

DISEASES AFFECTING THE STRUCTURE OF BONE

- Coordination of the activities of **bone-forming (i.e., osteoblasts), bonemaintaining (i.e., osteocytes), and bone-removing (e.g., osteoclasts)** cells regulates the normal structure of bone in the skeleton. Indeed, it has been estimated that approximately 5% to 10% of total bone volume in the adult skeleton is replaced per year.
- Disease processes may produce abnormalities in the bone through disturbances in the balance of serum concentrations of calcium and phosphate while others may dysregulate osteoblasts, osteocytes, and osteoclasts.
- A **disease that alters the balance** of calcium and phosphate levels can result in the abnormal formation of bone and teeth: For example, a low serum calcium concentration can mobilize calcium from bone, thus depleting bone.
- The resultant reduced calcium level in bone can **alter the pattern of trabeculation** within the bone, producing bone of **low density in the radiologic image**.

- **Bone dysplasias** are also characterized by a dysregulation of the normally coordinated activities of osteoblasts and osteocytes; however, here the normal bone is replaced with an exuberant **proliferation of fibrous connective tissue and immature and abnormal bone**.
- These lesions, sometimes referred to by the descriptive histopathologic term “fibro-osseous lesions”, encompass a broad group of disease processes ranging from conditions of bone cell dysregulation to neoplasms.
- In oral and maxillofacial radiology, the term “fibro-osseous” is therefore discouraged, and the more specific terminology reflecting the nature of this disease process (bone dysplasia) is preferred.

Because these diseases may affect the entire body, the changes manifested in the appearance of the jaws in diagnostic images are usually generalized and often nonspecific, making it difficult to identify the diseases based on imaging characteristics alone. The general changes that can be seen in the jaws may include the following:

- 1. Change in size and shape of the bone
- 2. Change in the number, size, and orientation of trabeculae
- 3. Altered thickness and density of cortical structures
- 4. Increase or decrease in overall bone density

TABLE 25.1**Changes in Bone Observed in Systemic Disease^a**

Systemic Disease	BONES				
	Density	Size of Jaws	TRABECULAE		
			Increase	Decrease	Granular
Hyperparathyroidism	Decrease	No	Yes	Yes	Yes
Hypoparathyroidism	Rare increase	No	No	No	No
Hyperpituitarism	No	Large	No	No	No
Hypopituitarism	No	Small	No	No	No
Hyperthyroidism	Decrease	No	No	No	No
Hypothyroidism	No	Small	No	No	No
Cushing syndrome	Decrease	No	No	Yes	Yes
Osteoporosis	Decrease	No	No	Yes	No
Rickets	Decrease	No	No	Yes	No
Osteomalacia	Rare decrease	No	No	Rare decrease	No
Hypophosphatasia	Decrease	No	No	Yes	No
Renal osteodystrophy	Decrease; rare increase	Large	Rare	Yes	Yes
Hypophosphatemia	Decrease	No	No	Yes	Yes
Osteopetrosis	Increase	Large			
Sickle cell anemia	Decrease	Large maxilla			
Thalassemia	Decrease	Large maxilla			

^aThis table summarizes the major imaging changes to bone with endocrine and metabolic bone diseases. It does not include all the possible variable appearances.

Some systemic diseases that occur during tooth formation may result in dental alterations. Lamina dura is part of the bone structure of the alveolar process, but because it is usually examined in conjunction with the periodontal membrane space and roots of teeth, it is included with the description of the dental structures . Changes to teeth and associated structures include the following:

- 1. Accelerated or delayed eruption
- 2. Hypoplasia
- 3. Hypocalcification
- 4. Loss of a distinct lamina dura

METABOLIC BONE ABNORMALITIES

OSTEOPENIA

- Osteopenia is an imbalance of bone deposition and resorption that results in a **net decrease** in bone formation
- Osteopenia occurs as **part of the aging process** of bone and can be considered a variation of normal
- When loss of bone mass becomes significant, patients may undergo a diagnostic test known as dual x-ray photon absorptiometry (DEXA), and they may be clinically diagnosed with osteoporosis
- The **most important clinical manifestation of osteoporosis is fracture**, which may involve the distal radius, proximal femur, ribs, and vertebrae. Patients may have bone pain. Postmenopausal women are most at risk.

IMAGING FEATURES FOR OSTEOPENIA

- Osteopenia results in an **overall reduction of bone density**, and this change can be observed by comparing the bone to the adjacent teeth.
- There may also be evidence of reduced density and **thinning of bone cortices**, such as the inferior cortex of the mandible (Fig. 25.2).
- The reduction in the number of trabeculae is the least evident in the alveolar processes, possibly because of the forces applied to bone in this area by the teeth.
- Occasionally the lamina dura may appear thinner than normal. In other regions of the mandible, a reduction in the number of trabeculae may be evident.



FIG. 25.2 Generalized osteopenia is evident as a loss of the normal trabecular bone pattern. Also, there is heterogeneity and thinning of the normal inferior cortex of the mandible. Note the prominence of the dentition on the background of the demineralized bone.

