Bone lesions

We talked the previous lecture about bone lesions including fibrous dysplasia (bone replaced by fibrous tissue) and today we will continue talking about other lesions.

***Cherubism***

Its name is taken from “cherub” which means angel looking

It is mostly a hereditary bone disorder transmitted as autosomal dominant (AD) and there are some sporadic cases caused by specific mutations in the gene SH3BP2**.**

Clinically:

It affects mainly the mandible, posterior side, symmetrical bilaterally increase in the size of the face. (the patient looks like moon face)

Age: very young children in the first ten years of birth and more specifically between 2-4 years.

Painless enlargement in the body and ramus of the mandible, also happening in the posterior maxilla "maxillary tuberosity" causing elevation in the orbital floor and a rim of sclera will be apparent giving the appearance of angel.

Fullness of the face "chubby face", upturned eyes with a visible rim of sclera "that’s why it is called Cherubism"

-There are some parts of the mandible always spared and not affected

-It affects **only** the jaws, it doesn’t affect other bones in the body

Presentation: at a very young age, between the ages of 2-4 years, as a bony **hard** swelling in the mandible or mandible and maxilla together, slowly painless progressive bony enlargement until the age of puberty then becomes stable for a while then starts to regress and to reduce in size and disappear at the age of 30’s.

Some patients could go for cosmetic surgery before reaching their 30’s if they are concerned in esthetics.

**Clinical findings**

Teeth:

Premature loss of primary teeth and displacement of developing teeth

Ectopic eruption and multiple impactions

Permanent teeth might be missing, malformed or hypoplastic

Significant malocclusion

Radiographic appearance:

The pathogomic sign for Cherubism is multilocular radiolucency with **expansion** on the body and ascending ramus bilaterally and maxilla might be affected as we said.

After regression of the disease: *Calcifications* starts and transformation happens from Multilocular radiolucency to mixed lesion to ground glass appearance which resembles fibrous dysplasia.

So **young age, family history and multilocular bilaterally symmetrical radiolucency** will help in the diagnosis

Histopathology:

Taking biopsy can help in the diagnosis

Sometimes we need to take a biopsy; because in the early phases of the lesion it will start unilaterally as multilocular radiolucency on the body and ramus, and there are many diseases present with this appearance. So in order to confirm diagnosis we refer to histopathology….

Under the microscope, you can see **multinucleate giant cell lesion**

Differential diagnosis that looks like Cherubism under the microscope

1- Peripheral giant cell granuloma "giant cell epulis"

2- Aneurismal bone cyst (ABC)

(Multiple giant cell lesions resembling osteoclasts in a fibro vascular connective tissue with a little bit of bone formation).

We can see this histopathology (giant cell lesions) in other lesions like

1-hyperparathyroidism

2-central giant cell granuloma

3- Giant cell tumor

The difference between these giant cell lesions and cherubism is the **eosinophilic vascular cuffing** in Cherubism around the small blood vessels and capillaries, which consists of eosinophilic collagen deposition.

***Inflammatory diseases of bone***

**Osteitis, osteomyelitis, periostitis:**

Periostitis: inflammation affecting the periostium not reaching bone

Osteomyelitis: extensive inflammation mainly in the bone marrow spaces and may extend to the periosteum "most severe form"

Osteitis: inflammation in localized area in cortical bone or in the socket but not spreading to adjacent bone.

**Alveolar osteitis (Dry socket):**

Inflammation in the socket following complicated extraction of a tooth caused by early lose of the blood clot leading to a dead space and starting of bacterial proliferation and infection.

Usually we find the mucosa and gingiva around the socket red , inflamed and tender.

Usually happens in the second or third day after extraction with severe pain at the site of extraction. Sometimes the patient comes blaming you that you have forgotten a piece of a tooth in the socket!

- Follows 1-3% of all extractions especially impacted mandibular third molars (because it is a difficult traumatic extraction).

- In a study in JU hospital: 9.5-10% of impacted wisdom teeth followed by dry sockets and it is close to the universal number.

- More in impacted mandibular wisdoms, nevertheless any tooth extracted has a chance to be followed by dry sockets

Aetiology:

Failure of clot formation or early clot loss from the socket

- Healing in normal conditions: clot – organization – bone formation

- Dry socket: no clotting and no normal healing which result in inflammation and pain after extraction

Predisposing factors:

Excessive trauma during surgery

Local anesthesia with epinephrine "vasoconstrictors” that cause ischemia to the area

Limited blood supply like patients taking radiotherapy

Osteosclerotic Bone diseases like osteopetrosis (marble bone disease) and Paget disease of bone.

Clinically:

-Deep-seated, severe throbbing pain in the extracted socket

Pain starts a **few days** after extraction and continues up to 2 weeks.

