Aurobindo College of Dentistry

Indore, Madhya Pradesh

INDIA

Module plan

- Topic : Diseases of bones and joints
- Subject: Oral Pathology
- Target Group: Undergraduate Dentistry
- Mode: Powerpoint – Webinar
- Platform: Institutional LMS
- Presenter: Dr.Shradha Jaiswal

Index

Definitions

- Osteochondrodysplasias- abnormalities of cartilage or bone growth and development.
- Dysostoses-malformations of individual bones, single or in combination, does not refer to generalised disorder of skeleton

Group 1
Defects in extracellular structural proteins
OSTEOGENESIS IMPERFECTA

- Brittle bones,lobstein's diseases
- Abnormality of type I collagen
- Hereditary, AD or AR
- Total 7 types described
- Four of them have genetic basis while type V,VI and VII- genetics have not yet been determined

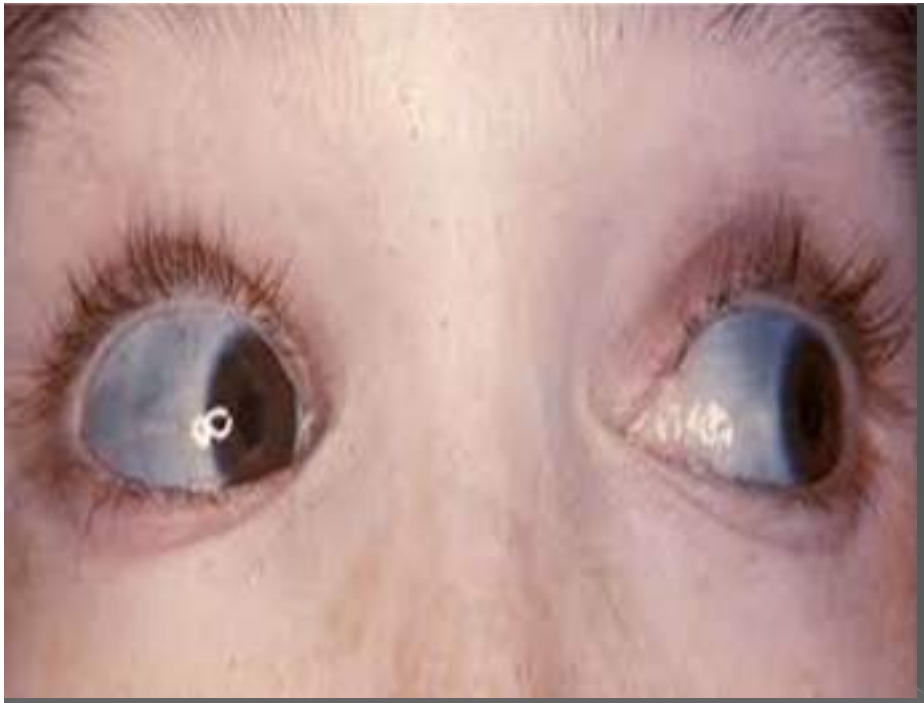
- Collagen composed of intertwining of proa1 and proa2 chains.
- Cause: mutation in loci coding for chainsCOL1A1 on band 17q21 and COL1A2 on band 7q22.1
- Both qualitative and quantitative defects exists