RICKETS AND OSTEOMALACIA

- The term *rickets* is applied when the disease affects the growing skeleton in infants and children, whereas the term *osteomalacia* is used when this disease affects the mature skeleton in adults.
- Rickets and osteomalacia may develop as a result of a lack of vitamin D or calcium in the diet, malabsorption of vitamin D in the gastrointestinal tract, or an inability to synthesize the active metabolite $1,25(\text{OH})_2\text{D}$, which is required for the gastrointestinal absorption of calcium.

CLINICAL FEATURES

- Children with rickets usually have short stature and deformity of the extremities.
- Swelling of the wrists and ankles.
- Eruption and development of the dentition is delayed.
- Enamel and dentin may be hypocalcified.
- Most patients with osteomalacia have some degree of bone pain as well as muscle weakness of varying severity. “penguin” gait, tetany, and greenstick fractures of the bone

IMAGING FEATURES EFFECTS ON THE TEETH AND JAWS.

- Rickets in infancy or early childhood may result in the hypoplasia of developing dental teeth, including the enamel
- involve both unerupted and erupted teeth
- retarded tooth eruption in early rickets.
- The lamina dura and the cortical borders of tooth follicles may be thin or missing.
- Jaw cortices such as the inferior cortex or the borders of the inferior alveolar canals may be thin
- reduced in density, number, and thickness; this imparts a generalized radiolucency to the jaws



FIG. 25.3 The bite-wing image shows thinning (hypoplasia) and decreased mineralization (hypocalcification) of the enamel in a child with rickets. (Courtesy H. G. Poyton, DDS, Toronto, ON, Canada.)

- Osteomalacia does not alter the teeth or the jaws because they are already fully developed before the onset of the disease. However, when manifestations are present in diagnostic images, there may be an overall radiolucent appearance to the bone, and the trabeculae may be sparse. In patients with long-standing or severe osteomalacia, the lamina dura may be thin.

HYPOPHOSPHATEMIC RICKETS

- Inherited conditions that produce renal tubular disorders causing an inability to resorb phosphorus in the distal renal tubules and resulting in a decrease in serum phosphorus (hypophosphatemia)
- The teeth may be poorly formed, with thin enamel and large pulp chambers and root canals .
- periapical and periodontal abscesses may occur frequently.
- The occurrence of rarefying osteitis without an apparent etiology may be a result of defects in the dentin and large pulp chambers allowing for the ingress of oral microorganisms and subsequent pulp necrosis.
- If the disease is severe, the patient will have premature loss of the teeth.
- The lamina dura may be sparse, and the cortices around tooth crypts may be thin or entirely absent.

