IADMFR Education

Radiological Interpretation of Anomalies of the Maxillofacial Bones

Slide 1. Introduction: The program on the radiological interpretation of maxillofacial bone anomalies was developed for the education program of the IADMFR by Professors Noffke and Raubenheimer under the auspices of the Department of Imaging & Pathology, University of Leuven, Belgium. The goal of the presentation is to provide students in an under-resourced environment with the skills to interpret the radiological features of bone anomalies. In order to facilitate the understanding of the basic principles, the pathology will be highlighted as a background to the radiological interpretation thereof. When considering the vast quantity of knowledge in the field, it is clear that students will be required to consult prescribed textbooks in order to master the contents at a postgraduate level. The list of prescribed textbooks is available through the course coordinator.

Slide 2: The discussion of maxillofacial bone anomalies will follow an etiology-based classification. The categories are grouped in anatomical variations, developmental anomalies and dysplastic bone lesions, idiopathic bone conditions, metabolic bone diseases, degenerative bone diseases, bone infections and bone tumors.

Slide 3: Under anatomical jaw bone variations we will discuss condylar hypo- and hyperplasia, coronoid hypo- and hyperplasia, prominent and flat mandibular angles, enlargement of the incisive foramen, mental foramen and marrow spaces, lingual mandibular salivary gland depression and bifid condyle. There are several other anatomical variations and students are referred to the prescribed textbook for more information.

Slide 4: The general characteristics of anatomical jaw bone variations are their bilateral symmetrical manifestation and tendency to remain stable after cessation of skeletal growth. Most are recognized in childhood and they are only managed for esthetics and/or functional reasons when skeletal growth seizes.

Slide 5: Variations of the condylar anatomy may manifest unilateral or bilateral, mild and only recognizable on radiographs, or extreme, resulting in facial asymmetry. With condylar hyperplasia, one or both condyles may be larger than normal and the condylar necks are often elongated as demonstrated. The etiology of condylar hyperplasia is unknown.

Slide 6: Condylar hypoplasia may be unilateral or bilateral and manifest with smaller than normal condyles and often shortened or absent condylar necks. The image demonstrates condylar hypoplasia which may manifest clinically with jaw asymmetry with the midline of the mandible displaced to the hypoplastic side. The most common cause for condylar hypoplasia is destruction of the growth center of the mandible (that is located in the neck of the condyle of the developing mandible) by a middle ear infection which breaks through into the temporomandibular joint area.

Slide 7: Coronoid hypoplasia is an undersized mandibular coronoid process and, although mostly idiopathic, may be the result of factors which impact on the development and growth of the coronoid processes. Therapeutic radiation exposure during the treatment of childhood malignancies such as Burkitt's lymphoma and rhabdomyosarcoma may result in arrest of coronoid growth. The term coronoid aplasia is used when the coronoid processes are absent.

Slide 8: Coronoid hyperplasia, or elongation of the coronoid processes above the inferior border of the zygomatic arch may impinge on the zygomatic arch and limit the jaw opening. The term Jacob's disease is used if a pseudo joint develops between the surface of the zygomatic bone and the coronoid process.

Slide 9: Prominent mandibular angles can be the result of masseter hypertrophy due to bruxism or stress disorders.

Slide 10: Flat mandibular angles may be an indication of systemic disease e.g., systemic sclerosis in which case the condition is bilateral and fairly symmetric. If there is no underlying systemic disease, it is an anatomical variation and of no further clinical concern.

Slide 11: An incisive foramen can measure up to approximately 6mm in diameter. Larger dimensions increase the likelihood of a nasopalatine duct cyst which develops in the nasopalatine canal.

Slide 12: An enlarged mental foramen projected over the apex of one of the premolars is easily confused with an odontogenic cyst, e.g., radicular cyst (vitality testing important!). A correct angle of projection is crucial. Enlargement of the mental foramen may also be an indication of a benign neural or vascular tumor, e.g., neurofibroma or hemangioma (rare) which may develop from the contents of the canal (see later).

Slide 13: Enlarged marrow spaces, also referred to as focal osteoporotic bone marrow defects, are common in the body of the mandible. The internal structure of the defect is more radiolucent than the surrounding bone due to fewer bone trabeculae, and the periphery may be well- to ill defined. It is important to distinguish these defects from periapical granulomas and small radicular cysts where the associated tooth is non-vital. If the defect fails to enlarge with follow-up radiographs, the interpretation of an enlarged marrow space is correct. Advanced imaging can also provide additional imaging in this regard.

Slide 14: Although enlarged marrow spaces are idiopathic, they may develop at sites with reduced mechanical stimulation such as sites of tooth extraction.

Slide 15: Lingual salivary gland depression, also known as Stafne's bone cavity, is a well-defined round to ovoid corticated or non-corticated radiolucency with a diameter of up to 30 mm. It is located anterior to the angle of the mandible and below the inferior alveolar canal. Anatomically, the Stafne's bone cavity is the result of an indentation of the lingual cortical plate of the mandible by an aberrant lobe of the submandibular salivary gland or fat tissue. Depending on the site and

angulation of the projection, the defect may also manifest with an open inferior cortical border as demonstrated by the right image.

Slide 16: A bifid condyle may be uni- or bilateral and manifest with a deep central notch on the superior condylar surface. The patient may experience pain or a clicking sound with jaw movement.

Slide 17: Developmental and dysplastic conditions are those which enlarge and stabilize when reaching maturity. Developmental conditions affect the jaw bones during the developmental stages and generally stabilize (or even regress) when maturity is reached. Dysplasia means abnormal development (*dys* equals abnormal, and *plasia* refers to development) and dysplastic lesions may enlarge but maturation introduces a cessation of growth and the lesions then generally stabilize in its mature form. This is different from neoplastic lesions which enlarge indefinitely and may achieve large dimensions.

Slide 18: Mandibular tori are a form of bony outgrowths that affect the lingual plate of the mandible. Most are bilateral and in the premolar and molar regions. Removal is frequently indicated as mandibular tori may interfere with a mandibular denture.

Slide 19: The axial view shows an asymmetrical distribution of mandibular tori.

Slide 20: On intraoral radiographs the ill-defined radiopaque shadows of mandibular tori may superimpose over the roots of the premolar teeth.

Slide 21: This cropped panoramic radiograph illustrates the characteristic bilateral symmetric distribution of the radiopaque mandibular tori in the premolar regions.

Slide 22: A maxillary torus, referred to as torus palatinus, is a single bony outgrowth in the middle third of the midline of the hard palate. It is more common in females and may be flat, oval, nodular or lobular. It is bony hard and may interfere with denture wearing. A maxillary pleomorphic adenoma of minor salivary gland origin may have a similar appearance except that it is usually eccentric and not bony hard.

Slide 23: Intraoral images reveal the radiopaque shadow of a maxillary torus.

