***bone lesions:***

***Today we will talk about bone disorders ,,there are many categories :***

1. Inherited and developmental disorders:
2. Osteogenesis imperfect

is defined as Excessive fragility of bone due to a Defect in synthesis of type I collagen. This leading to an Inadequate or less formation of bone ® generalized osteoporosis.

Features:

-Slender,weakness, deformity of bone and fracture tendency

- Dwarfism happen if they become alive

- Thin skull and bulge over ears

- Thin sclera and may be colored (sometimes blue)

-± deafness ,deformity in the ears

- joint hypermobility,

-heart valves defects ( for our dental concern, need prophylactic )

IMP ;not every pt have blue sclera nesserty to have OI

- 4 main types that are variable in severity and pattern of inheritance

 Type I: AD, Blue sclera, ± Dentinogenesis impefecta

 Type II: Ar lethal; pts die directly after birth

 Type III: AD or AR common type , Dentinogenesis imperfecta ,blue sclera  pts live but with progressive deformity that increases throughout life

 Type IV: AD, White sclera, ± Dentinogenesis imperfecta

-Dental aspects:

\*\*Dentinogenesis imperfecta

\*\*Extractions with precaution

\*\*class 3 malocclusion

\*\*imapction specially 1 and 2 molar

\*\*difficulty in respiratory system

1. Osteopetrosis bone like marble (رخام ) dense but easy to fracture ,bone formation more than resorbtion

 (marble bone disease)

Features:

• Solid dense but brittle bones

• Inactivity of osteoclasts (no bone resorption) excessive bone formation on expense of bone marrow spaces causing anemia

• Excessive bone formation but woven

• Fracture

• Anaemia and hepatospelonmegaly

 Rx:

\_ Generalized ⇑ in bone density

\_ Cortical = medullary ; no medullary spaces only dense bone

\_ Marked radio-opacity of skull base

\_ Greatly reduced sinuses, for example reduction in maxillary sinus space.

\_ greatly reduced skull foramina which causes pressure on the cranial nerves that pass these foramina leading to neuropathy and facial palsy.

Dental aspects:

1. Invisible roots

2. Unerupted teeth

3. spread of infection easily because of the dense bone leading to Osteomyelitis

1. ***Cleidocranial dysplasia***

Features:

• mostly AD/ some Sporadic cases

• affecting Face, skull & clavicles

• Fontal & occipital bossing, open fontanells & sutures  bulging deformity

• Underdeveloped midface, depressed maxilla & nasal bridge

• Normal size mandible however class 3 malocclusion cuz of maxilla

• Partial or complete absence of clavicles

Chest radiographs:

Partial or complete absence of clavicles

Dental aspects:

1. Retention of primary teeth

2.Multiple unerupted permanent teeth

3. Multiple supernumerary teeth & dentigerous cysts

4.Thin roots and hypercementosis ( in cellular cementum)

5.Achondroplasia

Features:

• Most common genetic skeletal disorder

• Short-limbed dwarfs

• Cartilage proliferation in epiphyses & skull base

• Head & trunk of normal size however Defective middle 1/3 of face (nonproportional unlike the dwarfism that is caused by defect in growth hormone secretion)

• Malocclusion class 3

2)Fibro-osseous lesions:

fibrous tissue replacing bone then gradual calcification occurs

A variety of diseases which, histologically, are characterized by the replacement of normal bone by cellular fibrous tissue within which varying amounts of predominantly woven bone and acellular islands of mineralized tissue develop.