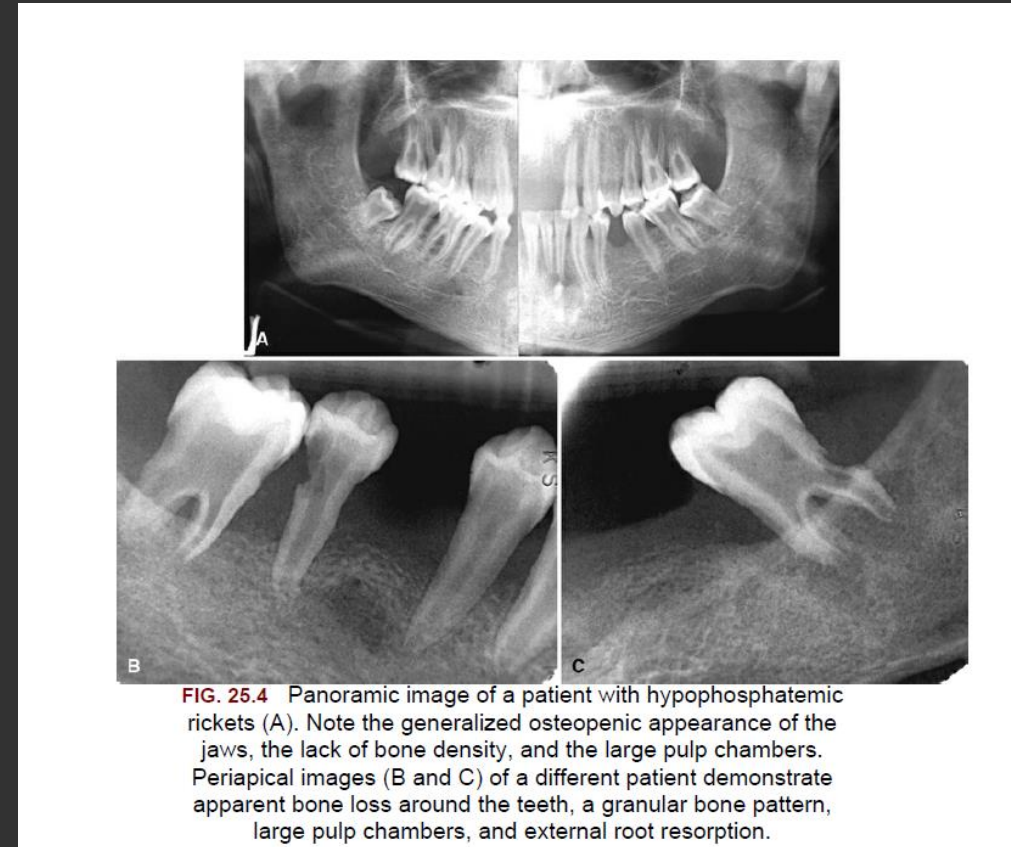


FIG. 25.4 Panoramic image of a patient with hypophosphatemic rickets (A). Note the generalized osteopenic appearance of the jaws, the lack of bone density, and the large pulp chambers. Periapical images (B and C) of a different patient demonstrate apparent bone loss around the teeth, a granular bone pattern, large pulp chambers, and external root resorption.

HYPOPHOSPHATASIA

- Hypophosphatasia is a rare inherited disorder caused by a **reduction in the activity of the tissue nonspecific alkaline phosphatases**, one of a family of enzymes produced by osteoblasts and odontoblasts that is required for the normal mineralization of osteoid and teeth.
- Both primary and permanent teeth have a thin enamel layer and large pulp chambers and root canals.
- The teeth may also be hypoplastic and may be lost prematurely.
- In the jaws, there is a generalized osteopenic appearance to the bones.
- The cortical bone and lamina dura are thin, and the alveolar processes are poorly calcified and may appear deficient.

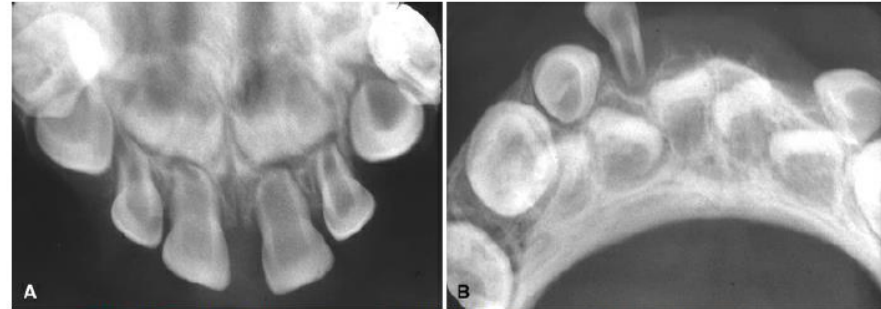


FIG. 25.5 Note the very large pulp chambers in the deciduous dentition in a patient with hypophosphatasia (A) and premature loss of the mandibular incisors (B) on these anterior occlusal images. (Courtesy Dr. H. G. Poyton, Toronto, ON, Canada.)

OSTEOPETROSIS

- Osteopetrosis, otherwise known as marble bone disease or Albers-Schönberg disease, is an inherited disorder of bone that results from a **defect in the differentiation and function of osteoclasts**.

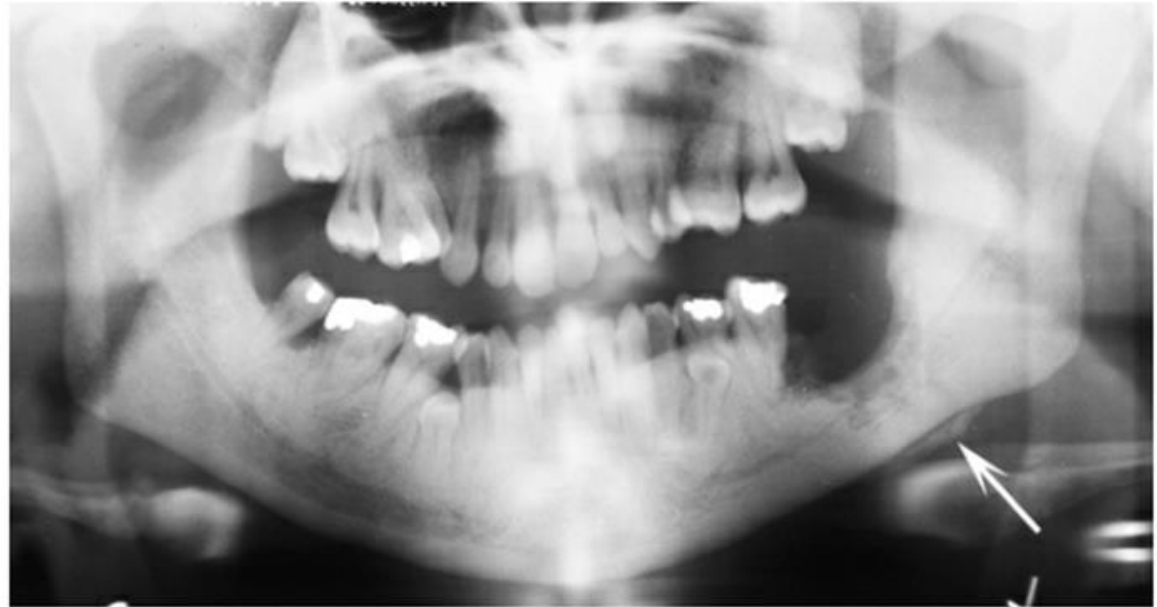


FIG. 25.6 A significant and generalized increase in the radiopacity of the bones is seen on this panoramic image of a patient with osteopetrosis. The inferior alveolar canals are prominently seen and narrowed. Also, there is osteomyelitis in the body of the left mandible with periosteal new bone formation (*arrow*).

