

CHAPTER 7
APICAL LESIONS

Periapical granuloma

Radicular cyst

Apical abscess

Apical scar

Surgical defect

Periodontal disease

Condensing osteitis

Osteosclerosis

Socket sclerosis

Periapical cemental dysplasia

Cementifying fibroma (ossifying fibroma)

Benign cementoblastoma

Florid osseous dysplasia

APICAL LESIONS

Whenever a lesion is observed on a radiograph, it must first be described in general terms before a differential diagnosis is attempted. Is the lesion radiolucent, radiopaque, or mixed (combination of radiolucency and radiopacity)? Where is the lesion located? The apices of which teeth are involved? What is the size of the lesion? Is the margin of the lesion ill-defined, well-defined, or well-defined with a radiopaque border? Is the appearance of the bone surrounding the lesion: normal, porous, or sclerotic?

The various radiographic appearances of the margins of lesions and the changes in the surrounding bone have been given clinical interpretation by some diagnosticians based largely on intuitive analysis rather than on research data. Although the significance of these signs is sometimes questionable, they are useful in radiographic interpretation. An ill-defined (diffuse, irregular) periphery is suggestive of a lesion enlarging by invading the surrounding bone. A well-defined (circumscribed) periphery is suggestive of a self-contained lesion enlarging by expansion. A well-defined periphery with a hyperostotic (sclerotic) radiopaque periphery is suggestive of an extremely slow-growing self-contained lesion enlarging by expansion.

If the bone surrounding a lesion does not show any change, the clinical interpretation is that of a static lesion. If it shows porosity (destructive breakdown), the interpretation is that of an invasive process resulting from osteolytic activity. If it shows sclerosis (hyperostosis), the interpretation is that of resistance to the pathologic process resulting from osteoblastic activity.

TYPES OF BORDERS

- Fig. 7-1 Well-defined radiolucent lesion at the apex of the maxillary lateral incisor. The circumscribed radiopaque (sclerotic) border signifies that the lesion is self-contained, enlarges by expansion, and is slow-growing. There is no change in the surrounding bone.
- Fig. 7-2 Well-defined radiolucent lesion at the apex of the mandibular left second premolar. The well-defined border signifies that the lesion is self-contained, and enlarges by expansion. The surrounding bone shows slight sclerosis (osteoblastic activity) which signifies resistance to the pathologic process.
- Fig. 7-3 A radiolucent lesion with diffuse, irregular borders at the apices of the roots of the maxillary left first molar. The diffuse margins, and the porosity (osteolytic activity) in surrounding bone signify an invasive process.

PERIODONTAL SPACE WIDENING (Apical periodontitis)

Radiographically, apparent widening (also called thickening) of the periodontal ligament space is caused by edema, resulting in accumulation of inflammatory exudate in the connective tissue of the periodontal ligament. The term apical periodontitis is often used to describe this pathologic periodontal space widening.

Pathologic periodontal space widening occurs as a result of infection, trauma, orthodontic treatment, or tooth extrusion. It is imperative that dentists be aware of trauma resulting from occlusal imbalance, especially that caused by an amalgam, gold, composite resin, or other restoration that is placed too high in the tooth and results in premature contact with the opposing tooth. Depending on the extent of the traumatic force and the individual's physiologic resistance, the widened periodontal space either returns to its normal appearance after the elimination of the trauma or may form a chronic apical inflammatory lesion such as an apical granuloma, a radicular cyst, or an apical abscess.

Non-pathologic periodontal space widening occurs as a result of the terminal stage of root formation (dental papilla), wide marrow space superimposed on a tooth apex, or superimposition of a tooth apex on a radiolucent anatomy (such as a nasal fossa, maxillary sinus, mental foramen or submandibular fossa) producing over-exposure or "burnout". An illusion of loss of lamina dura may also be produced when a tooth apex is superimposed on a radiolucent anatomy.

Fig. 7-4 Infection (caries) is the cause of the widened periodontal space of the first molar (apical periodontitis). In this case, the lamina dura is also thickened (radiopaque line).

Fig. 7-5 Trauma is the cause of the widened periodontal spaces of the central incisors (apical periodontitis). Clinically, the teeth were retruded.