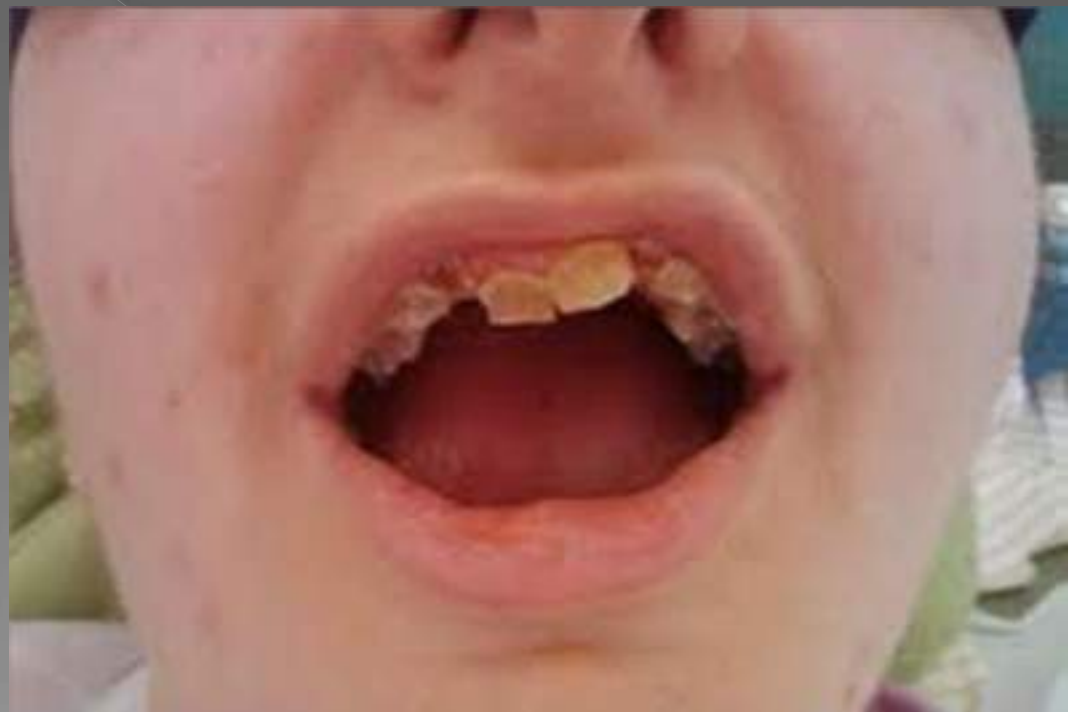
Pathogenesis

- Basic defect lies in organic matrix –failure of fetal collagen to get converted into mature collagen(qualitative defects)
- Quantitative defects-decreased production of normal collagen

Clinical features

- Extreme porosity and fragility of bones with a tendency for fractures
- Fractures heal readily but healing is by defective collagen=again fracture
- Prenatal screening in 2nd trimester shows bowing of bones, fractures, limb shortening and decreased skull ecogenecity
- Blue sclera- abnormally thin sclera-hence pigmented choroid visible
- Blue sclera also seen in- infants, osteopetrosis, fetal rickets, turner's syndrome, pagets disease, marfen's syndrome, Ehler's Danlo's syndrome.





Sillence classification

Features	Type I	Type II	Type III	Type IV
Age	Infancy	Utero	Half utero/ half neonatal	Infancy
Severity	Mildest form	Still born /die before 4 weeks	moderate	mild
Dentinogenesis imperfecta	Subtype A-absent Subtype B-present	May be present	+nt	Subtype A-absent Subtype B-present
Blue sclera	present	May be present	Variable hue	absent
Bone fragility	Mild-moderate	extreme	+nt	Fractures in infancy
Other features	Kyphoscoliosis, hearing loss,easy brusing, short stature	Small nose, micrognathia & short trunk	Limb shortening, triangular facies, frontal bossing, pulmonary hypertension	Bleeding diathesis not reported