- Effects on teeth may include early tooth loss, missing teeth, malformed roots and crowns, and teeth that are poorly calcified and prone to caries.
- The normal eruption pattern of the primary and secondary dentitions may be delayed as a result of the increased bone density or ankylosis.
- The lamina dura and cortical borders may appear thicker than normal.
- The increased radiopacity of the jaws may be so great that the diagnostic image may fail to reveal any internal structure, and even the roots of the teeth may not be apparent.
- The normal interface between cortical and cancellous bone may be lost, and in the mandible, the inferior alveolar canal may appear very prominent.
- On plain imaging, the image of the bone may appear underexposed due to failure of the incident x-ray beam to penetrate the bone.

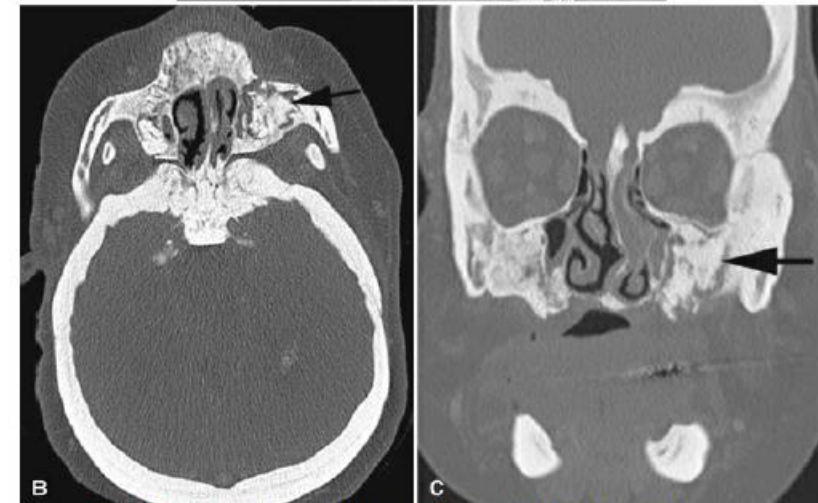


FIG. 25.7 Sagittal (A), axial (B), and coronal (C) multidetector computed tomography images show dense calcification of the bones in a patient with osteoporosis. Note the loss of definition of the cortical and cancellous bone interfaces and the uniformly increased density of all the bones. The case is complicated by osteomyelitis of the left maxilla with development of sequestra (arrows in B and C).

ENDOCRINE DISTURBANCES HYPERPARATHYROIDISM

- Hyperparathyroidism is an endocrine abnormality in which there is an excess of circulating parathyroid hormone (PTH).
- This excess favors osteoclastic resorption of bone, which mobilizes calcium from the skeleton.
- The net result of these changes is an increase in serum calcium levels



FIG. 25.8 This panoramic image shows generalized osteopenia in a patient with hyperparathyroidism. Note how the teeth stand out in contrast to the osteopenic bone.

IMAGING FEATURES

- In children, there may be hypoplasia and hypocalcification of the teeth, sometimes resulting in loss of any evidence of enamel in diagnostic images.
- loss of lamina dura
- root a tapered appearance because of loss of image contrast.
- mature teeth are immune to the systemic demineralizing process
- thinning of bone cortices including the inferior border of the mandible
- The density of the jawbones is decreased
- ground-glass appearance of the jaws
- In the skull, the calvarium may take on a granular appearance that is classically known as the “salt and pepper” skull

Brown tumors of hyperparathyroidism:

- They are giant cell lesions, appear in bone, and these are frequently found in the facial bones and jaws, particularly in cases of long-standing disease.
- The lesions are called brown tumors because of their brown or reddish-brown appearances on gross examination.
- Brown tumors may be solitary or multiple within a single bone.
- If a giant cell lesion occurs later than the second decade of life, the patient should be screened for increases in serum calcium, PTH, and alkaline phosphatase.



FIG. 25.11 Periapical images (A and B) show loss of lamina dura

around the teeth in a patient with hyperparathyroidism. Also, note the loss of definition of the floor of the maxillary antrum. The cropped panoramic image (C) of the same patient reveals a central giant cell lesion (brown tumor) developing apical to the mandibular right second and third molars.