- Fig. 7-6 Traumatic occlusion, that is, premature contact with the opposing premolars, is the cause of the widened periodontal spaces of the maxillary premolars (apical periodontitis). The patient also has alveolar bone loss due to periodontal disease.
- Fig. 7-7 Orthodontic treatment is the cause of the widened periodontal spaces of the incisors.
- Fig. 7-8 Developing root apices of the second molar may be mistaken for widened periodontal spaces. Root apices complete their formation approximately one to two years after tooth eruption.
- Fig. 7-9 Illusion of widened periodontal space of the root apex of the premolar because of superimposition on a radiolucent anatomy (maxillary sinus). The widened periodontal space of the canine may be due to a similar illusion and/or infection (caries). Also note the illusion of loss of apical lamina dura on both teeth.
- Fig. 7-10 Illusion of widened periodontal spaces of the root apices of the molar because of superimposition on a radiolucent anatomy (mandibular canal). Also note the illusion of apical loss of lamina dura.

APICAL LESION VERSUS ANATOMICAL LANDMARK

To differentiate an apical lesion from an anatomical landmark, make another radiograph with a different x-ray beam angulation. An anatomical landmark will change its position with a change in angulation of the x-ray beam. On the other hand, an apical lesion will maintain the same relationship to the involved tooth.

Another method of differentiation is to follow the continuity of the periodontal space. The periodontal space will be interrupted in the case of an apical lesion but will be continuous in the case of an anatomical landmark.

Fig. 7-11 A — Mental foramen. Notice the intact periodontal space.
 B — Chronic inflammatory apical lesion. Notice the loss of the apical periodontal space.

Fig. 7-12 Radiolucent anatomy mistaken for an apical lesion. Mental foramen superimposed on the root apex of the second premolar may be mistaken for an apical lesion.

PERIAPICAL GRANULOMA, RADICULAR CYST AND APICAL ABSCESS

(Inflammatory pulpo-periapical lesions)

The most common pathologic conditions that involve teeth are the inflammatory lesions of the pulp and periapical areas. Once inflammation (pulpitis) has spread from the dental pulp, it can produce a variety of apical pathologic changes, the most common of which are the periapical granuloma, radicular cyst, and apical abscess. Various factors such as the host's resistance and the virulence of the bacteria affect the local inflammatory response in the periapical area. Without a microscopic diagnosis, a clinician is frequently unable to differentiate between a periapical granuloma, a radicular cyst, and an apical abscess. Radiographic examination is inadequate in making a specific diagnosis.

Inflammatory periapical lesions have certain common clinical characteristics: 1) A history of painful pulpitis leading to the death of the pulp. 2) A nonvital reaction to electric pulp testing. In a multirrooted tooth where only one root is associated with the pulpo-periapical pathosis, the tooth will frequently give a vital reaction. 3) Presence of a deep carious lesion exposing the pulp or a restoration close to the pulp, or a fractured tooth and/or a discolored crown. 4) Destruction resulting in an interrupted lamina dura of the involved tooth.

A granuloma is formed from the successful attempt of the periapical tissues to neutralize and confine the irritating toxic products escaping from the root canal. This low grade inflammation in the tissues continues to induce the proliferation of vascular granulation tissue. A granuloma may evolve into a radicular cyst or an apical abscess. Clinically, the lesion is usually asymptomatic but may sometimes exhibit mild pain and sensitivity to percussion. The affected tooth is nonvital. Radiographically granulomas form small well-defined radiolucencies. They are the most common periapical lesions and constitute approximately 50% of all periapical radiolucent lesions.

A radicular cyst (also known as periapical cyst, dental cyst, periodontal cyst) has its origin from the cell rests of Malassez which are present in periodontal and periapical ligament, and in periapical granulomas. Most radicular cysts originate from pre-existing granulomas.

