paget's disease	Multiple bones Rare in the jaws Maxilla>mandible Bilateral	The entire bone is affected	3 stages: RL, mixed, RO RO: cotton-wool or sclerotic	Bone enlargement Teeth displacement and hypercementosis	Look for lytic lesions in the skull and increased diploic space Increased serum alkaline phosphatase
Langerhans cell histiocytosis	Solitary or multifocal Alveolar, intraosseous, or multiorgan Can affect tooth follicle	Well defined Noncorticated Scooped-out or lobulated	RL Variable fluid content and enhancement	Cortical expansion +/− sclerosis of surrounding bone +/− periosteal reaction parallel to cortical plate +/− displace tooth germ	Look for other lesions in bone or organs Wide range of presentation based on behavior; aggressive benign to indolent malignant

Abbreviations: COD, cemento-osseous dysplasia; COF, cemento-ossifying fibroma; FD, fibrous dysplasia; IANC, inferior alveolar nerve canal; RL, radiolucent; RO, radiopaque; PDL, periodontal ligament.

Table 2
Diagnostic tree of fibro-osseous and other bone lesions affecting the jaws based on common radiographic appearance

RL Solitary			RL Multifocal			Mixed or Predominately RO					
Unilocular			Multilocular	Unilocular	Multilocular	Solitary				Multifocal	
Expansile		Nonexpansile				Well defined			Diffuse	Above IANC	Beyond IANC
Concentric	Nonconcentric					Expansile		Nonexpansile			
						Concentric	Nonconcentric				
COF, CGCG, ABC, LCH	Early PCD, SBC	Early PCD, SBC, LCH	CGCG and ABC	COD, CGCG (brown tumor), LCH	Cherubism	COF, hybrid CGCG	COD	COD	Monostotic FD	Florid COD	Paget's, Polyostotic FD, FGC, HPT-JT
Ddx Ameloblastoma, OKC, residual cyst, fibrous scar. Malignancy (destructive)	Ddx Small odontogenic cyst or tumor	Ddx *Periapical* granuloma/ cyst Periodontal disease (crestal) Malignancy (destructive)	Ddx Ameloblastoma, odontogenic myxoma, OKC (less expansile)	Ddx *Periapical* granuloma/ cyst Periodontal disease (crestal) Malignancy (destructive)		Ddx Cementoblastoma (periapical), CEOT, ameloblastic fibro-odontoma, complex odontoma, COC, AOT	Ddx Healed SBC, odontoma (tooth-like density)	Ddx Healed SBC, odontoma (tooth-like density)	Ddx Osteomyelitis (sequestra + parallel periosteal bone reaction) Osteogenic sarcoma (destructive, sun-ray periosteal bone reaction)		Ddx Gardner's syndrome, Osteopetrosis

Abbreviations: ABC, aneurysmal bone cyst; AOT, adenomatoid odontogenic tumor; CEOT, calcifying epithelial odontogenic tumor; CGCG, central giant cell granuloma; COC, calcifying odontogenic cyst; COD, cemento-osseous dysplasia; COF, cemento-ossifying fibroma; Ddx, differential diagnosis; FD, fibrous dysplasia; FGC, familial gigantic cementoma; HPT-JT, hyperparathyroidism jaw tumors; IANC, inferior alveolar nerve canal; LCH, Langerhans cell histiocytosis; OKC, odontogenic keratocyst; PCD, periapical osseous dysplasia; RL, radiolucent; RO, radiopaque; SBC, simple bone cyst.

sclerosis (Fig. 16A–E). The bone destruction from alveolar LCH lesions may appear very similar to periodontal disease. Solitary intraosseous lesions can mimic squamous cell carcinoma and metastatic disease. Definitive diagnosis of bone LCH is confirmed by histopathological and immunohistochemical examinations.

RADIOGRAPHIC APPEARANCE AND DIFFERENTIAL DIAGNOSES

Fibro-osseous and other bone lesions affecting the jaws discussed in this review can have a wide range of radiographic features. Common features and important points are summarized in Table 1.

The radiographic differential diagnoses of fibro-osseous and other bone lesions affecting the jaws largely depend on periphery, distribution, internal structure, and effects on surrounding structures. It is important to take into consideration the clinical presentation and chronologic development of the lesion. As such, entities to be considered in the differential diagnosis can be anywhere from reactive to malignant diseases. Diagnostic tree of the lesions is provided in Table 2.

SUMMARY

Fibro-osseous and other bone lesions affecting the jaws discussed in this review can have overlapping histopathological picture or radiographic features. The clinical-radiographic correlation is key to understand the biological behavior and follow a diagnostic tree that culminates to proper management. Diversity in clinical-radiographic-histopathologic presentation along with hybrid occurrences of such lesions remains a challenge. The contribution of OMFRs in the comprehensive clinical and radiographic assessment is crucial to arriving at a diagnosis, recommending biopsy sites that can be representative of the lesion, and suggesting watchful waiting in cases where an intervention would be detrimental to the patient.