HYPOPARATHYROIDISM AND PSEUDOHYPOPARATHYROIDISM

- Imaging of the jaws may reveal dental enamel hypoplasia, external root resorption, delayed eruption, or root dilaceration

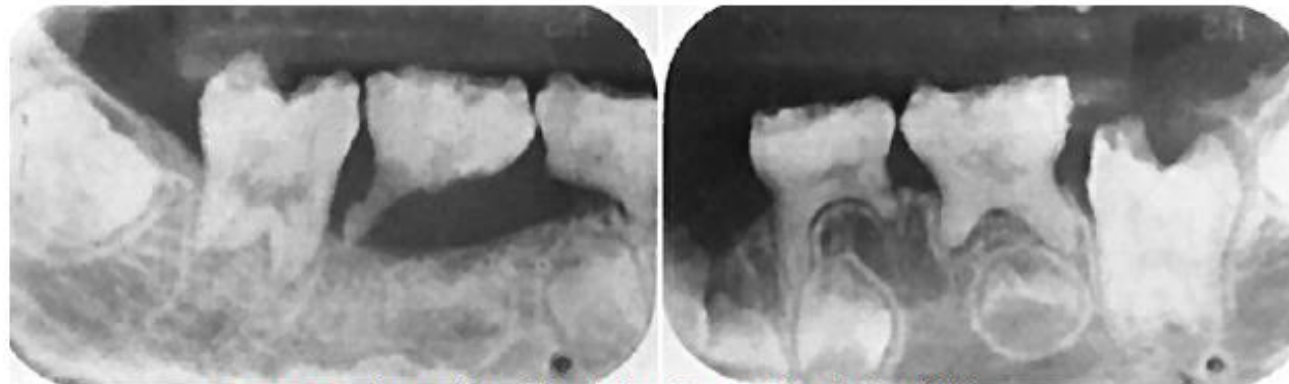


FIG. 25.13 Anomalies of Tooth Development in a Patient With Pseudohypoparathyroidism. (Courtesy Dr. S. Bricker, San Antonio, TX.)