Oral manifestations

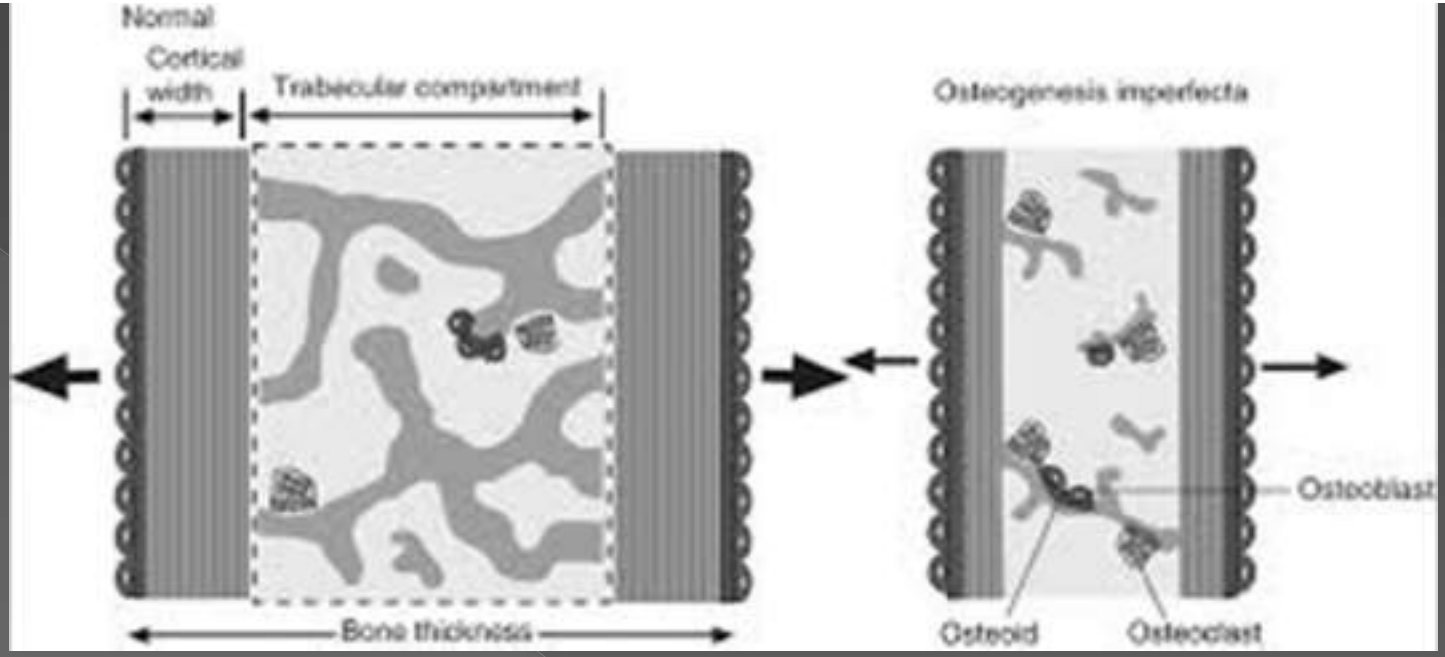
- Large sized head, Frontal and temporal bossing, Exaggerated occiput =Create class III malocclusion
- Anterior and posterior crossbite and anterior open bites seen
- Caused by maxillary hypoplasia rather than mandibular hyperplasia
- Impactions and ectopic erruptions
- Usually show unerrupted permanent 1st and 2nd molars

X-Ray

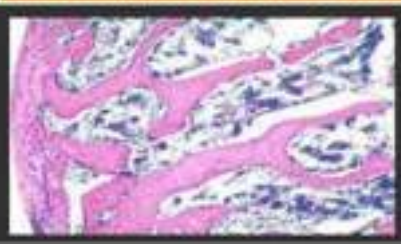
- Osteopenia
- Bowing
- Angulation & deformity of long bones
- Multiple fractures
- Womarian bones in shull

Histology

- Thin cortices
- Immature spongy bone
- Trabaculae of cancellous bone are delicate showing microfractures
- Osteoblastic activity retarded & imperfect



Microscopic changes of OI



Treatment

- Nil

Fibrous dysplasia

- Defect in osteoblastic differentiation and maturation
- Non heriditary
- Cause unknown
- Mutation in gene GNAS1
- Medullary bone replaced by fibrous tissue-appears radiolucent on xray
- Trabaculae of woven bone contain fluid filled cysts, embedded in collagenous matrix