Clinically, the lesion is usually asymptomatic but may sometimes exhibit mild pain and sensitivity to percussion. The affected tooth is nonvital. A radicular cyst may slowly enlarge and when large, may cause expansion of the cortical plates. Radiographically, a radicular cyst forms a large well-defined radiolucency with or without a radiopaque (hyperostotic) border. The more pronounced the hyperostotic (sclerotic) border, the more likely is the lesion to be a radicular cyst. It is the second most common periapical lesion and constitutes approximately 40% of all periapical radiolucent lesions. (See also chapter on "Cysts of the Jaws").

In approximately 90% of the cases, a well-defined radiolucency at the apex of an untreated asymptomatic tooth with a nonvital or diseased pulp is either a dental granuloma or a radicular cyst. Their sizes are the differentiating features: a granuloma is small whereas a radicular cyst is large. In practice, however, it is not necessary to differentiate a periapical granuloma from a radicular cyst because both lesions respond quite well to conservative root canal therapy.

An apical abscess, also called dental or dentoalveolar abscess, usually develops from a pulpo-periapical inflammatory condition. In the acute stage, the onset of infection is so sudden that there is no radiographic evidence of an apical lesion. An apical abscess can develop also from a pre-existing granuloma or cyst. The associated tooth is nonvital, very painful, extremely sensitive to percussion, and often slightly extruded. The patient will complain that the tooth feels "high" when it occludes with the opposing tooth. The tooth will not respond to electrical pulp tests. The application of ice will slightly relieve the pain but the application of heat will intensify pain. The tooth may demonstrate increased mobility. Radiographically apical abscesses form large radiolucencies with diffuse irregular borders. They are the least common of the three pulpo-periapical lesions and constitute approximately 2% of all periapical radiolucent lesions.

If an apical abscess is permitted to progress without treatment, it may penetrate the cortical plate at the thinnest and closest part of the tooth apex and form a swelling in the adjacent

soft tissues. This infection site in the soft tissues is painful. The skin or mucous surface over the abscess is warm and rubbery to palpation, and demonstrates fluctuance. Once drainage is established by a sinus tract permitting the pus to drain to the surface, the tooth and associated swelling are no longer painful since the pain-producing pressure of the abscess is reduced. The regional lymph nodes may be enlarged and painful. The systemic temperature may be elevated.

Fig. 7-13 Presence of either a periapical granuloma, a radicular cyst or an abscess at the root apices of the first molar. The small, well-defined radiolucency is suggestive (on a speculative basis) of a periapical granuloma. Periapical granulomas are the most frequently occurring of the three pulpo-periapical lesions. The involved tooth is nonvital.

Fig. 7-14 Presence of either a periapical granuloma, a radicular cyst or an abscess at the root apices of the right central and lateral incisors. The large, well-defined radiolucency with a radiopaque (sclerotic) border is suggestive (on a speculative basis) of a radicular cyst (periapical cyst). Radicular cysts are the second most frequently occurring of the three pulpo-periapical lesions. The involved tooth is nonvital.

Fig. 7-15 Presence of either a periapical granuloma, a radicular cyst or an abscess at the root apices of the first molar. The large radiolucency with diffuse irregular border is suggestive (on a speculative basis) of an abscess. Apical abscesses are the least frequently occurring of the three pulpo-periapical lesions. The involved tooth is nonvital.

APICAL SCAR

An inflammatory apical lesion treated by root canal therapy may respond well to treatment by filling new bone at the site of the lesion. However, the healing process may sometimes terminate abruptly and leave a small amount of dense scar tissue known as an apical scar. The scar tissue represents one of the possible end points of healing. It is composed of dense fibrous tissue and is situated at the apex of a pulpless tooth in which the root canals have been successfully filled. Microscopic examination reveals fibroblasts scattered in the collagen fibers. Unlike an apical granuloma, inflammatory cells are not a feature and vascularity is quite meager. An apical scar is a small, asymptomatic, and well-circumscribed radiolucency. When observed radiographically over the years, it will either remain constant in size or diminish slightly.

Fig. 7-16 Apical scar is an area at the apex of a tooth that fails to fill in with osseous tissue after endodontic treatment. The second premolar was successfully treated with root canal therapy. The apical radiolucency persisted after treatment. Patient was recalled after several months. The radiolucency slightly decreased in size but was still present.

Fig. 7-17 Apical scar. The molar was successfully treated with root canal therapy. Patient was recalled after several months. The apical radiolucency reduced in size and persisted.