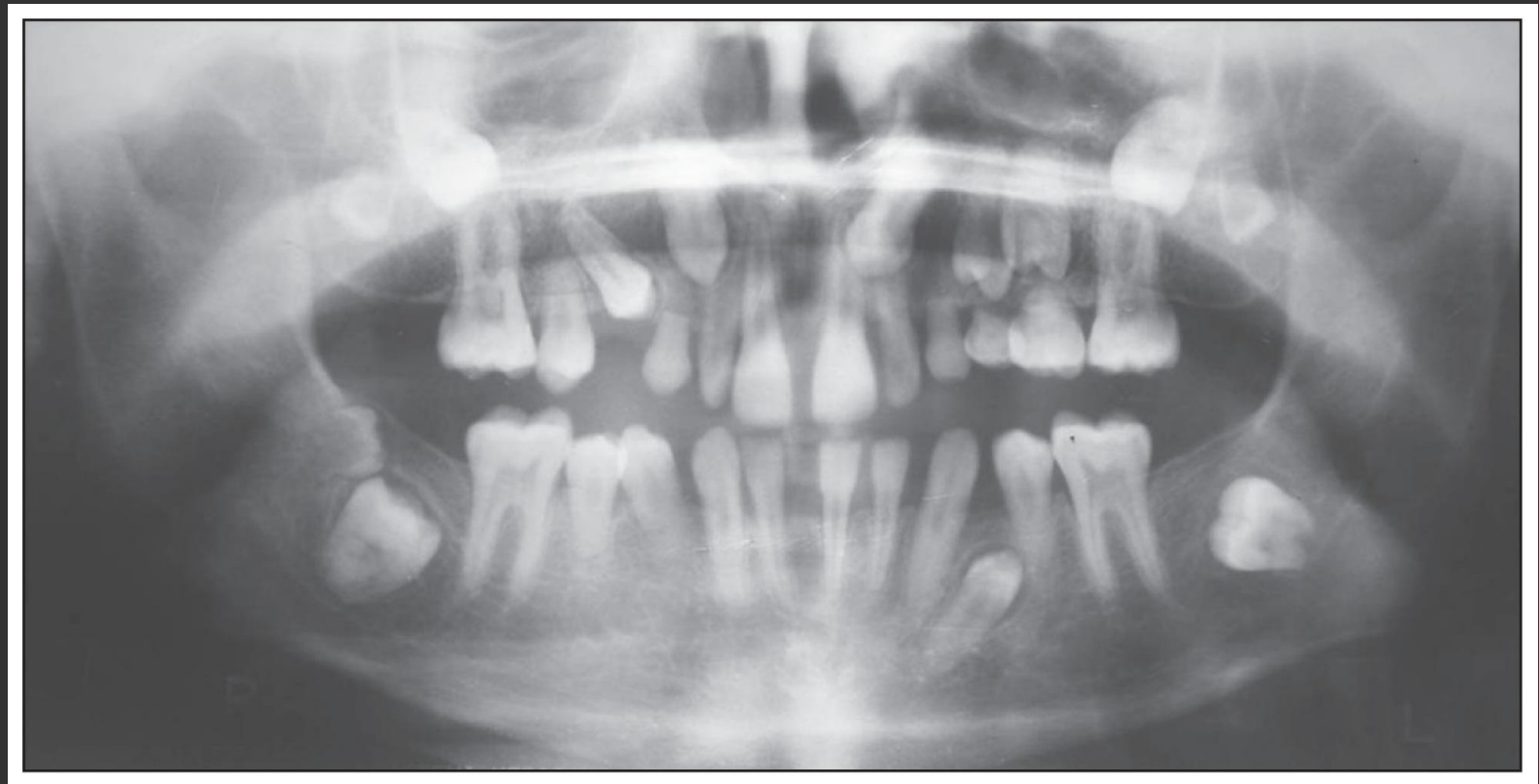
HYPERPITUITARISM

- Hyperpituitarism in the form of gigantism and acromegaly results from hyperfunction of the anterior lobe of the pituitary gland.
- Supraeruption of the posterior teeth may occur in an attempt to compensate for the growth of the mandible.
- Hyperpituitarism causes enlargement of the jaws, most notably the mandible and spacing of the teeth
- enlargement of the tongue (macroglossia)
- anterior open bite



HYPOPITUITARISM

- Eruption of the primary dentition occurs at the normal time, but exfoliation is delayed by several years.
- The crowns of the permanent teeth also develop normally, but their eruption is delayed several years.
- The third molar buds may be completely absent.



BONE DYSPLASIAS

Bone dysplasias include a group of conditions in which normal bone is replaced with fibrous connective tissue and abnormal, immature bone.

CEMENTO-OSSEOUS DYSPLASIA

- The cemento-osseous dysplasias are disease processes whereby one or more focal areas of normal bone metabolism are altered and cancellous bone is replaced by a mixture of fibrous connective tissue containing varying amounts of immature, abnormal bone.
- Periapical cemento-osseous dysplasia (PCOD) is the more localized form, affecting one sextant of the jaws.
- Florid cemento-osseous dysplasia (FCOD) is the more generalized form.



FIG. 25.22 The mixed radiolucent and radiopaque appearance of a more mature stage of periapical cemento- osseous dysplasia. Note the radiolucent rim encircling the radiopaque foci of dysplastic bone.



FIG. 25.26 Panoramic Image Showing Multiple Stages of Maturation of Florid Cemento-osseous Dysplasia. A simple bone cyst is developing at the apex of the mandibular right second premolar. (Courtesy Dr. C. Poon-Woo, Toronto, ON, Canada.)

- PCOD is a common bone dysplasia that typically occurs during the fourth and fifth decades of life.
- more often in women than men
- non-Caucasian populations
- teeth are vital, no history of pain or sensitivity.
- Can cause notable expansion of the alveolar process and thinning of adjacent cortices.
- Extensive lesions often have an associated bony swelling.
- Should lesions of FCOD become secondarily infected, some of the features of osteomyelitis may develop

Imaging Features

- The epicenter of an cemento-osseous dysplasia lesion is located in the periapical areas of the teeth, within the alveolar processes of the jaws
- the periphery of a PCOD lesion is well defined, and the cortex can vary in width to the point of appearing sclerotic
- Internally there may be a variably wide radiolucent rim surrounding the radiopaque center, which sometimes may be difficult to visualize in mature lesions
- As the lesions mature, a band of variably thick, almost sclerotic bone can appear.
- The lesion may be irregularly shaped or may have an overall round or oval shape centered at the apex of the tooth.
- The internal density of larger FCOD lesions can vary from an equal mixture of radiolucent and radiopaque regions to almost complete radiopacity.

Small lesions do not expand the jawbone; however, larger lesions can cause expansion of the jaw

FIBROUS DYSPLASIA

- Fibrous dysplasia is a disease process wherein normal bone metabolism is altered, and cancellous bone is replaced by a mixture of fibrous connective tissue containing varying amounts of immature, abnormal bone.
- The most common sites include the ribs, femur, tibia, maxilla, and mandible.
- McCune-Albright syndrome - Polyostotic fibrous dysplasia
- Monostotic fibrous dysplasia

Imaging Features

- Fibrous dysplasia affects the maxilla almost twice as often as the mandible and is more frequently seen in the more posterior regions of the jaw.
- The periphery of fibrous dysplasia lesions is most commonly poorly defined (blended)
- The internal density of the dysplastic bone may be radiolucent, radiopaque, or a mixture