Pathogenesis

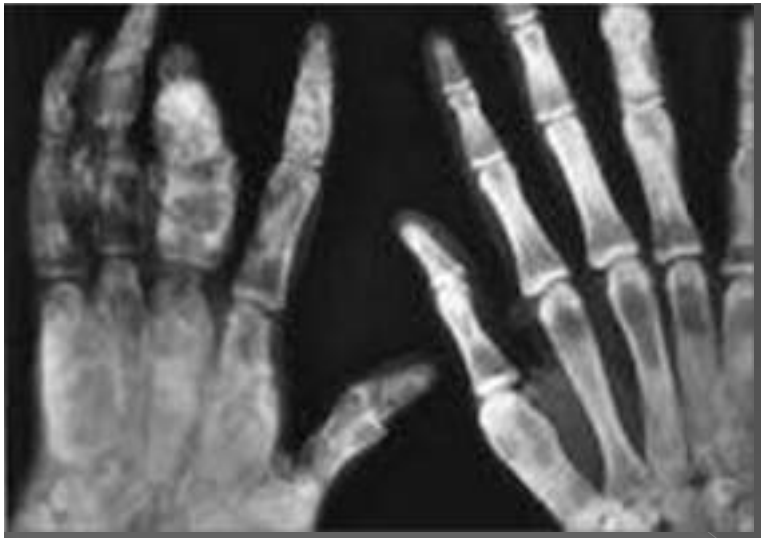
- Mutation in GNAS 1 gene
- Gene encodes G-protein that stimulates production of cAMP
- Mutation results in continious activation of of G protein leading to overproduction of cAMP in tissues
- This results in:
- Results in hyperfunction of affected endocrine glands- hyperthyroidism, growth hormone and cortisol overproduction
- Increased proliferation of melanocytes=café au lait spots with irregular margins
- Affects differentiation of osteoblasts

Types

- Monostotic
- Polyostotic
- Craniofacial
- Polyostotic form-3-15years(asymptomatic before 10yrs)
- Monostotic form-20-30yrs asymptomatic

Monostotic-Clinical features

- Common in children and young adults
- Mild female predominence
- Mean age 27-33yrs
- Constitutes 70-80 % of all fibrous dysplasia
- common in rib, femur, tibia, craniofacial bones, humerus
- Pain
- Pathologic fracture
- Bone deformity less sever