Slide 24: The term exostosis is used for bony outgrowths which are not located in specific areas mentioned for the torus palatinus and the mandibular tori. Exostosis are exophytic periosteal outgrowths which manifest clinically as a single or multiple asymmetric protrusions on the surface of alveolar bone. These lesions are distinguished from periosteal osteomas (which are benign tumors) by their lack of potential to enlarge. Although the etiology is mostly idiopathic, their development may be related to mechanical forces as they frequently occur in the vicinity of muscle attachments. Exostoses remain stable in size. The image on the right shows a rare mushroom-shaped exostosis in the right maxillary sinus. Failure of enlargement on follow-up images excluded an osteoma.

Slide 25: Osteopetrosis (marble bone disease) is a rare inherited bone disorder characterized by unopposed bone deposition. Bone resorption is inhibited with the result of displacement of bone marrow by abnormal bone. Due to the cranial nerves being compressed, blindness, deafness and facial paralysis may be associated with the condition. Bone pain and osteomyelitis, bone fractures and bone infections may also be a result of osteopetrosis. The axial view shows calvarial involvement with increased width and density of the skull bones and loss of the normal trabecular pattern. Note the partial obliteration of the frontal sinus.

Slide 26: Cherubism is a genetic disease which is prevalent in certain families. The manifestations occur in early childhood as progressive painless enlargement of the posterior section of one or both jaws. The enlargement of the maxilla pulls down the lower eyelid, and exposure of the sclera gives the affected children the appearance of staring up to heaven, reminiscent of the medieval cherubs.

Slide 27: Cherubism involves more the mandible than the maxilla. Bilateral expansion of the cortical plates is associated with multilocularity, unerupted teeth and teeth displaced in an anterior direction. The lesions consist microscopically of giant cells. No surgical intervention is necessary and the lesions regress during late childhood.

Slide 28: Hyperostosis manifests as retained and even enlarged alveolar bone in areas where teeth had been removed or in the retromolar area of edentulous jaws. After tooth extraction the usual general trend is loss of alveolar bone. The reason for the retention and increase of the bulk of alveolar bone when mechanical stimulation of functional teeth is removed, is speculative.

Slide 29: This is an example of unilateral hyperostosis. Note the osteoma in the right maxilla indicated by the circle.

Slide 30: The stylohyhoid ligament may ossify with advancing age manifesting as elongation of the styloid process. An elongated styloid process is defined as being longer than 30 mm. It may be seen unilaterally or bilaterally and with or without joint-like junctions (pseudo-articulations) as demonstrated with an arrow in the right image. No treatment is required in asymmetric cases. However, when interference with rotation of the head, pain with swallowing or yawning or throat pain occurs, it is referred to as Eagle's syndrome that requires surgical correction.

Slide 31: This is an example of bilateral elongation of the styloid processes.

Slide 32: Fibrous dysplasia generally develops during childhood and ceases to enlarge when adulthood is reached. The condition is divided in subtypes according to the number of bones involved. Monostotic fibrous dysplasia affects a single bone and polyostotic fibrous dysplasia more than one bone. Craniofacial fibrous dysplasia is reserved for a single lesion in the craniofacial region which extends across more than one contiguous craniofacial bone. Polyostotic fibrous dysplasia is further subdivided in two types: The McCune-Albright syndrome with bone lesions and pigmented skin lesions described as café au lait spots as well as endocrine hyper function. The Jaffe-Lichtenstein type of polyostotic fibrous dysplasia manifests without endocrinal changes.

Slide 33: The endocrine hyperfunction in the McCune Albright syndrome may manifest with precocious puberty, hyperthyroidism, hyperparathyroidism, acromegaly or Cushing syndrome.

Slide 34: The genetic defect in fibrous dysplasia is a mutated GNAS gene. The earlier during embryogenesis the mutation occurs, the more extensive the manifestation of the condition. A late mutation will result in the monostotic type.

Slide 35: The café au lait spots in the McCune Albright syndrome are easier to identify on lighter pigmented skin and show an irregular outline.

Slide 36: Microscopically fibrous dysplasia consists of fibrous connective tissue with woven bone trabecula. Fibrous dysplasia bone merges with the premorbid bone and there is subsequently no capsule which separates the lesion from the surrounding bone, an important distinction with ossifying fibroma. Because of the overlap in microscopic appearances, fibrous dysplasia is often classified with ossifying fibroma as a fibro-osseous lesion.

Slide 37: The common characteristics of all forms of fibrous dysplasia are their early onset in life, gradual painless expansion, poor circumscription and stabilization of growth with adulthood when skeletal growth ceases.

Slide 38: One of the earliest clinical signs of fibrous dysplasia that frequently involves the maxilla, is obliteration of the canine fossa on the buccal surface of the maxillary cortical bone.

Slide 39: The cropped panoramic radiograph shows monostotic fibrous dysplasia affecting the corpus of the mandible. Note the typical oval expansion, poor demarcation with blending into the surrounding bone and the characteristic ground glass-like internal structure.

Slide 40: On a periapical radiograph the lesion often shows an orange peel appearance. Note the blending of the lesion with the surrounding bone.

Slide 41: The clinical image shows an example of a craniofacial type, which is a single lesion but which involves more than one contiguous craniofacial bone, in this case the maxilla and zygoma. The lesion is painless, bone-hard, expands slowly and the main complaint is facial asymmetry. As soon as the multifocal lesions are separated by normal bone, the diagnosis changes to polyostotic fibrous dysplasia.

Slide 42: Craniofacial fibrous dysplasia is a unifocal (single) lesion which involves contiguous bones. Note the ground glass appearance and blending with the surrounding bone.

Slide 43: Involvement of the calvaria often manifests with a copper-beaten appearance.

Slide 44: Polyostotic fibrous dysplasia shows more than one lesion. Note the typical ground glass appearance and blending with the surrounding bone.

Slide 45: Lesions frequently confused with fibrous dysplasia are ossifying fibroma, osteosarcoma and proliferative periostitis. Although the internal structure of ossifying fibromas may resemble

fibrous dysplasia, these benign tumors enlarge concentrically and are always well demarcated. Osteosarcoma is poorly demarcated but presents with a destructive and infiltrative growth. Proliferative periostitis also occurs in children, is however associated with a source of inflammation and manifests radiologically with periosteal bone formation. These lesions will be discussed later.

Slide 46: Cemento-osseous dysplasia is a term describing fibro-osseous lesions which develop in later life and contain bone and psammomatoid mineralized deposits, often referred to as cementum, in a fibrous matrix. Most lesions in this group mature and fail to cause significant expansion. This group is referred to as the conventional types cemento-osseous dysplasias and according to the distribution of the lesions they are subdivided in three subtypes: periapical-, florid- and focal cemento-osseous dysplasia. The rare expansive cemento-osseous dysplasia manifests with gross jaw expansion. This group, which is often erroneously referred to as familial gigantiform cementoma, occurs in familial and non-familial types.

Slide 47: The common features of all cemento-osseous dysplasias are a higher incidence in females, painless unless infected, their fibro-osseous composition which matures, location outside the periodontal ligament space as indicated in the image on the right side, a tendency to form simple bone cyst-like cavities and sequestration when secondarily infected.