SURGICAL DEFECT

A surgical defect is that portion of bone that fails to form osseous tissue. It is frequently seen periapically after root resection in which the site is filled with dense fibrous (collagen) tissue instead of bone. It is an asymptomatic persistent radiolucency. An extraction site can also form a surgical defect. Approximately 75% of all surgically treated periapical radiolucencies require 1 to 10 years or longer for complete resolution. In the remaining 25%, complete healing does not occur.

Fig. 7-18 Surgical defect in bone is an area that fails to fill in with osseous tissue after surgery. An apicoectomy was performed at the apex of the left central incisor and the lesion was curetted. The radiolucency persisted as a surgical defect.

Fig. 7-19 Surgical defect at the extraction site of the mandibular third molar. The radiolucency of the tooth socket persisted after 1 year.

PERIODONTAL DISEASE

Periodontal disease is discussed in detail in a previous chapter. Nevertheless, it is considered here because it produces a relatively common periapical radiolucency in the advanced stages. The entire bony support of the involved tooth may be completely destroyed or the tooth may appear to be floating in a radiolucency. Sometimes a narrow vertical pocket may extend to the apex and appear as a well-defined periapical radiolucency. This may lead the unwary clinician to a false conclusion of a pulpo-periapical pathosis. In the advanced stages, these teeth are usually quite mobile and may become sensitive to percussion; but surprisingly many remain vital, and the demonstration of such vitality aids the clinician in differentiating a periodontal disease radiolucency from an inflammatory apical radiolucency (periapical granuloma, radicular cyst and apical abscess). A clinical examination of supporting tooth structures should be undertaken by probing all periodontal pockets.

Fig. 7-20 Periodontal disease producing a periapical radiolucency. Vertical bone loss around the second premolar extends to the apex and appears as a fairly well-defined radiolucency. The affected tooth is vital.

Fig. 7-21 Periodontal disease producing a periapical radiolucency around the mesial root of the second molar. Vertical bone loss extends to the apex of the root. The affected tooth is vital.

CONDENSING OSTEITIS (Focal sclerosing osteomyelitis)

Condensing osteitis is a reaction of bone induced by inflammation. It occurs mainly at the apex of a tooth from an infected pulp. The infection from tooth caries reaches the pulp and progresses to the apical tissues to produce a small periapical radiolucency called rarefying osteitis. The small rarefying osteitis may be either a periapical granuloma, a radicular cyst or an abscess. The bone surrounding this rarefying osteitis becomes dense in order to prevent further spread of the lesion. This dense radiopacity surrounding the rarefying osteitis is called condensing osteitis. The pulp of the involved tooth is nonvital.

1. Condensing osteitis is a reaction to periapical infection resulting in the formation of dense bone. The infection usually originates from caries (sometimes from periodontal disease).
2. Condensing osteitis occurs usually at the apex of a nonvital tooth. On a radiograph, the tooth may exhibit a large carious lesion or a large restoration close to the dental pulp.
3. Radiographically, the lesion shows a diffuse radiopacity surrounding a small central radiolucency at the apex (or apices) of a tooth.
4. Treatment consists of removing the infection either through tooth extraction or root canal therapy.
5. Prognosis - the high radiopacity of bone tends to disappear partially or completely in a majority of cases after treatment is given to remove the infection. Approximately 30% of the cases will not resolve to normal appearing bone after adequate treatment. The persistence of the radiopacity should not be of concern to the clinician because of the innocuous nature of the lesion.

Fig. 7-22 Condensing osteitis at the root apices of the carious first molar. Condensing osteitis is a reaction to an inflammatory process. Note the diffuse

radiopacities (condensing osteitis) surrounding the apical radiolucencies (rarefying osteitis). The involved tooth is nonvital.

Fig. 7-23 Condensing osteitis at the root apices of the carious first molar. Note the diffuse radiopacities (condensing osteitis) surrounding the apical radiolucencies (rarefying osteitis). The involved tooth is nonvital.

Fig. 7-24 Condensing osteitis at the root apices of the carious first and second molars. The involved teeth are nonvital.