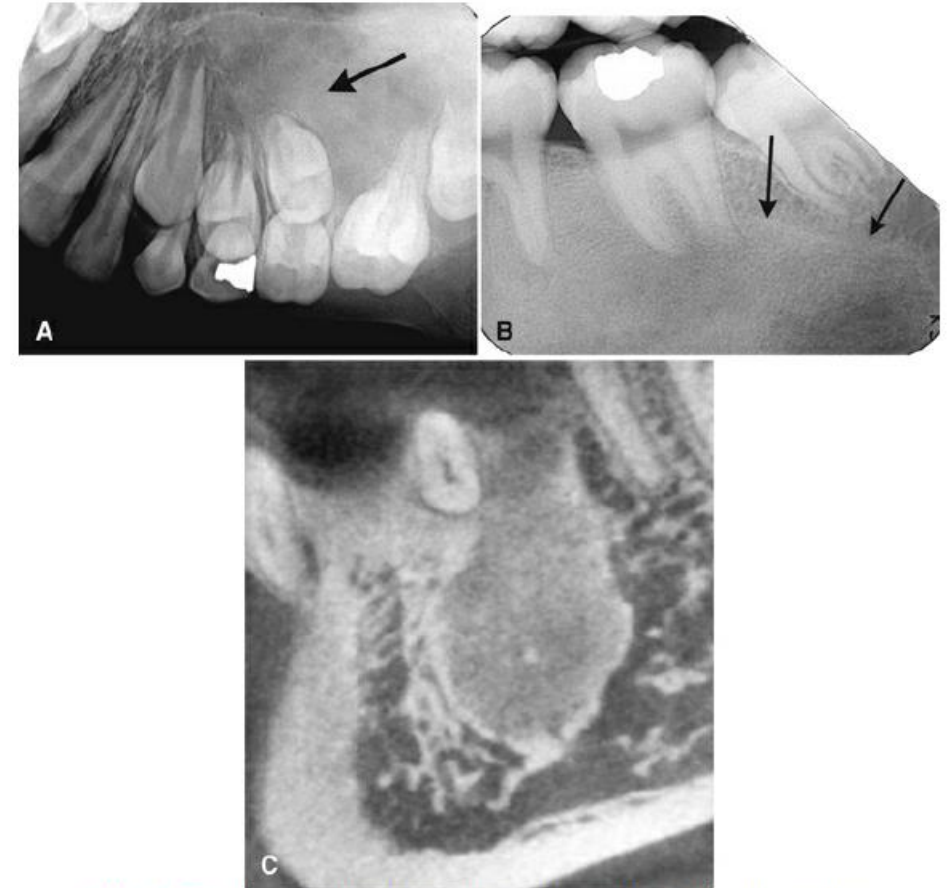


FIG. 25.30 This dysplastic granular bone pattern blends into the adjacent normal bone pattern in the region of the unerupted maxillary canine (A). In contrast (B), the border in this case of fibrous dysplasia of the mandible has a better-defined, almost corticated border (*arrows*). This sagittal cone beam computed tomography image of a small focus of fibrous dysplasia (C) has a border that appears more definitively corticated.

lamina dura disappears as the dysplastic bone engages this structure. If the fibrous dysplasia increases the bone density, the PDL space may appear to be narrow. Fibrous dysplasia can displace teeth or interfere with normal eruption, complicating orthodontic therapy. In rare cases, some root resorption or hypercementosis may occur.



FIG. 25.32 A Series of Images Showing the Range of Internal Patterns of Fibrous Dysplasia. A periapical image (A) shows the fingerprint-like pattern around the roots of the first molar (arrow). Note the change in the lamina dura around the molars into the abnormal bone pattern. A periapical image (B) shows a granular or ground-glass pattern (arrow). The cropped panoramic image (C) shows a more heterogeneous cotton-wool pattern. Note the almost circular radiopaque regions (arrows). The periapical image (D) shows the more stippled orange-peel pattern. A buccolingual cone beam computed tomography image (E) shows a granular internal bone pattern with strands of more amorphous bone (arrow).

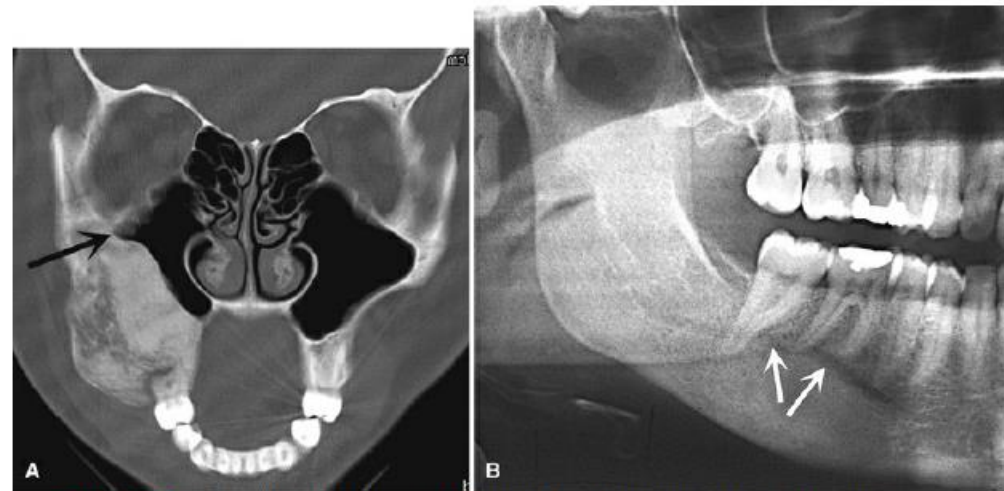


FIG. 25.35 Coronal multidetector computed tomography image of a region of fibrous dysplasia in the right maxilla (A) shows nonconcentric expansion of the lateral surface of the maxilla and the residual airspace within the maxillary sinus. Note that the right maxillary sinus appears as a smaller version of the unaffected left maxillary sinus and the retained right zygomatic recess (arrow). The cropped panoramic image (B) shows superior displacement of the inferior alveolar nerve canal by a focus of fibrous dysplasia (arrows).