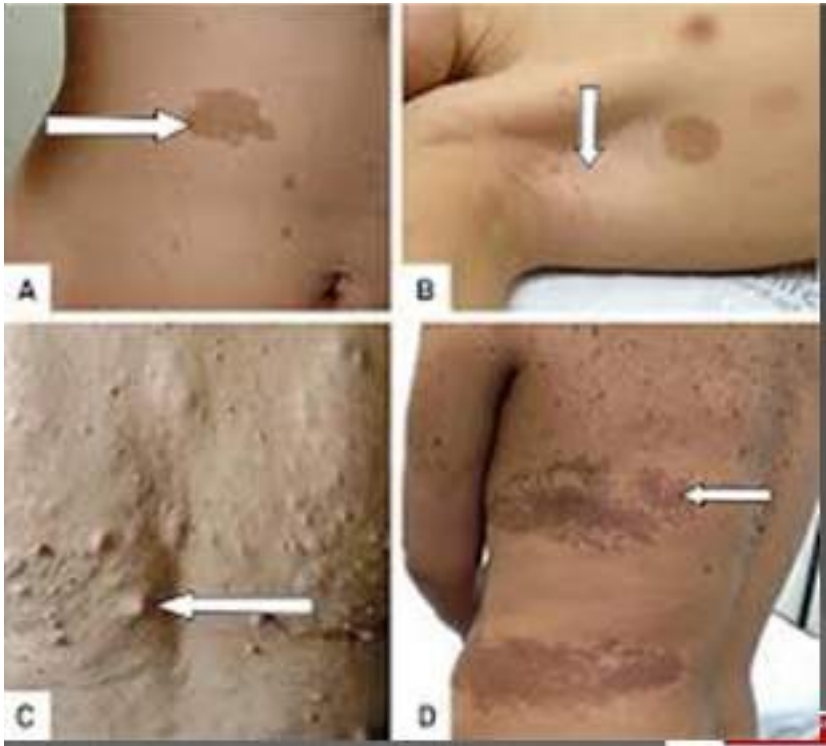
Oral manifestations

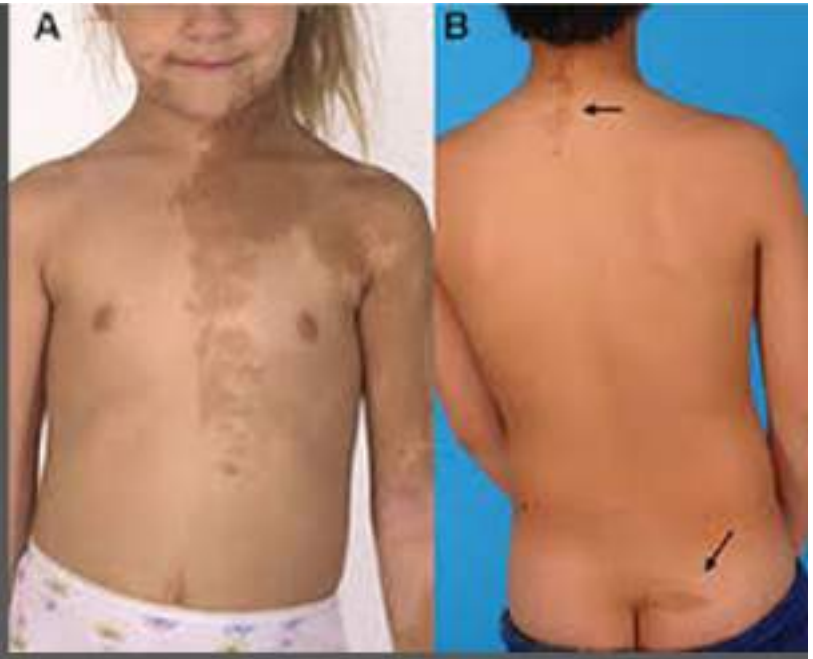
- First sign-painless swelling involving buccal/ labial cortical plate
- Displacement of teeth
- Tenderness
- Overlying mucosa intact
- Maxillary involvement serious-extends to sinus, zygoma, floor of orbit

Polyostotic-Clinical features

- Constitutes 20-30% of all fibrous dysplasias
- Femur, pelvis, ribs ,skull, facial bones, clavicle, spine
- May be uni/bilateral
- Pain, Pathologic fracture
- Endocrine disturbances-hyperthyroidism, acromegaly, cushings syndrome, hyperpara thyroidism,hypophosphatemic rickets
- May also show GIT, hepatic, cardiac

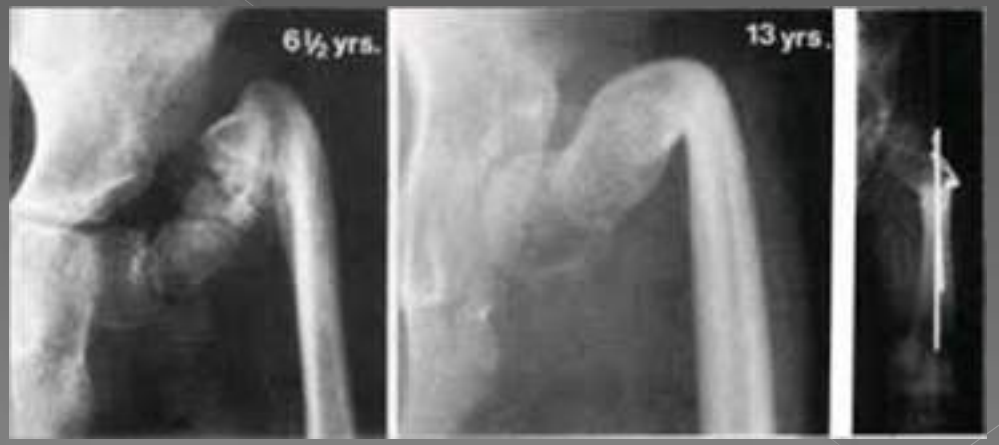
- Cutaneous pigmentation on the side of bony lesions
- Pigmentation may occur at birth or preceed the development of skeletal/endocrinal abnormalities
- Mazabraud's syndrome=fibrous dysplasia+ intramuscular myxoma
- Malignant transformation seen in McCune- Abright syndrome- osteosarcoma, chondrosarcoma,fibrosarcoma,