Types :benign

1. Osseous dysplasia
2. Fibrous dysplasia enlargement of bone

 ( most common; as in 1 or 2 cases may be encountered all through your career life)

\_ Monostotic FD: unilateral affecting one bone any part of body

• 80% of cases most common

• One bone

• Max > Mand

• could be Craniofacial FD affecting more than one bone

•diagnosed during the growth period in Childhood, arrest in adulthood

 Clinically:

\_clinical presentation: Facial asymmetry; best inspected from above the head

\_ Not well defined in early stages, hard,smooth, round, painless bony swelling

\_normal skin, don’t confuse it with infection, persistent, been there all life long

\_ Disturbs function & cases malocclusion

\_ Extension to max sinus if in maxilla

\_found in posterior Mandible mostly in body of mandible

\_the diagnosis depend on biopsy

 Rx: depending on the stage

\_ it appear Radiolucent in early stage (fibrous )then calcification to appear radio-opaque(

Lateground glass or orange peel or thumb print pattern appearance. Not a normal trabecular appearance)

\_ Displacement of roots, obscured lamina dura; & thin PL space in the affected area

\_superior displacement of mandibular canal as it push the mandibular canal

\_ Polyostotic FD:

• Expansion in multiple bones

• Segmental or one side of body

• F>M

affecting multiple bones + café-au-lait spots on skin + endocrine defects; hyperthyroidism, hyperparathyroidism, precocious puberty called Albright syndrome at all

Histology:

• Initially: cellular CT replacing normal bone

• Gradually: deposition irregular, immature trabeculae,delicate woven bone = Chinese characters

• Osteoclast-like giant Cs may appear

• Border: well defined( no capsule )

• Late stage decrease in fibrous connective tissue change of woven to lamellar bone

• Spherical calcification

Blood chemistry:

± increase in Alkalinephosphatase cuz increase in bone activiy

Aetiology:

• unknown

developmental occurring in growth period (childhood), defect in gene controlling growth and differentiation of osteoblasts

Prognosis:

• Fibrosarcoma 1% speacilly pt treated by radiotherapy

• Cosmetic surgery

1. Cemento-osseous dysplasia

***¬ Periapical cemental dysplasia***

• Asymptomatic, diagnosed radiografically by chance (incidental), no deformity present, no expansion, no pain

• Middle-age F, African-Americans

• Below apices of mandibular incisors

• Multiple radiolucencies

• Vital  this point cuts out the diagnosis if nonvital we would have thought of other DDs such as: chronic alveolar abscess, periapical granuloma.

• Very rare and not found in our region

Rx:

\_ Well-defined radiolucency below apices separated by normal PL

\_ Increasing radio-opacity

\_ Thin radiolucent margin like a fibrous capsule

\_root resorption is rare not like the cementoblastoma which is also not multiple like this periapical cemental dysplasia

Histology

\_ Cellular CT & then gradual calcification

***¬ Focal cemento-osseous dysplasia***

A localized variant of periapical cemental dysplasia that is not multiple, white people, posterior mandible area , well defined, starts as radiolucency then mix then radiopaque, normal PL space

***\_. Florid cemento-osseous dysplasia***

• Extensive Periapical cemental dysplasia ≥ 3 quadrants

• More dangerous and more common

 Clinically:

\_ Same group of pts ( African American

\_ Asymptomatic unless infected led to osteomyelitis

\_ expansion is present in bone

 Rx:

\_ Radio-opaque, irregular masses

\_ Frequently symmetrical

\_ May involve 4 quadrants

•Histology: = PCD ,Cellular CT & then gradual calcification; irregular abnormal bone trabecullae

2. Benign neoplasia

¬ Cemento-ossifying fibroma

\_ Similar to Fibrous dysplasia histologically & clinically

\_bony enlargement

Similarities:

\_ Slowly growing, painless swelling, gradual calcification on Rx.

Differences:

1\_age group: 20-40ys

2\_well defined radiolucency

3\_radiolucent rim (easily resected),,,,,, capsule mean

4\_ Mandibular molar & premolar region

Imp one is the spherical calcification is more

• Rx:

\_ well defined radiolucency

\_ Gradual calcification

\_ Radiolucent rim

• Histology:

\_ Cellular FCT

\_ Outer zone of FCT

One type of cemento-ossifying fibroma is: Juvenile ossifying fibroma.

\_ rapidly growing

\_ < 15 ys

\_ Richly cellular & mitotically active FCT w trabeculae of woven bone

\_ ⇑ (high recurrence rate)

DONE BY :FARAH ALNAJDAWI

**GOOD LUCK**