Part 2 sheet 4

RICKETS & OSTEOMALACIA

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

-<u>rickets</u> \rightarrow the disease affects the growing skeleton in infants & children(affect teeth since they are still developing)

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

****radiogrphic features :**

Looks radiographically same as hyperparathyrodism

Thin cortex, bone radiolucent,

-Radiographic changes associated with the teeth in <u>Rickets</u>, rickets in infancy or early childhood may result in hypoplasia of developing dental enamel . 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 <u>does not</u> 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.

Pt usually has taken for them skull and panoramic x-ray

-bone changes are the same in the rickets & osteomalacia.

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" .

-infant &adult type depends on teeth development.

-radiograph same as OSTEOMALASIA —>can't differentiate between them

-absent of cortex in sever cases

-less opaque bone, thin cortex, wide pulp, small roots, delayed development of teeth.

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

- the teeth may be hypoplastic & may be lost prematurity .

RENAL OSTEODYSROPHY

-Renal failure disease →affect Ca&pi balance -

-long term renalfailure may be give radiolucent appearance <u>OR</u> radiopaque - appearance"sclerotic appearance " in some patients depends on what stage of failure & what treatment they are taking.

-thin cortex -

-bone resorption -

-in the radiographs \rightarrow radiolucency, no cortex "very thin " black area",loss of - bone mass, loss of lamina dura "resorpative pattern"

- in other pic \rightarrow sclerotic "radiopaque" bone"sclerotic pattern" -

- <u>HYPOPHOSPHATEMIA</u>

-in phosphate enzyme while PHOSPHOTASIA in phosphorus —>both looks the same radio graphically

-infant &adult type

- in the radiographs ightarrow 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

-easily detected on radiograph Problem in osteoclast

-sereously dense bone

-the bone is dens ,fragile that are susceptible <u>to fracture</u>" 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
-imbalance between osteoclasts and osteoblasts
-bone marrow spaces don,t exist enough—>affect vascularity of the bone
-foraminae getting nearly moderne &title—>neural issues
<u>Clinical features:</u>-multiple fractures
-neural issues
Pt grow full potential
it's not one disease it bas 2 subtypes &there's types incompotent with life some

-it's not one disease, it has 3 subtypes, & there's types incompetent with life, some are mild enough so they can came to our clinic.

-in some cases the dense bone prevent teeth from eruption(impact ion problems)

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

- pt has high tendency to fractures &osteomyelitis,infections(especially in ill fitting dentures)

Other systemic disese:

Sickle cell anemia & thalasemia

-hemolytic disorders, defective RBCs

-theres active bone marrow space (hyper plastic bone marrow)

-hair on end appearance(bone marrow is growing,trabecular cells space more من التركيبة العشوائية العادية(vertical

-can't differ between them but depend on pt origin (African pt—>sickle cell anemia,Lebanese pt—>thalassemia)

The face develops prominent cheekbones & protrusive premaxilla

-n the radiographs :

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

-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 Crystal bone level is normal,lamina dura is intact&no pocketing(to differentiate from periodontitis).

DONE BY:RANEEM ALBAGHDADI