Slide 48: The principles in the management of the cemento-osseous dysplasias are the following: 1. The final diagnosis must be established on clinical and radiographic information only 2. A biopsy is contra-indicated as the procedure may introduce an infection into the lesion with devastating consequences. This is due to the dense relative avascular mineralized deposits in the lesion which provide an ideal immune protected environment for bacterial proliferation. 3. Asymptomatic lesions are managed conservatively with only measures to prevent secondary infection such as improved oral hygiene and the prevention of dental infections. 4. Infected symptomatic lesions and expansive cemento-osseous dysplasias must be removed surgically.

Slide 49: Now let us look deeper into the conventional (non-expansive) cemento-osseous dysplasias. The generic characteristics of all three subtypes are: They mainly affect adult females, are generally asymptomatic, are located at the apices of vital teeth, cause no- or minimal expansion and rarely cause tooth displacement or root resorption. They mature centrifugally (from the center outwards) and simple bone cyst-like cavities are possible.

Periapical cemento-osseous dysplasia is located periapical to vital mandibular incisors and simple bone cyst-like cavities are rare.
Focal cemento-osseous dysplasia is a solitary lesion located in the mandibular corpus, is usually less than 2 cm in diameter and simple bone cyst-like cavities are rare.
Florid cemento-osseous dysplasia is characterized by multi-quadrant involvement, affects mainly middle-aged females and simple bone cyst-like cavities are common.

Slide 50: Let us discuss periapical cemento-osseous dysplasia in greater detail. The light blue print on the top of the slide is a repeat of the general characteristics of the conventional cemento-osseous dysplasias which also apply to periapical cemento-osseous dysplasia. The lesions are multiple and restricted to the periapical region of vital mandibular incisor teeth. Simple bone cyst-like cavities are rare.

Slide 51: The three stages of maturation of periapical cemento-osseous dysplasia are shown on this slide. The image on the left shows the early stage which is characterized by radiolucencies that surround the apices of vital asymptomatic teeth. As the lesions enlarge and mature, the intermediate stage presents as mixed radiodensities which often become confluent as demonstrated with the image in the middle. The image on the right shows a mature stage that becomes progressively radiopaque and lobular. Note that the lesions are located outside the periodontal ligament space. This is a radiological feature distinguishing these lesions from periapical inflammatory pathology.

Slide 52: The microscopic appearances of the three stages correlate with the radiological images shown in the previous slide. In the early radiolucent stage fibrous tissue predominates. The mineralized component progressively increases at the expense of the fibrous tissue until the late stage is reached and the composition changes to predominantly dense mineralized tissue as demonstrated in the micrograph on the right side. It is important to note that biopsies should not be taken because of the risk of infection and subsequent sequestration. The lesions must be diagnosed radiologically, and not treated if asymptomatic.

Slide 53: Loss of teeth associated with periapical cemento-osseous dysplasia may lead to migration and sequestration of mature lesions.

Slide 54: This slide shows two mature confluent periapical cemento-osseous dysplasias which are secondarily infected. Infected lesions are painful and radiologically present with transition from smooth lobular- to sharply pointed masses. Infection may induce external root resorption of associated teeth.

Slide 55: The most important differential diagnosis of periapical cemento-osseous dysplasia is periapical inflammatory dental pathology. This is a common mistake made in dental practice and leads to unnecessary endodontic therapy.

Slide 56: The second conventional cemento-osseous dysplasia is the focal type. The light blue text on the top of the slide gives the generic features which apply to all conventional cemento-osseous dysplasias. The focal type is a solitary lesion which presents in the 3rd decade and in the body of the mandible. The maximum size is approximately 1.5 mm and simple bone cyst-like cavities are rare.

Slide 57: Early focal cemento-osseous dysplasia is a slow-growing well defined radiolucency with a corticated/sclerotic border. The intact periodontal ligament space of the associated tooth is a helpful radiological feature as osseous dysplasias are located outside the periodontal ligament space. A radiolucent rim around the intermediate stage, demonstrated in the middle image, shows growth potential. The mature lesion on the right is radiopaque with a narrow radiolucent rim which indicates a stable and mature stage.

Slide 58: In edentulous areas, most focal cemento-osseous dysplasias remain in the same location, however occlusal migration has been recorded.

Slide 59: The differential diagnosis of focal cemento-osseous dysplasia is listed here. The lesions mentioned will be discussed in subsequent sections.

Slide 60: The last of the conventional cemento-osseous dysplasias is the florid type. The light blue text on the top of the slide gives the generic features of florid cemento-osseous dysplasia which apply to all conventional cemento-osseous dysplasias. Florid cemento-osseous dysplasia is characterized by multi-quadrant jaw involvement and a common association with simple bone cyst- like cavities.

Slide 61: The panoramic view shows the typical painless multi-quadrant involvement in the early stage. Note the round or irregular radiolucencies which may coalesce.

Slide 62: The intermediate stage presents with multi-quadrant mixed radiopaque masses with radiolucent borders of varying widths.

Slide 63: The mature stage florid cemento-osseous dysplasia presents with multi-quadrant lobular radiopacities with no- or narrow radiolucent borders which indicate stabilized growth; however, lesions may expand if simple bone cyst-like cavitation occurs.

Slide 64: Secondarily infected lesions of florid cemento-osseous dysplasia are symptomatic, the lobular radiodensities develop pointed margins and the lesions are surrounded by an inflammatory exudate which reflects as a wide radiolucent zone.

Slide 65: When teeth are extracted, the consequence is often a fulminant sequestrating osteomyelitis that requires extensive surgical debridement.

Slide 66: The panoramic image demonstrates the formation of simple bone cyst-like cavities in a patient with florid cemento-osseous dysplasia. The cavities are radiolucent with scalloped margins and may cause root resorption.

Slide 67: The differential diagnosis is listed and will be discussed later.

Slide 68: The expansive group of cemento-osseous dysplasias is incorrectly designated as familial gigantiform cementomas. This group can be subdivided in familial and non-familial subtypes.

Slide 69: In our selection of cases the majority are non-familial and present as single or multiple expansive lesions which ultimately perforate the bone cortices. In the early stage the enlargement is faster than the process of mineralization. When an extreme size is reached, the lesions mineralize and growth stabilizes. This is in contrast with ossifying fibromas which are benign neoplasms of fibro-osseous origin, lack maturation and do not cease to grow. Simple bone cyst-like cavities and root resorption are common in the expansive type.

Slide 70: When the expansive type is small, it may be difficult to differentiate it radiologically from small conventional types. Predicting their growth potential may be difficult. The expansive type generally occurs at a younger age and the extent of the radiolucent zone is an indicator of growth potential as the growth center of the lesion is in the radiolucent area. This case demonstrates a lesion with a relatively large radiolucent (growth) component indicating a potential for future expansion.

Slide 71: The cross section through a surgical specimen of an expansive cemento-osseous dysplasia shows the fibrous tissue component that represents the growth center of the lesion. The more centrally located stable mineralized component resembles a ginger root-like mass.

Slide 72: This coronal slice shows an expansive osseous dysplasia in the corpus of the right mandible. Note the ginger root-like calcification and ground glass-like appearance of the fibrous component of the lesion. A mature conventional cemento-osseous dysplasia is present in the third quadrant.