Empty socket with food debris filling the socket

Sever pain and trismus(if u put a probe it would be very painful)

-Healing is slow

Management by saline irrigation to reduce bacteria and putting Eugenol in the socket could relief pain. Also give painkillers and instruction

**Focal sclerosing osteitis (condensing osteitis):**

Bony reaction to a **low-grade** periapical inflammation

Caries → irreversible pulpitis →non vital tooth →chronic periapical lesion → bone deposition

-With acute inflammation usually we have bone resorption

- In this lesion there will be **condensation of bone** around the apical area

- Easily recognized: **non vital tooth**

-Most common in the lower first molar, young patients

- Asymptomatic

Management:

Management is for the symptomatic tooth. Do "RCT" or extraction

Radiographically:

Radio-opaque area below the apex, we can also see the radio-opaque area in the socket around an extracted tooth previously having condensing osteitis.

Histopathology:

- Thickening of bone trabeculae(bone scaring)

- Lamina dura and periodontal ligaments are present "unlike ankylosis".Ankylosis usually happen in patients with hypercementosis

**Osteomyelitis :**

Extensive inflammation spreading in the marrow spaces of the jaws, including cortical bone and periosteum.

- Uncommon disease in general to occur

Predisposing factors:

1- local:

\*Fracture in the mandible or maxilla

\*Radiotherapy: decreases blood supply (ischemia)

\*Osteosclerotic diseases: osteopetrosis, paget's disease→ decreases blood supply →more prone to infections

2- Systemic:

Related to loss of **immunity** →more prone to infections

Like acute leukemia, Diabetes Miletus, alcoholism, malnutrition, anemia, any immunodeficiency (genetic or secondary), AIDS

- Comes as **suppurative** "production of pus"

- classified into acute and chronic: depending on the duration and symptoms (acute or chronic)

- Source of infection:

Bacteria coming from the oral cavity like coming from the socket after extraction, periapical lesion, fractures, and sometimes coming from bacteria in the blood (bacteremia)

- Because the oral cavity is full of bacterial types, the infection is **poly microbial** not restricted to specific type so it needs broad spectrum antibiotic

Clinically:

- More in the mandible: related to the vascularity because the blood supply is less in the mandible because it depends on one major artery to supply it.

- Severe, throbbing, deep-seated pain

- Redness and swelling in the area "because it extends beyond the periosteum"

- Red, swollen & tender gingiva, pus coming from gingival sulcus around the teeth

Sinus tract and puss can appear in the oral mucosa or the skin of the face

- generalized loosening in teeth and tender on percussion

- Trismus: limitation of mouth opening & dysphagia( difficulty in swallowing)

- Enlarged tender lymph nodes (submandibular and cervical lymph nodes)

-Fever and malaise because it is systematic

- Anaesthesia or paraesthesia to the nerves passing through the lesion

Radiographs:

Immediately after infection: you can't see anything in the early stage; there won't be bone resorption

After 10-14 days, you will see **moth-eaten appearance**, there would be decreased in bone density

Histopathology:

Acute inflammation, necrosis, pus formation in the marrow spaces

Sinus opening or tract

Inflamed periosteum

Pieces of bone trabecuale detached and separated from the surrounding bone can be seen swimming inside the pus (**sequestrum**) that should be removed surgically.

**Sclerosing osteomyelitis:**

Bone formation instead of bone resorption

There is a debate that if it is really a chronic sclerosing osteomyelitis or superimposition of infection on a florid cemento-osseous dysplasia

**Chronic osteomyelitis with productive periostitis (Garre’s osteomyelitis):**

- Osteomyelitis inside the bone causing inflammation in the periosteum with reactive *subperiosteal new bone formation* causing swelling

- Bony hard swelling not soft tissue swelling as a result of deposition of bone under the periosteum

- Uncommon

In the area of the first molar in the body of the mandible and the source of infection is tooth-related mainly the first molar and then the infection starts spreading slowly from the periapical area till it reaches the periostium

- occurs in young adults

- Mild pain or painless swelling

- Non-tender, hard swelling along lateral border or the body of the mandible

Occlusal radiograph is the best radiograph to confirm diagnosis

You can notice layers of bone (***onion shape*)** deposited in the first molar region

**Chronic periostitis associated with hyaline bodies:**

Read about it for your own knowledge

**Osteoradionecrosis:**

Necrosis in the bone caused by radiotherapy that will affect mostly the mandible

It is not an infection but as radiation causes ischemia to the bone, it becomes more susceptible to infection

- radiation will cause changes within the bone→ decreased vascularity of the bone → bone necrosis→ this will make bone susceptible to infection (osteomyelitis) after any extraction, fracture or periapical infections

- This may lead to spreading of infection, exposure of necrotic bone, and ulceration and loss of soft tissues.

- Difficult to treat

\*Patients should visit the dentist and fix their teeth including extractions, RCTS and periodontal treatment before starting radiotherapy.