A
B
C
D


A
B


NEVER CROSS MIDLINE
CROSS THE MIDLINE
IRREGULAR BORDERS
SMOOTH BORDERS
COAST OF MAINE
COAST OF CALIFORNIA

- Bone deformity – sheperds crook- curvature of shaft of femur



Cauf –au- lait spots

Jaffes type

- FD involving variable bones
- Pigmented lesions of skin

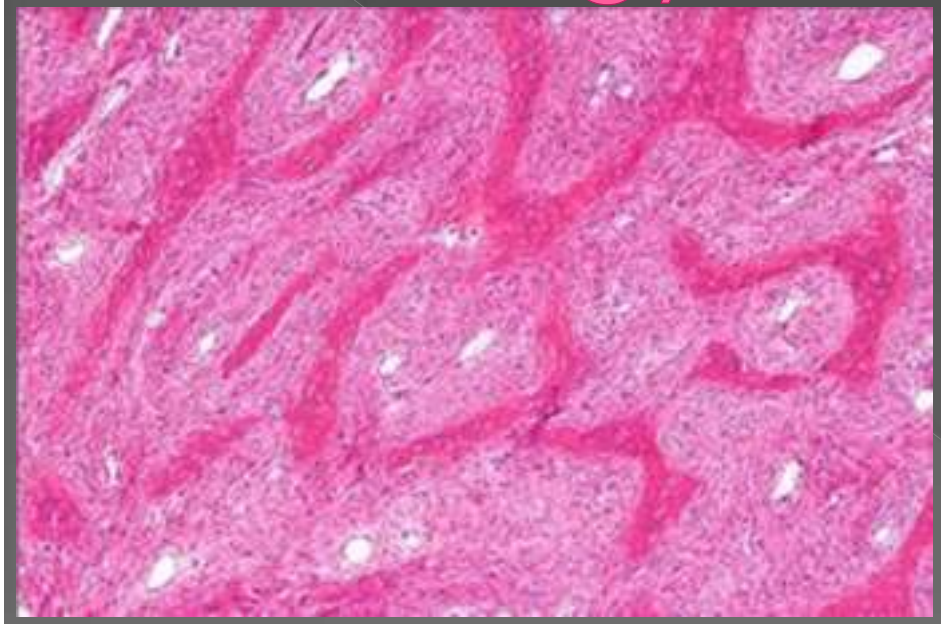
Albright type

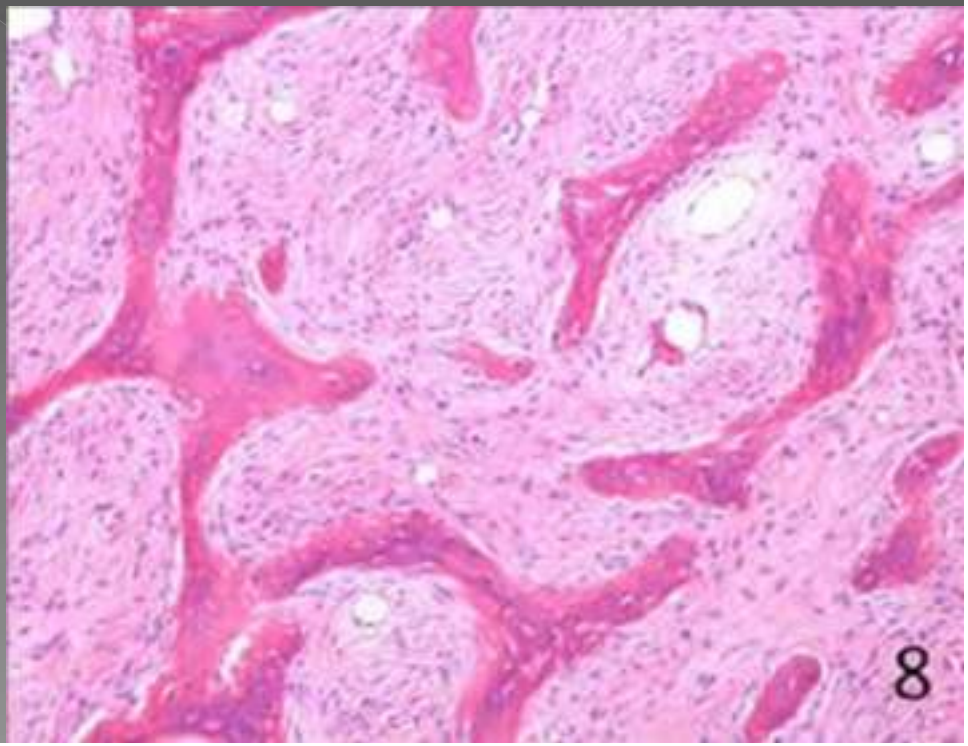
- Almost all bones
- Pigmented lesions of skin
- Endocrine disturbances