Slide 73: This large example of an expansive type cemento-osseous dysplasia shows the radiolucent fibrous growth zones and more stable lobular mineralized regions in the tumor. Note the areas of cavitation which indicate simple bone cyst-like cavities.

Slide 74: The lesion most commonly confused with expansive cemento-osseous dysplasia is ossifying fibroma, a benign tumor which will be discussed later in the program.

Slide 75: The next category of bone pathology in this series is unrelated. The lesions share an unknown cause.

Slide 76: Dense bone islands are also known as idiopathic osteosclerosis. They manifest as radiopacities that are not associated with inflammatory lesions and their differential diagnosis

includes chronic focal sclerosing osteomyelitis that has an association with a focus of inflammation, usually of dental origin. A clinical examination with tooth vitality tests is important in distinguishing idiopathic osteosclerosis from chronic focal osteomyelitis.

Slide 77: The characteristic feature of focal idiopathic osteosclerosis is a radiopacity without a radiolucent border. On CBCT images these lesions manifest as outgrowths of cortical bone with radiating thorny projections.

Slide 78: Focal idiopathic osteosclerosis can become large and may even cause root displacement. Root resorption is rare.

Slide 79: Paget's disease of bone affects the spine, weight-bearing bones, skull and jaw bones of the elderly. The maxilla is affected twice as often as the mandible. It is the result of overactivity of osteoblasts and cementoblasts with over production of bone and cementum. Paget's disease is characterized by elevated serum alkaline phosphatase concentration which is a marker for generalized activity of bone cells. It manifests clinically with bowing of weight-bearing bones (occasionally fractures), enlargement of one or both jaws and the calvaria. Neurologic pain is due to sensory nerve compression. Both jaw halves of either the maxilla and/or the mandible are involved. The lesions start as radiolucencies that mature through a ground glass stage into cotton wool appearing radiopacities. Multiple diastemas due to deposition of bone are common. Generalized hypercementosis is a frequent feature.

Slide 80: The reason for activation of osteoclasts which form the basis of a central giant cell granuloma, is unknown. It affects the mandible of young adults, is more common in females and long-standing cases present clinically with expansion. The lesion develops due to activation of osteoclasts (for reasons unknown) and therefore shows multiple osteoclast-like giant cells with microscopic examination. From a microscopic perspective a central giant cell granuloma cannot be distinguished from brown tumors of hyperparathyroidism. Helpful clues in this distinction are the multiplicity of skeletal giant cell lesions in hyperparathyroidism, whereas central giant cell granulomas are always solitary. The definitive distinction is the concentration of parathyroid hormone and calcium in the serum that is elevated in hyperparathyroidism but normal in central giant cell granuloma. The management is based on curettage (most are not aggressive) or resection (for the rare aggressive types). The radiological differential diagnosis includes ameloblastoma, odontogenic myxoma, aneurysmal bone cyst and simple bone cysts due to their multilocular appearances. It must be noted that a peripheral soft tissue giant cell granuloma also exists, which is located in the gingiva and which does not originate in bone. Peripheral giant cell granulomas may cause a cup-shaped indentation in the cortex of the underlying bone. Due to the identical microscopic appearance, radiographs are essential in distinguishing peripheral from central giant cell granulomas.

Slide 81: The cropped panoramic radiograph shows a well-defined radiolucent central giant cell granuloma. It presents with tooth displacement, root resorption, a scalloping border which causes expansion of the inferior cortex and a soap bubble appearance. The internal straight septum originates from the inferior mandibular cortex with an angle, which is characteristic for the lesion.

Slide 82: The axial CBCT slice shows buccal expansion with erosion of the cortical plate which is seen in large central giant cell granulomas. Note the granular internal septum in this lesion.

Slide 83: Aggressive giant cell lesions can grow large and become difficult to distinguish radiologically from an ameloblastoma when displaying a soap bubble or honeycomb appearance. The adjacent molar is displaced and resorbed. Note the corticated border and the characteristic scalloping outline at the inferior border of the mandible.

Slide 84: Simple bone cysts are not true cysts as they lack the epithelial lining. They were previously known as traumatic bone cysts, occur most frequently in the posterior premolar/molar region and infrequently in the anterior area of the mandible. This process of bone cavitation is painless and manifests as a poorly or moderately defined radiolucency with an undulating border. Minimal or no displacement or resorption of roots and also minimal or no bone expansion are present. The upper border of the lesion scallops characteristically between the roots of the teeth. The surgical specimen shows multicystic irregular cavitation which is empty and blood stained.

Slide 85: Simple bone cysts can also involve the anterior mandible and extend into the basal bone.

Slide 86: Metabolic diseases affecting the jaw bones will be covered superficially and students are referred to more detailed text in this regard. We will briefly show images of rickets, hyperparathyroidism and osteoporosis.

Slide 87: Rickets is a disease of childhood resulting from abnormal calcium/phosphorous/ vitamin D metabolism. It leads to growth retardation with bowing of weight bearing bones, prominent osteochondral junctions and frontal bossing. Its adult equivalent is osteomalacia. The generalized regressive bone change is due to a lack of mineralization.

Slide 88: On dental radiographs the mineralized bone appears reduced with thin cortical structures and reduced and thinned trabeculae. The teeth characteristically show large pulpal cavities with thin dentinal walls, and in some cases hypoplasia (hypocalcification) of the enamel.

Slide 89: Another example of rickets that shows reduced trabecular bone, thin cortices and large pulpal cavities with thin dentinal walls.

Slide 90: Although hyperparathyroidism is a complex endocrinal disorder, the bone changes are a result of parathyroid hormone overproduction. This can be either by a tumor or hyperplasia of the parathyroid glands producing parathyroid hormone excessively, or secondary due to a lack of calcium, vitamin D or phosphorus which can be due to kidney failure or malnutrition. The condition occurs more in females than males and mainly between 30 and 60 years. The skeletal changes in all types are based on over-secretion of parathyroid hormone with stimulation of osteoclasts followed by generalized skeletal bone resorption. The panoramic radiograph shows a case of hyperparathyroidism secondary to kidney failure. Note the decreased density of the jaw bones with generalized loss of cortical boundaries such as the inferior border, mandibular canal, borders of the sinuses and lamina dura. Students are referred to the reference textbooks for further information.

Slide 91: In cases where the concentration of parathyroid hormone is persistently high such as in primary hyperparathyroidism, multiple radiolucent skeletal lesions develop that are referred to as brown tumors of hyperparathyroidism which may cause expansion of the bone. Resorption of bone is directly linked to the osteoclast stimulation by the excessive parathyroid hormone concentration. The brown color of the lesions is due to the extravasation of red blood cells with hemosiderin deposits in the specimen. Differential diagnoses of multiple brown tumors are multiple myeloma, Langerhans cell histiocytosis and metastatic disease. A solitary brown tumor resembles radiologically and histologically a central giant cell granuloma and also a small aneurysmal bone cyst. Due to the giant cells in all these lesions, the diagnosis may be challenging.