OSTEOSCLEROSIS AND SOCKET SCLEROSIS

Osteosclerosis (enostosis) is one of the common bony lesions found incidentally on a radiographic examination. It is a well-defined radiopaque mass without any associated radiolucency. The cause of the lesion is not known (idiopathic). The lesion is asymptomatic and the teeth associated with it are invariably healthy with vital pulps. There is neither any pain nor any cortical bone expansion. The lesion may occur either in the alveolus, in the periapical region, in the intra-radicular or inter-radicular region, below the crest of the ridge, or in the body of the mandible. It has been suggested that excess occlusal stress may be one of the factors in osteosclerosis associated with roots of teeth. When it occurs near the first and second premolars, its formation is speculated to be that of deposition of sclerotic bone around the root fragment of a deciduous molar which acts as a nidus. It has been reported that only 10% to 12% of patients with osteosclerosis show root resorption of an adjoining tooth.

When osteosclerosis occurs in the socket of an extracted tooth as a reparative process, it is known as socket sclerosis. Radiographically, it is often impossible to distinguish socket sclerosis from a retained root of a tooth. A dentist may be wrongfully accused of having failed to remove the tooth in its entirety.

1. Osteosclerosis refers to a localized region of abnormally dense bone. The cause of the sclerotic bone is either idiopathic or reparative. It is not associated with any infection.
2. Osteosclerosis can occur in any part of the jaw even at the apices of vital teeth. Socket sclerosis occurs in the sockets of extracted teeth and may sometimes be mistaken for retained roots.
3. Radiographically, the lesion shows a well-defined radiopacity. There is no association with any radiolucency. 10% to 12% of patients show root resorption.
4. No treatment is necessary.
5. Prognosis - the radiopacity persists without any increase in size.

- Fig. 7-25 Osteosclerosis at the root apices of the first molar. The involved tooth is vital.
- Fig. 7-26 Osteosclerosis between the second premolar and first molar, and at the mesial root apex of the first molar. The involved tooth is vital. 10% to 12% of the patients show root resorption.
- Fig. 7-27 Osteosclerosis at the distal root of the second molar.
- Fig. 7-28 Osteosclerotic bone to the mesial of the molar, preventing the tooth from further tilting or drifting.
- Fig. 7-29 Socket sclerosis. Sclerosing bone deposited in the socket of the extracted tooth. A dentist may be erroneously accused of having failed to extract the root. There are no definite radiographic criteria of distinguishing a retained root from a socket sclerosis.
- Fig. 7-30 Socket sclerosis. Although there are no definite radiographic criteria of distinguishing a retained root from a socket sclerosis, in this case, the diffuse border distinguishes socket sclerosis from a retained root.
- Fig. 7-31 Socket sclerosis between the second premolar and the mesially drifted second molar.

Fig. 7-32 Osteosclerosis around the walls of the nutrient canal at the apex of the mesial root of the first molar. A nutrient canal wall acts as a nidus for deposition of sclerotic bone. The involved tooth is vital.

Fig. 7-33 Osteosclerosis around the walls of the nutrient canal at the distal root apex of the first molar. A nutrient canal wall acts as a nidus for deposition of sclerotic bone. The involved tooth is vital.

Fig. 7-34 Retained root tips of deciduous second molar. It is hypothesized that a retained deciduous root tip acts as a nidus for osteosclerosis.

Fig. 7-35 Hyperostosis is osteosclerotic bone that appears on the crest of the alveolar ridge.

Fig. 7-36 Hyperostosis beneath a bridge pontic.

CEMENTOMAS

- a. Periapical cemental dysplasia (periapical cementoma).
- b. Cementifying fibroma (ossifying fibroma, cemento-ossifying fibroma)
- c. Benign cementoblastoma (true cementoma)

Periapical cemental dysplasia, cementifying fibroma, ossifying fibroma, cemento-ossifying fibroma, benign cementoblastoma, and florid osseous dysplasia have been grouped by some under the term "benign fibro-osseous lesions of periodontal ligament origin." These benign fibro-osseous lesions of periodontal ligament origin are a group of cementum and bone producing tumors and tumor like proliferations that arise from the cells of the periodontal ligament. In this text, the florid osseous dysplasia is described as a separate entity.