CLINICS CARE POINTS

- Analysis of all the correlating clinical, radiographic, and histological factors in a case is key. The ultimate goal is to provide the patient with accurate diagnosis and facilitate adequate management.
- Histopathologic examination is not needed or may even be contraindicated in some bone conditions (e.g., COD).
- Hybrid presentations may complicate the diagnostic process. Thorough selection of imaging modalities prior to biopsy elucidates the different tissues and lesion behavior.
- The oral and maxillofacial radiologist can explain the lesion's growth pattern, internal structure, and extent using different imaging modalities that reveal different aspects of the lesion.

DISCLOSURE

The authors have nothing to disclose.

REFERENCES

1. Nelson BL, Phillips BJ. Benign Fibro-Osseous Lesions of the Head and Neck. Head Neck Pathol 2019;13(3):466–75.

2. El-Naggar AK, Chan JKC, Takata T, et al. The fourth edition of the head and neck World Health Organization blue book: editors' perspectives. Hum Pathol 2017; 66:10–2.
3. Brody A, Zalatnai A, Csomo K, et al. Difficulties in the diagnosis of periapical translucencies and in the classification of cemento-osseous dysplasia. BMC Oral Health 2019;19(1):139.
4. Decolibus K, Shahrabi-Farahani S, Brar A, et al. Cemento-Osseous Dysplasia of the Jaw: Demographic and Clinical Analysis of 191 New Cases. Dent J 2023;11(5).
5. Cohen MM. Fibrous dysplasia is a neoplasm. Am J Med Genet 2001;98(4):290–3.
6. Andreu-Arasa VC, Chapman MN, Kuno H, et al. Craniofacial Manifestations of Systemic Disorders: CT and MR Imaging Findings and Imaging Approach. Radiographics 2018;38(3):890–911.
7. MacDonald-Jankowski D. Fibrous dysplasia: A systematic review. Dentomaxillofacial Radiol 2009;38(4):196–215.
8. Feller L, Wood NH, Khammissa RA, et al. The nature of fibrous dysplasia. Head Face Med 2009;5(1). https://doi.org/10.1186/1746-160X-5-22.
9. Majoor BCJ, Van De Sande MAJ, Appelman-Dijkstra NM, et al. Prevalence and clinical features of mazabraud syndrome: A multicenter european study. Journal of Bone and Joint Surgery - American 2019;101(2):160–8.
10. Rahman AMA, Madge SN, Billing K, et al. Craniofacial fibrous dysplasia: Clinical characteristics and long-term outcomes. Eye 2009;23(12):2175–81.
11. Ricalde P, Magliocca KR, Lee JS. Craniofacial Fibrous Dysplasia. Oral Maxillofac Surg Clin North Am 2012;24(3):427–41.
12. Stanton RP. Surgery for fibrous dysplasia. J Bone Miner Res 2007;22(SUPPL. 2). https://doi.org/10.1359/JBMR.06S220.
13. Holmes KR, Holmes RD, Martin M, et al. Practical Approach to Radiopaque Jaw Lesions. Radiographics 2021;41(4):E1164–85.
14. MacDonald-Jankowski D, Yeung R, Li T, et al. Computed tomography of fibrous dysplasia. Dentomaxillofacial Radiol 2004;33(2):114–8.
15. Petrikowski CG, Pharoah MJ, Lee L, et al. Radiographic differentiation of osteogenic sarcoma, osteomyelitis, and fibrous dysplasia of the jaws. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology. 1995;80(6): 744–50.
16. Chadwick JW, Alsufyani NA, Lam EWN. Clinical and radiographic features of solitary and cemento-osseous dysplasia-associated simple bone cysts. Dentomaxillofac Radiol 2011;40(4):230–5.
17. Koketsu Y, Tanei T, Kuwabara K, et al. Secondary aneurysmal bone cyst of the frontal bone with fibrous dysplasia showing rapid expansion: a case report. Nagoya J Med Sci 2023;85(2):395–401.
18. Alsufyani NA, Lam EWN. Osseous (Cemento-osseous) Dysplasia of the Jaws: Clinical and Radiographic Analysis. J Can Dent Assoc 2011;77.
19. Ahmad M, Gaalaas L. Fibro-Osseous and Other Lesions of Bone in the Jaws. Radiol Clin North Am 2018;56(1):91–104.
20. Friedrich RE, Reul A. Periapical Cemento-osseous Dysplasia Is Rarely Diagnosed on Orthopantomograms of Patients with Neurofibromatosis Type 1 and Is Not a Gender-specific Feature of the Disease. Anticancer Res 2018;38(4):2277–84.
21. Yeom HG, Yoon JH. Concomitant cemento-osseous dysplasia and aneurysmal bone cyst of the mandible: a rare case report with literature review. BMC Oral Health 2020;20(1):276.
22. Moshref M, Khojasteh A, Kazemi B, et al. Autosomal dominant gigantiform cementoma associated with bone fractures. Am J Med Genet 2008;146A(5):644–8.