Slide 92: Senile osteoporosis is particularly prevalent in elderly females. The lack of estrogen, inactivity, dietary lack of vitamin D, calcium and phosphorous change the metabolic dynamics of bone anabolism, with resultant bone loss. Students are referred to the literature for the use of more sophisticated quantitative imaging to determine osteoporosis.

Slide 93: Mandibular osteoporosis is accelerated by the loss of teeth. This results in reduced mechanical stimulation for the maintenance of alveolar bone, subsequent alveolar bone loss and thinned cortical structures. Note the oblique line that remains prominent.

Slide 94: The first degenerative bone disorder involving the mandible is osteoarthritis or wear and tear disease of the articular surface of the condylar head. It affects mainly the elderly and is characterized by pain with joint movement, and sometimes with spasm of the masticatory muscles. Radiological changes include flattening of the anterior-superior aspect of the articular surface of the head of the condyle, osteophyte formation (ossifications), which may extend into the synovial membrane and subchondral sclerosis (increased density) of the condylar head. Erosion of the articular surface of the head of the condyle and subchondral cysts are rare. The main distinction with rheumatoid arthritis, which may have overlapping features, is the presence of an autoimmune antibody (the rheumatoid factor) in the serum of patients with rheumatoid arthritis.

Slide 95: Resorption of the maxillary alveolar bone occurs after the removal of teeth. The loss of maxillary bone in this setting leads to passive enlargement of the maxillary sinus, which scallops into sites where the tooth or teeth are absent. This phenomenon is referred to as pneumatization of the maxillary sinus. Pneumatization of the maxillary tuberosity is rarely seen and of no clinical relevance.

Slide 96: Another example of bilateral pneumatization of the maxillary sinus due to tooth loss. The lack of bone in the posterior maxillary quadrants complicates the placing of implants. In this case, the mandibular alveolar process retained normal height after loss of teeth. The reason for this change is speculative, but may be related to the mechanical stimulation of the alveolar bone due to edentulous chewing. Note the flattening of the right condylar head.

Slide 97: The next section deals with infections of the jaw bones. Several definitions are important in mastering the concepts involved. Periostitis is inflammation of the periosteum, osteitis is inflammation of dense cortical bone and osteomyelitis is inflammation of bone and bone marrow.

Slide 98: The diagram represents all the pathologies that converge in osteomyelitis. The most prevalent direct causes are indicated with the bold arrows. These are pulpal infections and open fractures or penetrating wounds. A significantly less common cause is septicemia, where circulating bacteria lodge and proliferate in a specific site in the jaw bone.

Slide 99: The line diagrams depicted in this slide illustrate the difference in the pathogenesis of osteomyelitis of the mandible compared with osteomyelitis of the maxilla. In the mandible the thick and non-porous bony cortex contributes to the high pressure that results from the inflammatory exudate in the marrow space. This pressure reduces the inflow of oxygenized blood which perpetuates bone necrosis. Mandibular bone sequestration is therefore more extensive than in the maxilla where the bone cortex is porous, which releases the pressure of the inflammatory exudate. Blood supply of the maxilla is also through more than one artery and the healing capacity in the maxilla is therefore better than in the mandible.

Slide 100: The most common precursor pathology of osteomyelitis is dental infection with pulp non-vitality. The following slides are a superficial revision of the lesions that may develop around the apex of a non-vital tooth and that may give rise to an osteomyelitis. A periapical abscess manifests clinically with a slightly extruded tooth (due to the inflammatory exudate in the apical periodontal ligament that displaces the tooth in a coronal direction) and intense pain on percussion. The inflammatory infiltrate is acute, consisting mainly of neutrophils. Radiological changes only become evident after a few days and manifest initially as a widened periodontal ligament space. With breakdown of the periapical lamina dura a poorly defined radiolucency presents at the apex as demonstrated in the images. The two images on the left illustrate advanced periapical abscesses

that are poorly defined and radiolucent. The image on the right shows a periapical abscess associated with a primary molar and located coronal to the developing crown of the second permanent premolar. This infection may lead to enamel hypoplasia of the developing permanent successor, a condition referred to as Turner's tooth.

Slide 101: Widening of the periodontal ligament space with the development of a poorly circumscribed radiolucency is characteristic for a periapical abscess. The lesion is accompanied by pain, which is severe upon percussion of the tooth. The micrograph demonstrates the origin of the abscess in the periodontal ligament space, usually in relation to the apical foramen of the tooth. The abscess can also develop in relation to the opening of a lateral accessory pulpal opening, in which case the lesion will be situated on the lateral root surface.

Slide 102: Untreated periapical abscesses become chronic with chronic inflammatory cells (lymphocytes and plasma cells) which become the dominant cell types in the infiltrate. The dormant epithelial remnants of Malassez, which are the remnants of the sheath of Hertwig, proliferate and appear as small solid nests of epithelial cells in the chronic inflammatory infiltrate. The epithelial proliferation ultimately gives rise to the development of a radicular cyst, which is a cavity lined by odontogenic epithelium.

Slide 103: A periapical granuloma manifests on a radiographic image as a well-defined radiolucency, usually smaller than 12mm in diameter and often surrounded by dense bone (chronic focal sclerosing osteomyelitis) indicating a chronic process. Note the association with a non-vital tooth, which is a requirement for the diagnosis of a periapical granuloma. The acute pain on percussion subsides and is replaced by a low-grade dull pain sensation.

Slide 104: In the maxilla, a periapical granuloma or cyst associated with a single tooth may elevate the bony wall of the sinus and the sinus lining, resulting in a halo-appearance. This lesion is distinguished from an antral pseudo cyst, which is a dome-shaped elevation of the mucosal lining of the sinus above the bony floor of the sinus. The radiograph shows the non-vital tooth associated with the granuloma or cyst (microscopic confirmation is necessary to distinguish between granuloma and cyst).

Slide 105: Although radicular cysts may be small, most are more than 10 mm in diameter. The cysts are well defined, radiolucent, usually corticated and always associated with a non-vital tooth. Inflamed radicular cysts show a lack of the peripheral cortex. Students are referred to more detailed literature on the radiological presentations of periapical abscess, -granuloma and radicular cysts, which are interrelated conditions that may predispose to exacerbation and progression to osteomyelitis. Slides 100 – 105 serve as a brief reminder of the periapical inflammatory lesions that may progress into an osteomyelitis. Other causes include open trauma, penetrating wounds,

open fractures and the rare possibility of infective agents lodging in the jaw bones during a septicemia.

Slide 106: There are several classification systems of osteomyelitis. The following system divides osteomyelitis in acute and chronic types, depending on the duration of the infection. In general, acute osteomyelitis persists for the first 3 weeks after onset of symptoms. It is this phase that is painful due to the inflammatory exudate that causes a generation of pressure, which has consequences particularly in the mandible (explained earlier, slide 99). The acute phase merges into a chronic phase (through an intermediate phase often referred to as sub-acute osteomyelitis). In sub-acute osteomyelitis the inflammatory infiltrate is characterized by the appearance of chronic inflammatory cells and the initiation of reactive bone formation in the surrounding vital bone. Fully developed chronic osteomyelitis is earmarked by a chronic inflammatory infiltrate with reactive bone formation, manifesting on radiographs as radiopacities. The formation of bone is the result of the release of cytokines, such as PG E2 (prostaglandin E2) by the inflammatory cells, which are potent inducers of bone forming activity. Osteomyelitis of infants and children are placed in a separate category due to their unique impact on development and jaw growth.

