***Diseases of Bone Manifested in the Jaws***

Systemic Bone diseases are very characteristic in radiographic images :

1-they can be present in all the body bones

2-affect bilaterally.

3-and if it affect the head and neck you will find them as will in the rest of the body bone .

- generally all systemic diseases manifested in the jaws change in size , shape , trabecule , thickness and density of bone …and some of them have affect on the teeth .

- Most of them decrease in bone density but some of them increase in the bone density .

Some of these diseases are related to a metabolic disease (ex: osteoporosis , hyperphosphatemia , hypophosphatemia ) to the endocrinal ( hyper/hypo parathyroidism , hyperpityotary ).

-a lot of these diseases have the same radiological appearance but what is important to us as a GP is to know that this is a systemic disease (I would not take a biopsy instead I would regulate the hormonal level in the body ) because it change the way of thinking . also it is important to see wither it is affecting the pt in young age or in adulthood .

\***\*hyperparathyroidism** :

Increase of the hormonal secretion that leads to More mobilization of calcium outside the bone and increasing its amount in the blood .-

Caused by : 1- problems in parathyroid gland cells ( primary ) .-

2- problem in calcium phosphorous metabolism ( secondary ) associated with renal disease .

3- state of excessive secretion of [parathyroid hormone](http://en.wikipedia.org/wiki/Parathyroid_hormone) (PTH) after a long period of [secondary hyperparathyroidism](http://en.wikipedia.org/wiki/Secondary_hyperparathyroidism) and resulting in [hypercalcemia](http://en.wikipedia.org/wiki/Hypercalcemia). ( tertiary ) also associated with renal disease .

So it affect the phosphate and calcium balance but that does not means there is no minerals in the body it actually means that it is going to be in the soft tissue .

-signs and symptoms:

Pain in the bone -1

2-Pathological fracture

3-GI disturbances and kidney stones .

In the radiographs :

Cortical bone will be very thin or goes away in severe cases.

More bone marrow spaces and less amount of bone trabeculae

Lamina Dura is not visible any more.

Brown tumor ( brown because of hemosiderin , and filed with giant cells ,so it is a giant sell lesion ) it is the only manifestation of the disease that you need to surgically approach ( by inoculation ) , because the other problems will go back to normal if you correct the hormonal imbalance .

\*\* how to know that there is a brown tumor not something else :

Since it is a systemic disease that affect all the body bone and the symptoms appears every where including this lesion(brown tumor) (localized lesion with systemic back ground), while if you see it by itself and the symptoms appears in localized area and not in other area (localized lesion without systemic back ground manifested in the bone ) we may think of something else like giant cell granuloma.

-so the main cause is the systemic disease while the localized lesion is one of its manifestations .

Since osteoporosis and hypophosphotemia have the same radiographic appearance as will some other diseases how to differentiate between them :

Any systemic disease that take the minerals out of the bone or prevent the bone from being completely mineralized they all will be manifestating the same radiographic appearance so they are differentiated by the clinical sign and symptoms because if for Ex a pt who is 15 y old inter the clinic with this radiographic appearance you do not think of osteoporosis we would think of ricket disease if the teeth are involved or osteogenesis imperfecta if there is a fracture in the Wight baring area or problems in the sclera ,also we need blood tests to see if there is an abnormal disturbances like (ca and phosphate level is disturbed by for example hyper parathyrodesim ).so we can not only depend in the radiographic image.

\*\* **Hyperpituitarism**

Tow type 1-giantesime (from childhood)

2-acromegalea (adult )

When you take a cephalometric image everything will be enlarged especially the mandible will be protruded . also the sella turcica will be large .

\*\* **Hyperthyroidism**

It Increase the metabolism and it does not affect the bone in particular (redoigraphicly)

But if it affect young pt it will affect the teeth eruption and exfoliation )

Like hypothyroidism ….lead to retuned primary and delayed eruption if the permanent .

\*\***Osteoporosis:**

Similar to any disease that lead to thinning if the cortex and irregularities if the trabiclea (mostly affect the females )

\*a lot of studies have been done to see if it is possible to make the panoramic x-ray used as screening tool , the only thing that was logical and possible so fare is the *thickness of the inferior cortex* / vertical level of the mental foramen they meagered it and see if it is less than 5 mm it may means osteopenic pt.