***Metabolic and endocrine disorders of bone***

**Osteoporosis:**

Bone formation is less than bone resorption during bone remodeling so the density and the amount of bone will be less

- Trabeculae will be thin, spaces will be more, and cortical plates will be thinner than normal bone

- increased susceptibility to fractures because the bone becomes weaker as the volume decreases

-Bone quantity decreases but bone as quality is normal.

-Radiolucency in bone is increased on radiograph because of more bone marrow spaces

-One of the signs we can detect is curvature in the vertebra and decrease in the height of the patient with time

Risk factors:

After menopause in woman

Hyperthyroidisim and hyperparathyroidisim

Cushing syndrome: increased corticosteroid level affecting collagen and bone density

-The mandible of patients with Osteoporosis is thin and alveolar height is decreased so there is difficulty in denture construction (retention wise) and implant placement

- bone loss causing periodontal disease→ loose teeth →teeth loss

- Upon surgery or extraction: fracture to the mandible could occur

- Alveolar bone resorption in the maxilla → closer to the sinus, so any extraction could cause oroantral complications

**Rickets and osteomalacia:**

Vitamin D deficiency or poor absorption of vit D and lack of adequate [calcium](https://en.wikipedia.org/wiki/Calcium) in the diet may also lead to rickets

Children: Rickets

Adults: osteomalacia

- Vitamin D could be present but not functioning

- Patients with renal failure and malabsorbtion of calcium are more susceptible

- Failure of mineralization of bone and osteochondral ossification will not produce bone "failure of mineralization of osteoid and of cartilage"

- Weak bones with susceptibility of **bending** to the knees

- Parathyroid gland will react and increase the secretion of Parathyroid hormone in order to increase the concentration of [calcium](http://en.wikipedia.org/wiki/Calcium) in the blood

- Calcium levels will be normal or reduced, phosphate level will be reduced, and alkaline phosphatase is increased

Patients with rickets should take vitamin D supplement as soon as possible

Dental aspects:

Enamel hypoplasia

Hypocalcification of enamel and dentine

Large pulp horns and chambers, dentine is thin: any attrition could cause early involvement of the pulp and pulpitis

Short roots: so early loss o teeth

Resorption of lamina dura

Hypoplastic alveolar bone

Late eruption because there is a problem in the roots

Premature loss and exfoliation

Condyles are affected: condyles are centers of growth and that could lead to patients with small mandible

**Acromegaly**:

Increase of growth hormone in the adult patient

Prolonged and excessive secretion of growth hormone after the epiphyses closed.

Affects **flat** bones more than long bones

- Excessive growth of the bone and soft tissues

- The mandible is enlarged more than maxilla with deposition of periosteal bone, broadening and prognathisim, and cross bite (skeletal class 3)

Diastema and spacing: the jaws are enlarged and teeth stay in their original position

Lips and nose enlargement and coursed facial features

Tongue enlargement "macroglossia"

Hands and feet enlargement

Radiographically on lateral Cephalometry we can see Silla Tursica (where the pituitary gland is located) enlargement

**Primary Hyperparathyroidism:**

Caused by adenoma, hyperplasia or carcinoma in the pituitary gland

Parathyroid hormone is responsible for regulation of the concentration of [calcium](http://en.wikipedia.org/wiki/Calcium) in the blood. It brings calcium form the GIT and from the bone to the blood if there is a decrease in its level in the blood

So on the long run, there is a decrease in bone density

Predominantly seen in post menopausal women

- can be seen in young patients and therefore would be responsible for the dental abnormalities

-Hyperparathyroidisim will cause hypercalcemia, calcium deposition in certain areas, metastatic calcification, renal stone formation and renal failure on the long run!

Patients usually have renal stones, psychic moans and abdominal groans

- It induce bone resorbtion by osteoclasts in order to increase blood calcium level and that will decrease the density of bone and so looks radiographically like osteoporosis

- Increased osteoclastic activity and bone resorption in certain areas result in the formation of lesions called **brown tumors (giant cell lesion of bone)**

Brown tumor: collection of osteoclasts in a fibrovascular stroma with multilocular radiolucencies in the mandible (resemble: cherubism and central giant cell granuloma)

- It is called brown tumor because of the presence of hemosiderin pigment caused by high blood concentration.

- Radiographically, it resembles osteoporosis, thinning of the cortical base of the skull, salt and piper scatter (mottled Radiolucency)

Loss of the normal trabecular pattern in mandible and maxilla

Loss of lamina dura

Loosening of teeth

Multiple radiolucencies in the area of brown tumor

Biochemistry (blood test), increase in calcium and parathyroid hormone and alkaline phosphatase

Phosphate levels are abnormally low as a result of decreased renal tubular phosphate reabsorption. This contrasts with secondary hyperparathyroidism, in which serum phosphate levels are generally elevated because of renal disease.

Urine sample: increased calcium and phosphate

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