Slide 107: Untreated middle ear infections in infants and young children may perforate into the TMJ space and damage or even destruct the mandibular growth center, which is located in the neck of the condyle. This leads to a unilateral lack of mandibular growth which will manifest as a shift of the mandible to the affected side and present with unilateral mandibular hypoplasia and facial asymmetry.

Slide 108: A sequestrum is a necrotic bone fragment which can be removed from the site of infection in a suppurative osteomyelitis. Sequestra in the mandible are more common and usually more extensive for reasons previously explained. A sequestrum is present in pus which has a white creamy color and consists of necrotic neutrophils and bacterial colonies. Radiologically a sequestrum manifests as a radiopacity surrounded by a poorly defined radiolucent zone (the pus). The pus may drain through a fistula which discharges on the mucosal- or skin surface.

Slide 109: If the infection responsible for the osteomyelitis spreads to the jaw bone during a septicemia, the bone my present with a diffuse mixed radiopaque-radiolucent appearance, mimicking metastatic disease (see later).

Slide 110: Localized acute alveolar osteomyelitis occurs post tooth extraction. It usually affects the mandibular molar region and is characterized by a history of previous extraction with a painful and foul-smelling non-healing lesion at the site of the extraction.

Slide 111: The characteristic radiological appearance is loss of the lamina dura and the interradicular bone at the extraction site with a poorly demarcated radiolucent outline following

the anatomical location of the tooth socket. A root remnant or a dislodged bone fragment are often present in the extraction socket in localized alveolar osteomyelitis.

Slide 112: The consequence of chronic inflammation of bone is the deposit of dense bone, often referred to as sclerotic bone. Although most cases of acute osteomyelitis may develop in chronic osteomyelitis, the latter may also develop *de novo*. This occurs particularly in patients with periapical granulomas or infections with a low virulence.

Slide 113: Radiologically a moderately or ill-defined focal area of bone sclerosis is seen in the apical area of a non-vital tooth in chronic focal sclerosing osteomyelitis. If the tooth is extracted, the radiopacity remains and is designated a bone scar. Over time the scar may be remodeled into normal bone but mostly persists.

Slide 114: Extraction socket sclerosis manifests as a painless radiopacity following the outlines of the tooth socket at a previous extraction site.

Slide 115: The differential diagnosis of chronic focal osteomyelitis includes idiopathic osteosclerosis (will be discussed later), odontoma (see chapter on odontogenic tumors), a root remnant, focal cemento-osseous dysplasia (discussed previously) and cementoblastoma (see chapter on odontogenic tumors).

Slide 116: Chronic diffuse sclerosing osteomyelitis is often associated with extensive periodontal disease. It shows a mixture of diffuse radiopacities and radiolucencies, often designated as a cotton wool appearance. Note the ramus is not involved, which excludes the mature stage of Paget's disease.

Slide 117: The differential diagnosis of chronic diffuse sclerosing osteomyelitis includes Paget's disease of bone, florid cemento-osseous dysplasia (discussed previously) and Gardner's syndrome (discussed later).

Slide 118: The etiologies of chronic osteomyelitis with proliferative periostitis (or Garre's osteitis as it is often referred to) are periodontal- or periapical infections in children, as well as pericoronitis. The patient typically manifests with a bony hard swelling in the area of the infection that may lead to facial asymmetry. Radiologically periosteal bone formation may manifest with subsequent layers of bone on the cortical surface of the mandibular bone plate in the zone of infection, resembling an onion skin appearance. These subsequent layers of bone are deposited by the reactive process of bone formation, reminiscent of the year-rings of a tree. The lesion regresses after treatment of the infection, and surgical correction is not indicated.

Slide 119: The CBCT slices demonstrate the radiological features of Garre's osteitis.

Slide 120: The main differential diagnosis is fibrous dysplasia (discussed previously).

Slide 121: The etiology of medication related osteonecrosis of the jaw (MRONJ) is long term bisphosphonate therapy, which is a group of anti-bone resorbing drugs used in the treatment of osteoporosis and bone catabolism due to malignant disease. The diagnosis depends on a thorough drug history. Clinically, the bone penetrates through the mucosa (see left side). Bisphosphonates suppress bone defense by neutralizing osteoclasts. Foci of infection often flare-up. Radiologically, the features are those of chronic osteomyelitis with ill-defined radiolucencies, destruction of cortices, diffuse sclerosis and sequestration of alveolar bone often with teeth included and no healing of extraction sites. MRONJ can be prevented by maintaining oral hygiene and treating jaw bone infections and carious teeth before commencing with bisphosphonate therapy. When an infection occurs during the course of therapy, a drug vacation with debridement of the infected site is recommended.

Slide 122: The etiology of osteoradionecrosis is high doses external beam ionizing radiation as used in radiation therapy of oral cancer, particularly with dosages of 60 Gray and more. The mandible is affected twenty-four times more frequent than the maxilla and the patient presents with pain, fistula formation and failure of healing of extraction sites. Radiologically the patient presents with an acute suppurating osteomyelitis with extensive jaw involvement and frequently a pathological fracture, but no periosteal bone reaction. Osteoradionecrosis is resilient to treatment and prevention is therefore important. All foci of jaw bone infection must be cleared before radiation therapy. In the post radiated phase debridement, antibiotics and hyperbaric oxygen therapy may be necessary. Note that osteoradionecrosis can be triggered by tooth extraction, trauma or infection years after radiotherapy.

Slide 123: The next section will deal with bone tumors occurring in the maxillofacial region. The list is not complete and students are referred to the prescribed textbook to cover the whole field. As is customary the case, the tumors are divided in benign and malignant categories. Benign tumors are generally well circumscribed, slow growing and manifest with displacement of surrounding structures, whereas malignant tumors are faster growing, infiltrate and destruct surrounding structures. Malignant tumors may be primary (develop in the jaw bones) or secondary (develop at distant sites), and spread to the jaw bones. Students are referred to prescribed textbooks for information on the long list of benign and malignant tumors others than those discussed.

Slide 124: One of the more common unique benign primary jawbone tumors is ossifying fibroma NOS (not otherwise specified) also referred to as the adult type. Ossifying fibroma (OF) is often classified with fibrous dysplasia as a fibro-osseous lesion due to its composition of fibrous tissue and bone, which makes microscopic distinction difficult. Pathologists therefore rely on clinical and radiological features to establish the diagnosis. The tumor usually occurs in the third or fourth

decade of adult life as a single lesion. Unlike the juvenile type, it affects only the jaw bones, mainly the mandible, and is more common in females than males. Clinically OF is characterized by an expansive and slow growing painless tumor. Large tumors may perforate the cortical bone. The main feature which distinguishes the adult type OF from expansive cemento-osseous dysplasia is the lobular radiopaque masses in cemento-osseous dysplasia. The adult type OF shows most commonly concentric enlargement with expansion of the cortical plates of the jaw bones. This distinguishes it from fibrous dysplasia that occurs in children and presents with a more diffuse enlargement (see previous discussion).