23. Prasad C, Kumar KA, Balaji J, et al. A family of familial gigantiform cementoma: clinical study. J Maxillofac Oral Surg 2022;21(1):44–50.
24. Nel C, Yakoob Z, Schouwstra CM, et al. Familial florid cemento-osseous dysplasia: a report of three cases and review of the literature. Dentomaxillofacial Radiol 2021;50(1).
25. MacDonald DS. Classification and nomenclature of fibro-osseous lesions. Oral Surg Oral Med Oral Pathol Oral Radiol 2021;131(4):385–9.
26. Alsufyani N, Lam E. Cemento-osseous dysplasia of the jaw bones: key radiographic features. Dentomaxillofacial Radiol 2011;40(3):141–6.
27. Nadler C, Perschbacher SE, Septon D, et al. Important radiographic features in the identification of osseous dysplasia–related osteomyelitis. Oral Surg Oral Med Oral Pathol Oral Radiol 2021;131(6):730–7.
28. Speight PM, Takata T. New tumour entities in the 4th edition of the World Health Organization Classification of Head and Neck tumours: odontogenic and maxillofacial bone tumours. Virchows Arch 2018;472(3):331–9.
29. MacDonald-Jankowski D. Ossifying fibroma: a systematic review. Dentomaxillofacial Radiol 2009;38(8):495–513.
30. El-Mofty SK. Fibro-Osseous Lesions of the Craniofacial Skeleton: An Update. Head Neck Pathol 2014;8(4):432–44.
31. Torresan F, Iacobone M. Clinical Features, Treatment, and Surveillance of Hyperparathyroidism-Jaw Tumor Syndrome: An Up-to-Date and Review of the Literature. Int J Endocrinol 2019;2019:1–8.
32. El-Mofty S. Psammomatoid and trabecular juvenile ossifying fibroma of the craniofacial skeleton: Two distinct clinicopathologic entities. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology 2002;93(3):296–304.
33. Ahmad M, Gaalaas L. Fibro-Osseous and Other Lesions of Bone in the Jaws. Radiol Clin North Am 2018;56(1):91–104.
34. Gomes CC, Diniz MG, Bastos VC, et al. Making sense of giant cell lesions of the jaws (GCLJ): lessons learned from next-generation sequencing. J Pathol 2020; 250(2):126–33.
35. Nagar SR, Bansal S, Jashnani K, et al. A Comparative Analysis of p63 Expression in Giant Cell Tumour (GCT), Central Giant Cell Granuloma (CGCG) and Peripheral Giant Cell Granuloma (PGCG). Head Neck Pathol 2020;14(3):733–41.
36. Alsufyani NA, Aldosary RM, Alrasheed RS, et al. A systematic review of the clinical and radiographic features of hybrid central giant cell granuloma lesions of the jaws. Acta Odontol Scand 2021;79(2).
37. Papadaki ME, Lietman SA, Levine MA, et al. Cherubism: best clinical practice. Orphanet J Rare Dis 2012;7(Suppl 1).
38. Beaman FD, Bancroft LW, Peterson JJ, et al. Imaging Characteristics of Cherubism. Am J Roentgenol 2004;182(4):1051–4.
39. Liu Y, Zhou J, Shi J. Clinicopathology and Recurrence Analysis of 44 Jaw Aneurysmal Bone Cyst Cases: A Literature Review. Front Surg 2021;8:678696.
40. Omami G, Mathew R, Gianoli D, et al. Enormous aneurysmal bone cyst of the mandible: case report and radiologic-pathologic correlation. Oral Surg Oral Med Oral Pathol Oral Radiol 2012;114(1):e75–9.
41. Charles JF, Siris ES, Roodman GD. Paget Disease of Bone. In: Primer on the metabolic bone diseases and disorders of Mineral metabolism. Wiley; 2018. p. 713–20. https://doi.org/10.1002/9781119266594.ch92.
42. Ralston SH. Paget's Disease of Bone. N Engl J Med 2013;368(7):644–50.
43. Cheng YSL, Wright JM, Walstad WR, et al. Osteosarcoma arising in Paget's disease of the mandible. Oral Oncol 2002;38(8):785–92.

44. Reisi N, Raeissi P, Harati Khalilabad T, et al. Unusual sites of bone involvement in Langerhans cell histiocytosis: a systematic review of the literature. Orphanet J Rare Dis 2021;16(1):1.
45. Petrikowski CG. Paget Disease. In: Koenig LJ, Tamimi D, Petrikowski CG, et al, editors. Diagnostic imaging: oral and maxillofacial. 2nd Edition. Elsevier; 2017. p. 449. https://doi.org/10.1016/B978-0-323-47782-6.50130-7.