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**RICKETS & OSTEOMALACIA**

-result from inadequate serum & extracellular levels of calcium &phosphate ,minerals required for the normal calcification of bone & teeth.

-result from adefect in the normal activity of the metabolites of vitamin D.

-rickets 🡪 the disease affects the growing skeleton in infants & children

-osteomalacia 🡪 this disease affects the mature skeleton in adults .

**\*\*radiogrphic features :**

**-Radiographic changes a**ssociated with the teeth in **Rickets** , rickets in infancy or early childhood may result in hypoplasia of developing dental enamel . if the disease occurs before the age of 3 years ,enamel hypoplasia is fairly common . radiographs may reveal this early manifestation of rickets in unerupted & erupted teeth . lamina dura & cortical boundary of tooth follicles may be thin or missing .

-Osteomalacia does not alter the teeth because they are fully developed before the onset of the disease , the lamina dura may be thin with long standing or severe osteomalacia . in osteomalacia , bone problem occur , thin cortices and pseudo fractures but teeth are not involved.

-bone changes are the same in the rickets & osteomalacia , bones are soft in general , deformity occur especially in weight bearing bones , so legs bowing occur and greenstick fractures appear .

**HYPOPHOSPHATASIA**

-rare inherited disorder that is caused by either reduced production or defective function of alkaline phosphatase "this enzyme is required for normal mineralization of osteoid" .

-we see this disease in adults , because in the children🡪 rare , in infant 🡪very severe'' fetal''

-teeth + bone are affected….. "this is the idea 🡪 in systemic desease🡪 the bone & teeth are involved"

-like any disease without proper mineralization …. Poor growth ,fractutes , closure proplems,poor calcification .

-generalized radiolucency of the mandible & maxilla , cortical bone & lamina dura are thin ,& alveolar bone is poor calcified & may appear deficient….. both primary & permenant teeth have thin enamel & large pulp chambers & canals , the teeth may be hypoplastic & may be lost prematurity .

**RENAL OSTEODYSROPHY**

* -Renal disease is associated with secondary hyperparathyroidism 🡪 with radiolucent apperance
* -long term renalfailure may be give radiolucent appearance OR radiopaque appearance''sclerotic appearance ''
* -in the radiographs 🡪radiolucency, no cortex "very thin " black area",loss of bone mass, loss of lamina dura ,"resorpative pattern"
* - in other pic 🡪 sclerotic "radiopaque" bone"sclerotic pattern"

- **HYPOPHOSPHATASIA**

-vitamin D resistant rickets , it exactly look like it ,,but the gentic background is the deffrence

- teeth will be affected

-soo clinically & radiographs are similar

- in the radiographs 🡪 thers deffrential diagnosis .. may br rickets ,hypophosphatemia, hypophosphatasia,hyperparathyroidism ….. sooo it depends on history background ("family history ,,, or about sclera "about osteogenesis imperficta " ,,,renal disease …..)

**OSTEOPETROSIS**

-albers-schonberg &marble bone disease

-inherited(recessive & dominant)

-we have too much bone

-sereously dense bone

-the bone is dens ,fragile that are susceptible **to fracture**" brittle"& infection

-results from defect in the defrentiation & function of osteoclasts . the lack of normally osteoclasts results in abnormal formation of primary skeleton & generalzed increase in bone mass

-the pt. has progressive loss of the bone marrow & its cellular products & severe increase in bone density.

-good case to osteomylitis start

- orally 🡪 impaction problems

-osteopetrosis showing dense clacification of all the bones , skull , facial,chest, pelvis …..

- we cant do surgery"risk of osteomylitis" for this pts. Soo …prevention,fluoride. scaling

Other systemic disese:

**Sickle cell anemia & thalasemia**

-hemolytic disorders

-theres active bone marrow space

-hair on end appearance

-clinically …

زي السنجاب

The face develops prominent cheekbones & protrusive premaxilla

-n the radiographs :

Thick diploic space , thin cortex , hair-on-end bone pattern , granular appearance of the skull , large bone marrow spaces , change in the bone shape,,,,,, thick body of mandible

-they come to clinic because of face shape " CC" ,,,

**-SCLERODERMA**:

- connective tissue problem,, in collagen ,,, the pt come to clinic with limited mouth opening, tight skin………

- causes 🡪symmetric & generalized widening of periodontal ligament space

***Islam AL-Dagag***

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