Slide 125: The clinical photo shows an OF in the maxilla causing facial asymmetry.

Slide 126: OF's have a wide range of radiological manifestations. These two cropped panoramic radiographs show the typical features which include well demarcated and usually corticated borders, concentric growth, radiolucent- or ground glass internal structure and tooth resorption associated with larger tumors. The internal structure is the result of differing volumes of mineralized bone formed within the lesion. Radiolucent examples show no or small bone volumes and more bone containing tumors show a mixed or more radiopaque density.

Slide 127: Tumors with a multilocular appearance may overlap with the features of an ameloblastoma. However, internal radiopacities make a differential diagnosis of an ameloblastic fibro-odontoma more likely. Note the cortical thinning with expansion of the inferior border of the mandible.

Slide 128: This adult type OF shows a concentric non-maturing growth with bowing of the inferior cortex, good demarcation and an orange peel appearance. The expansive type cemento-osseous dysplasia differs from this neoplasm in that it forms mature lobulated mineralized deposits which ultimately result in confluency and minimal radiolucent zones. The adult type OF shells out from its capsule which is in contrast to fibrous dysplasia that blends with the surrounding normal bone. Recurrences of OF's are rarely reported and the tumor has no malignant counterpart.

Slide 129: The juvenile type ossifying fibroma is subdivided in the trabecular and psammomatoid varieties. The trabecular variety occurs mainly in the jaw bones, whereas the psammomatoid variety may occur extra-gnathial with examples as high as close to the base of the skull.

Slide 130: The trabecular variety juvenile OF occurs in children younger than 12 years, without race or gender predilection. The tumors are single and affect mainly the tooth bearing regions of the maxilla. They are characterized by a rapid and destructive concentric expansive growth, which earned them the title of *aggressive* juvenile ossifying fibromas in the past. They have a tendency to recur if incompletely removed. Secondary aneurysmal bone cyst formation is rare.

Slide 131: Two examples of the trabecular type juvenile OF are shown in this slide. The skin over the tumors remains freely moveable, which distinguishes it from malignant disease where the skin is infiltrated and fixed.

Slide 132: Radiologically the juvenile trabecular OF presents as a well-defined mixed density with delicate trabeculation, tooth displacement and root resorption.

Slide 133: Smaller trabecular juvenile OF's may present with ground glass appearance, root resorption, tooth displacement and an expanded thin cortex.

Slide 134: The microscopic features are characteristic with notable microscopic heterogeneity. Microscopic fields with the typical fibro-osseous features alternate with areas exhibiting osteoclast activity with bone resorption and a transition in zones which are fibrous.

Slide 135: Psammomatoid juvenile ossifying fibroma is usually seen between 16 and 20 years and presents as a single lesion that is located extra-gnathial. The psammomatoid type OF is just as destructive as the trabecular type. Large tumors perforate the cortical plates and have a high tendency to recur after surgical removal.

Slide 136: This slide demonstrates a psammomatoid type in the sinonasal passage with extension into the orbit and proptosis.

Slide 137: Involvement of the base of the skull may cause bone erosion with perforation into the cranial fossa. Note the mixed density of the tumor.

Slide 138: Microscopically the psammomatoid type is characterized by round mineralized deposits resembling psammomatoid spheres in a fibrous stroma.

Slide 139: The differential diagnoses of OF include the expansive type cemento-osseous dysplasia, ameloblastoma, mixed odontogenic tumors, fibrous dysplasia and odontogenic cysts with calcifications. Students are referred to relevant textbooks for further information.

Slide 140: An osteoma is most frequently a solitary lesion (when multiple, one should consider Gardner's syndrome). It is common in the mandible and paranasal sinuses (especially frontal sinus), and generally asymptomatic. According to the anatomical distribution 2 categories have been recorded: 1. On the bone surface (periosteal or peripheral type), 2. Within medullary bone (central or endosteal type). There are two main types, the compact type that consists of compact bone only, and the cancellous type that consists of bone trabecula. There are also variations in between that may even mimic a ground grass appearance. When small, osteomas are difficult to distinguish from condensing osteitis, chronic focal sclerosing osteomyelitis or idiopathic

osteosclerosis. Larger osteomas may show a sclerotic margin with central trabeculation (cancellous type) that may opacify (compact type). Treatment is generally not required unless they become large or interfere with function.

Slide 141: Central compact osteomas presenting in the third and fourth quadrants as well circumscribed radiopaque masses resembling a ground-glass appearance.

Slide 142: The PA view shows an osteoma in the frontal sinus and the coronal section one in the left maxillary sinus. Note the sialolith in the mouth floor.

Slide 143: The coronal and sagittal view show a peripheral compact osteoma at the right posterior mandible.

Slide 144: The differential diagnosis of an osteoma includes focal cemento-osseous dysplasia, torus, sialolith (superimposed), chronic focal sclerosing osteomyelitis, idiopathic osteosclerosis, osteoid osteoma, osteoblastoma, odontoma and ossifying fibroma. Students are referred to the relevant sections where these lesions are discussed.

Slide 145: Gardner's syndrome is an autosomal dominant familial disorder with multiple osteomas, dense bone islands, frequently unerupted permanent and supernumerary teeth in both jaws, odontomas and epidermoid cysts. The syndrome is associated with multiple adenomatous polyps in the intestine that have a high incidence of malignant change. Patients must be referred to a gastroenterologist for management.

Slide 146: The differential diagnosis of Gardner's syndrome includes florid cemento-osseous dysplasia and chronic diffuse sclerosing osteomyelitis.

Slide 147: Capillary and cavernous vascular malformations consist of thin-walled blood vessels which can be small (capillary type), large (cavernous type) or a mixture of the two types. It is a low-pressure lesion that is common in the facial bones and vertebra and presents radiologically with mild expansion and a honeycomb radiolucency. The coronal scan shows an example affecting the left maxillary sinus and nasal cavity. Note also the bulging of the orbital floor as well as erosion of the lateral maxillary wall. The micrograph shows the dilated vascular spaces filled with red blood cells between bony trabeculae that give the lesions a honeycomb appearance.

Slide 148: Arteriovenous malformation develops when high pressure arterial blood shunts directly into thin-walled blood vessels. These lesions bleed profusely when biopsied.

Slide 149: The blow-out ballooning of the blood vessels presents radiologically as an expansive radiolucency with thinning and erosion of the cortex. An arteriogram can aid in the diagnosis.

Slide 150: The etiology of an aneurysmal bone cyst (ABC) is unknown. It presents in young patients, and has a predilection for the posterior mandible. The radiological features show an expansive radiolucent lesion, also referred to as “ballooning” of the jaw, with perforation of the cortex and an internal honeycomb appearance. An ABC can be primary or develop secondary in an ossifying fibroma. It consists of blood-filled spaces with giant cells, and the management is excision.