Craniofacial form

- Occurs in 10-25% cases of monostotic and 50% of polyostotic
- May also occur as isolated lesion-frontal, ethmoidal, sphenoidal and occipital bones
- Hypertelorism, cranial assymetry,facial deformity,visual impairment, exophalmos,blindness due to involvement of orbital and peri-orbital bones
- Involvement of sphenoid and temporal bones results in tinnitus, vestibular dysfunctionand hearing loss

Histology





Pagets disease

- Also known as osteitis deformans.
- Chronic progressive disorder of bone.
- Named after a British surgeon Sir James Paget who first discovered it.
- skeletal disorder of middle aged and elderly patients involving multiple bones.
- Very rarely a single bone may be involved.

- Characterized by abnormal and excessive remodelling of bone.
- Increased osteoclastic activity.
- Osteoclasts show increased sensitivity to 1,25-dihydroxy vitamin D, receptor activator of NF kappa B ligand (RANKEL), and interleukin -6, which stimulates osteoclasts and promotes bone resorption.

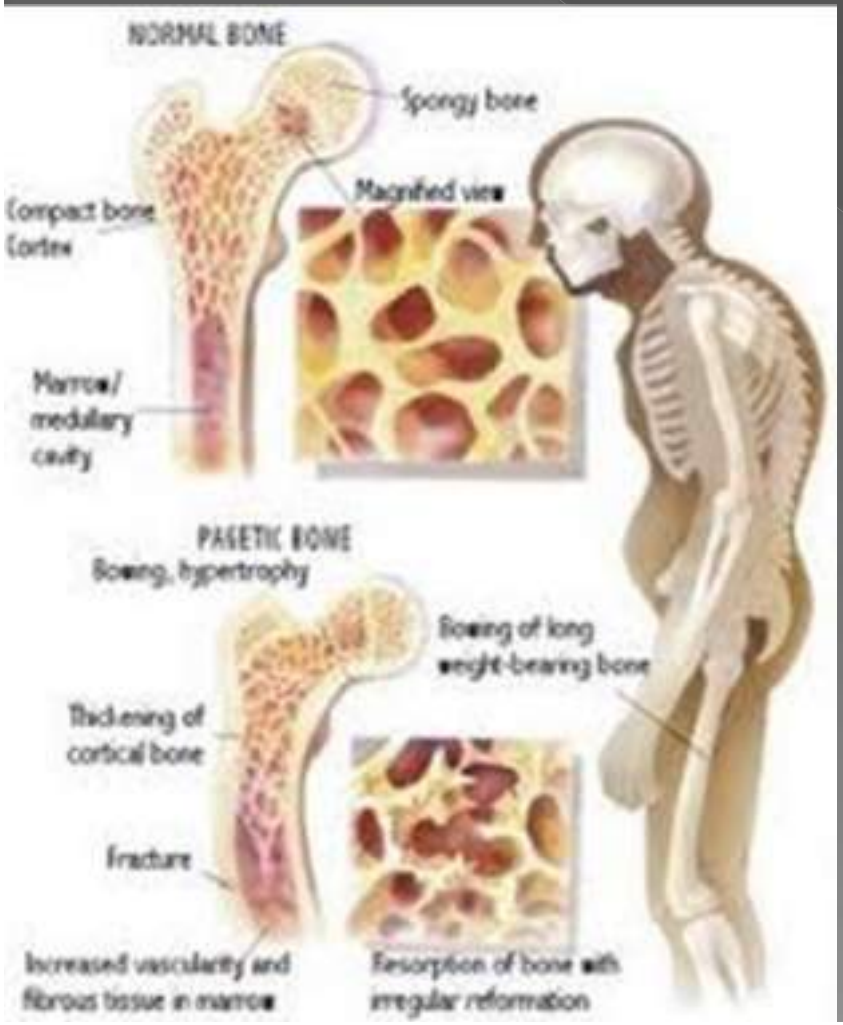
- An increase in number of osteoblasts is also seen at the site of pagetiod bone which is to compensate for the bone resorption.
- Linked to genes PDB1-PDB-7, spanning over chromosomes 2,5,6,10 &18.
- Viral etiology has also been suggested