DDX

- rarefying osteitis
- PCOD
- Healing of a simple bone cyst
- Hyperparathyroidism
- Paget disease of bone
- Osteomyelitis
- Cementoossifying fibroma
- Osteosarcoma

PAGET DISEASE OF BONE

- Abnormal osteoclasts produce an intense wave of bone resorption; after a period of time, this is followed by vigorous osteoblastic activity, forming poor-quality woven bone.
- The affected bone is enlarged and commonly deformed because of the poor quality of bone formation.
- there is enlargement of the jaws, which can cause tooth movement, diastema, and malocclusion. Dentures may become tight or may fit poorly in edentulous patients.

IMAGING FEATURES

- Whenever the jaws are involved, the maxilla is affected approximately twice as often as the mandible.

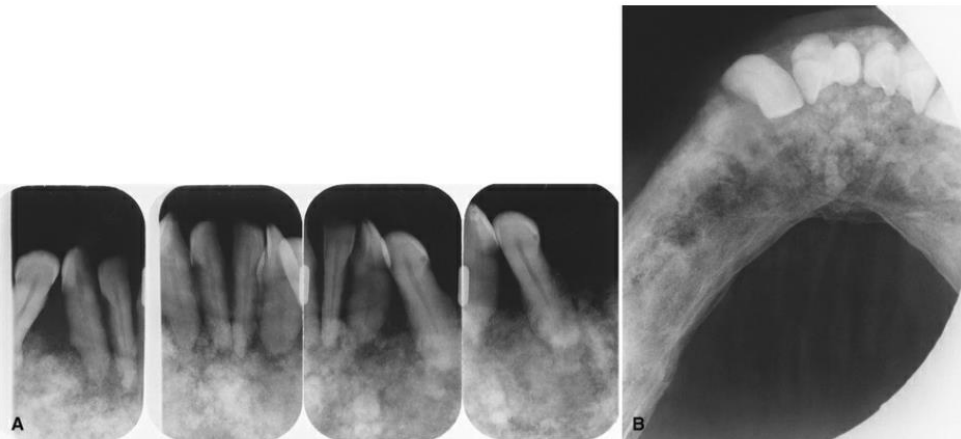


FIG. 25.38 Periapical and occlusal images of Paget disease of bone showing multiple radiopaque masses in the mandible with a cotton-wool appearance (A) and mandibular expansion (B). Note maintenance of the thin cortices on the occlusal image.



FIG. 25.37 Sagittal (A) and coronal (B) multidetector computed tomography images in a patient with Paget disease of bone. Note increase in thickness of the bones and the bone density.

- DDX
- fibrous dysplasia
- cemento-osseous dysplasias
- Metabolic
- Bone diseases

Effects on adjacent teeth.

The lamina dura may become less evident and may be altered into the abnormal bone pattern. Hypercementosis often develops on a few or most of the teeth in the involved jaw. This hypercementosis may be exuberant and irregular, which is characteristic of Paget disease (Fig. 25.39).

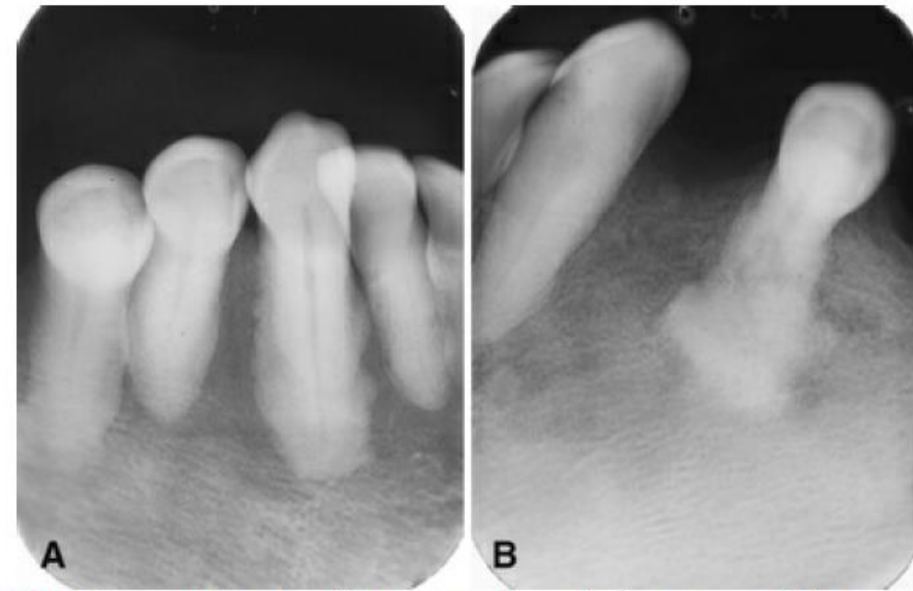


FIG. 25.39 Two periapical images Paget disease of bone showing exuberant hypercementosis of the roots.