Slide 151: The osteoid osteoma and osteoblastoma are rare benign tumors involving the body of the mandible and commonly present with pain. Both tumors occur in young patients. An intact periodontal ligament space can be followed around the roots of adjacent teeth. The osteoblastoma grows larger than 2 cm in diameter and causes expansion and thinning of the cortical bone plates. The osteoid osteoma is usually not larger than 1 cm in diameter. Both tumors are well-defined and the internal morphology of both is initially radiolucent with progressive radiopacity. The osteoid osteoma shows a wide sclerotic rim around the lesion.

Slide 152: The desmoplastic fibroma is the bone equivalent of soft tissue fibromatosis. The lesion is characterized by connective tissue proliferation with an infiltrative margin. The diagnosis is microscopic. Desmoplastic fibromas occur mainly in the mandible and presents radiologically with a well- to moderately defined radiolucency with an irregular margin and cortical perforation.

Slide 153: A panoramic view of bilateral neurofibromas in a patient with neurofibromatosis. Note the well-defined fusiform widening of the inferior alveolar canals.

Slide 154: Malignant bone tumors can be divided into those that originate in bone (primary malignancies) and those that influence the bone secondary from an external site. The primary malignancy that we will concentrate on is osteosarcoma, which is also the most common. Readers are referred to the textbook for information on other tumor types and particularly the malignant odontogenic tumors, which fall outside the scope of this section. A simple prognostic sub classification of osteosarcoma divides them in two groups: 1.The conventional osteosarcomas are high-grade malignancies which develop from the endosteum and periosteum, whereas low-grade types may also have endosteal origin, but can also originate in the parosteal soft tissue outside the jaw bone. For the purpose of simplification, we will divide secondary malignancies involving the jaw bones in three subtypes: Direct invasion, metastatic disease and disseminated disease.

Slide 155: An early conventional osteosarcoma usually presents with dental pain associated with an extruded and mobile tooth in a traumatic occlusal relationship. These malignancies are common in the posterior quadrants of both jaws, and present radiologically with widened periodontal ligament spaces (due to the invasion of the periodontal membrane by malignant cells). External root resorption may present with narrowing of the roots, which is referred to as spiking resorption. A poorly defined radiolucent area with breakdown of the lamina dura around one or more teeth as well as paresthesia are early signs of an osteosarcoma. Widening of a neurovascular canal with

loss of its cortical margin may also be seen due to infiltration along the path of least resistance (IAC).

Slide 156: More advanced cases present with poorly defined destructive growths that invade the soft tissue. The volume of bone produced in the malignancy results in variable internal radiopacities with obliteration of the normal morphology of bone.

Slide 157: The image of the surgical specimen of a maxilla shows a moth-eaten mixed radiopaque-radiolucent osteosarcoma with poor circumscription and loss of anatomical structures. Although rare, infiltration of the periosteum may produce the classical sunray or sunburst appearance described for osteosarcomas in other skeletal regions.

Slide 158: Periosteal osteosarcomas originate from the periosteum and infiltrate the soft tissue. They present on occlusal images with a sunburst appearance. This shows perpendicular trabeculae on the surface of the bone that often presents with a scalloped outline. These malignancies are high-grade in biological behavior. Radiodensity depends on the volume of bone formed by the tumor, which is superimposed on the jaw bone. An axial view (CBCT,CT) is necessary to exclude central origin of the tumor.

Slide 159: The rare parosteal osteosarcoma is located in the soft tissue adjacent to the jaw bone and does not involve the bone. These lesions are usually of a lower grade.

Slide 160: Low-grade osteosarcomas have a better prognosis than the conventional types, and usually manifest as a moderately circumscribed radiolucency. Paresthesia is key to the radiological diagnosis. (Benign jaw bone diseases, e.g., desmoplastic fibroma, manifest with intact nerve sensation). This case demonstrates a mandibular example with moderate circumscription and foci of cortical perforation.

Slide 161: A panoramic image of a low-grade myofibrosarcoma in a 19-year-old male. Note tooth displacement, external root resorption and the moderately- to poorly defined radiolucency.

Slide 162: the most common malignancies involving jawbones are mucosal carcinomas, which secondarily invade bone. This slide shows a cross section of the mandible with a carcinoma of the floor of the mouth (black arrows), and invasion of the alveolar bone (indicated with a blue line). Note the position of the inferior alveolar nerve (red arrow). The reader is advised to consult the prescribed textbook on the etiology, clinical appearance and management of mucosal malignancies.

Slide 163: Invasion of the mandible by an external squamous cell carcinoma presents with a cup-shaped irregular loss of alveolar bone and teeth without bone support, referred to as floating teeth.

Slide 164: A carcinoma of the maxillary antrum manifests late with perforation of the antral cortices and infiltration of the soft tissue. Radiologically it presents as a poorly defined radiolucent destructive process, loss of cortical borders and alveolar bone.

Slide 165: Due to the content of hemopoietic bone marrow in the corpus and ramus of the mandible, these sites are common receptor sites for metastatic disease. Lungs, breast and prostate are the most frequent primary sites. The image shows the typical radiolucent irregular moth-eaten appearance of a metastatic deposit of breast carcinoma.

Slide 166: Metastatic deposits may also show bone formation, specifically metastatic prostate carcinoma where the neoplastic deposits stimulate osteoblastic activity. The osteosclerotic metastasis presents with a moth-eaten mixed radiodensity.

Slice 167: Multiple myeloma is an example of a bone marrow malignancy which disseminates through all skeletal bone marrow sites. It is characterized by the proliferation of plasma cells which produce a single immunoglobulin type (monoclonal immunoglobulin). The plasma cells release cytokines, which stimulate bone resorption resulting in multiple round well-defined but un-corticated radiolucencies, referred to as a punched-out appearance. It is a disease of the elderly and displacement of the bone marrow leads to pancytopenia with all its consequences. Plasmacytoma refers to a solitary, well-defined radiolucency consisting of plasma cells. No monoclonal protein can be demonstrated in circulation, and the treatment is complete excision. The rate of transformation of a plasmacytoma into multiple myeloma is speculative.

Slide 168: Maxillary involvement by a large B-cell lymphoma in a HIV positive patient showing a poorly defined radiolucency and floating tooth roots. A second lesion was present in the spine.

Slide 169: A 3D reconstruction of a Langerhans cell histiocytosis (Hand-Schüller-Christian disease) in the left temporal bone of a 6-year-old child. Note the multifocal lesions and pattern of destruction with cortical perforation.

Slide 170: The patient is a 5-year-old female with acute leukemia. Note the following features in the right posterior jaw: Widening of tooth follicles of permanent teeth 43, 44 and 47, the tooth follicle of 47 with an under developed crown is displaced prematurely into the oral cavity, multifocal areas of ill-defined bone destruction below 84 and 85, resorption of the 45 and roots of 46.

Slide 171: Conclusion: Thank you for your attention. Our contact email address is provided at the beginning of the lecture. You are advised to consult the prescribed textbooks for more information as the goal of this presentation is to stimulate further reading on the topics.