All the three types of cementomas have three radiographic appearances:

- 1) early or osteolytic stage appears radiolucent.
- 2) mixed or cementoblastic stage appears as a radiolucency containing radiopacities.
- 3) final or calcified stage appears as a homogeneous radiopacity surrounded by a thin radiolucent border.

All cementomas are associated with teeth having vital pulps unless, otherwise involved with caries or trauma.

Periapical Cemental Dysplasia:

Black persons and females of 40 years and older are more commonly affected than white persons and males to develop periapical cemental dysplasia. Although periapical cemental dysplasia occurs usually in the mandibular anterior region, it may sometimes be found in the mandibular posterior region. On a radiograph, the early radiolucent stage of periapical cemental dysplasia could be misdiagnosed for inflammatory periapical radiolucencies (periapical granuloma, radicular cyst, abscess). An unalert clinician might needlessly extract or institute endodontic treatment on a tooth with a normal pulp. The periapical cemental dysplasia is associated with a vital tooth and is totally asymptomatic. These features are in contrast to those of an inflammatory lesion which is associated with a nonvital tooth and may exhibit symptoms.

Periapical Cemental Dysplasia (Periapical cementoma)	*Cementifying Fibroma (Ossifying fibroma, cemento-ossifying	*Benign Cementoblastoma (True cementoma)
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	fibroma)		
1.	Believed to be a reaction of periapical bone. Actual cause unknown. Asymptomatic.	Benign mesenchymal odontogenic neoplasm.	Benign mesenchymal odontogenic neoplasm.
2.	Average age is 40 years. Predominantly in black persons and in females.	In young and middle-aged adults.	In teenagers and young adults.
3.	Around apices of teeth. Usually mandibular anteriors.	Around apices of teeth. Usually the mandibular posteriors.	Attached to the apex or apices of mandibular molar or premolar.
4.	Vital teeth. Position of teeth not affected.	Vital teeth. Displacement of teeth or divergence of roots.	Vital teeth. Position of teeth not affected.
5.	Usually multilocular. Sometimes unilocular.	Usually unilocular.	Usually unilocular.
6.	Radiographic appearance has three developmental stages.	Radiographic appearance has three developmental stages.	Radiographic appearance when discovered is usually of the third stage.
7.	No expansion of cortical plates. Self-limiting lesion.	Expansion of cortical plates.	Expansion of cortical plates when large.
8.	No treatment necessary.	Excision of lesion.	Extraction of involved tooth and excision of lesion.

*For details, refer chapter on "Odontogenic Benign Tumors of the Jaws"

Fig. 7-37 Periapical cemental dysplasia (early stage). Multiple radiolucencies at the apices of the mandibular anterior teeth. In periapical cemental dysplasia, the teeth are vital unless otherwise involved with caries or trauma. The radiolucencies should not be misdiagnosed as inflammatory apical lesions (granulomas, cysts, abscess) in which the teeth are nonvital and maybe symptomatic.

Fig. 7-38 Periapical cemental dysplasia (early stage). Unilocular large radiolucency at the apices of vital mandibular anterior teeth. The radiolucency should not be misdiagnosed as an inflammatory apical lesion. In periapical cemental dysplasia, the teeth are vital and totally asymptomatic.

Fig. 7-39 Periapical cemental dysplasia (mixed stage). Multiple radiolucencies containing faint opacifications at the apices of the mandibular anterior teeth.

Fig. 7-40 Periapical cemental dysplasia (calcified stage). Each radiopacity is surrounded by a radiolucent border at the apices of mandibular incisor teeth.

Fig. 7-41 Cementoblastoma (early stage). Unilocular circular radiolucency at the apices of the mandibular first molar. The involved tooth is vital.

Fig. 7-42 Cementoblastoma (mixed stage). Unilocular circular radiolucency containing faint opacification. The involved tooth is vital.

Fig. 7-43 Cementoblastoma (calcified stage). A dense circular radiopacity surrounded by a radiolucent border at the apices of the mandibular first molar. A cementoblastoma is usually discovered radiographically in the calcified stage.

Fig. 7-44 Cementifying fibroma or ossifying fibroma (osteolytic stage) in the mandibular first and second molar region. Unilocular radiolucency containing faint opacification. Well-defined cortical margins.

Fig. 7-45 Cementifying or ossifying fibroma (calcified stage). Occlusal view shows buccal expansion of the mixed radiopaque-radiolucent lesion.

Fig. 7-46 Cemento-ossifying fibroma (mixed stage). The large unilocular radiolucency containing calcifications shows expansion of the cortical plate near the inferior border of the mandible.

FLORID OSSEOUS DYSPLASIA (Diffuse cementosis, Chronic diffuse sclerosing osteomyelitis, Gigantiform cementomas, Multiple enostoses, Chronic sclerosing cementosis)

Florid osseous dysplasia, also known as chronic diffuse sclerosing osteomyelitis, is a reactive type of fibro-osseous bone disease with a marked predilection for black, middle-aged females of 40 years and older. Some pathologists consider this bony dysplastic lesion as an exuberant form of periapical cemental dysplasia. Mandibular involvement is more common than maxillary involvement. The teeth are vital unless otherwise involved with caries or trauma. The lesion consists of multiple dense radiopacities surrounded by narrow peripheral radiolucent rims. These radiopacities are symmetrically distributed in two or all four jaw quadrants. The coalescing radiopaque masses may sometimes obscure the peripheral radiolucent rims, resulting in diffuse radiopacities without any associated radiolucency. Usually there is no jaw expansion and is discovered incidentally during radiographic examination. Sometimes, advanced cases may show a painless expansion of the alveolar process; necessitating the need to adjust dentures. The lesion is restricted to the jawbones and must be differentiated from generalized conditions like Paget's disease and osteopetrosis.

Tooth extraction in the affected area exposes the avascular cementum to the oral cavity. This avascular cementum is prone to infection and may lead to chronic suppurative osteomyelitis with draining fistulous tracts.

- Fig. 7-47 Florid osseous dysplasia. Diffuse opaque masses with radiolucent borders involving both sides of the mandible.
- Fig. 7-48 Florid osseous dysplasia(also known as chronic diffuse sclerosing osteomyelitis). Pagetoid cotton-wool appearance with diffuse irregularly shaped radiopaque areas without any associated radiolucency and involving all four quadrants of the jaws.
- Fig. 7-49 Florid osseous dysplasia. Bilaterally located in the mandible, well-circumscribed, radiopaque masses surrounded by radiolucencies. In florid osseous dysplasia, the teeth are vital unless otherwise involved with caries or trauma.
- Fig. 7-50 Florid osseous dysplasia. Bilaterally located radiopaque masses surrounded by peripheral radiolucent borders similar to those of periapical cemental dysplasia.

MEDULLARY SPACES, AND TORI

Fig. 7-51 Large medullary space (large marrow space, wide inter-trabecular space) between the roots of the first molar. May be misdiagnosed as an apical lesion.

Fig. 7-52 Large medullary spaces (large marrow spaces, wide inter-trabecular spaces) between the roots of the first molar. May be misdiagnosed as a lesion.

Fig. 7-53 Mandibular tori superimposed on the roots of the lateral incisor, canine and first premolar.

Fig. 7-54 Torus palatinus (palatal torus).

Fig. 7-55 Torus palatinus (T) as seen on anterior films. May be misdiagnosed as osteosclerosis.

Fig. 7-56 4 small arrows — Exostosis evident on buccal surface of alveolar ridge. Exostoses are similar to torus palatinus and mandibular tori but are located on the buccal aspect of the alveolus.

R — Ramus of mandible

S — Anterior border of ramus

Periapical Radiolucencies

Granuloma

Radicular Cyst

Abscess

Apical scar

Surgical defect

Periodontal disease

Cementomas (osteolytic stage)

Chronic suppurative osteomyelitis (Described in chapter on "Osteomyelitis")

Periapical Radiopacities

Cementomas (calcified stage)

Condensing osteitis

Osteosclerosis

Socket sclerosis

Florid osseous dysplasia (diffuse cementosis)

Tori

Hypercementosis