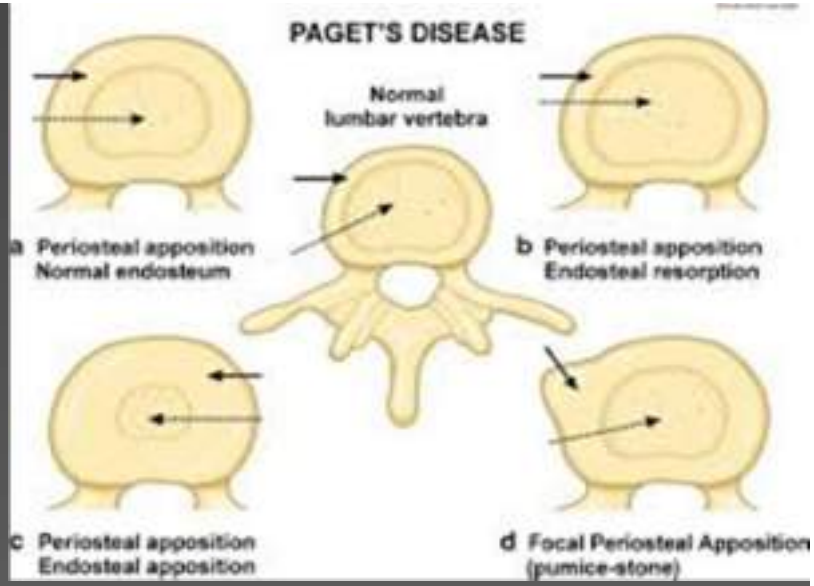
Clinical features

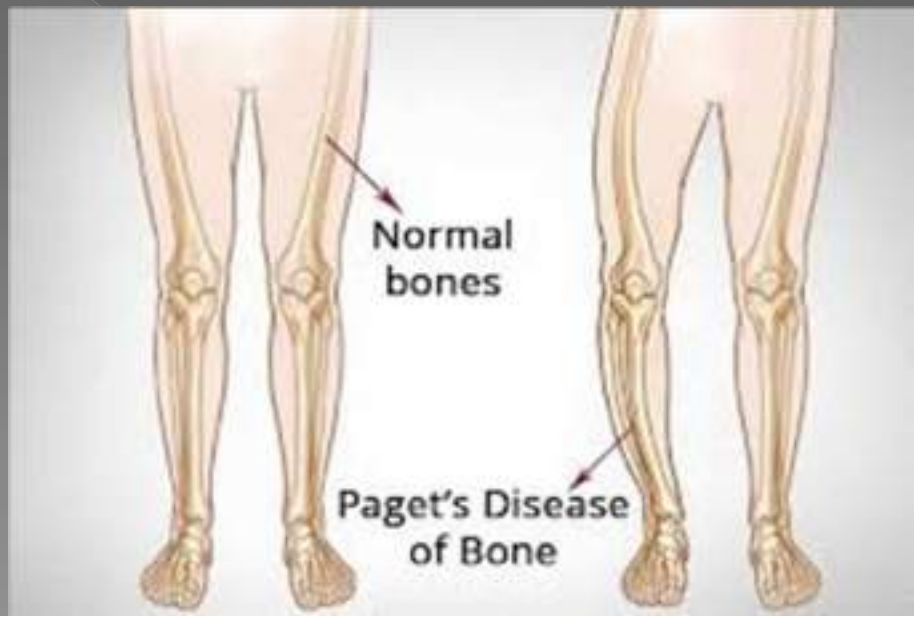
- seen after 50 yrs
- no sex predilection
- more common in European countries
- Usually asymptomatic
- Some patients may complain of bone pain, bone deformity, neurologic, muscoloskeletal and CVS abnormalities
- Predilection for axial skeleton

- Commonly involved bones include femur, spine, tibia, skull etc.
- Affected bones show bone expansion and deformity
- Bone pain worsens at night
- Long bones may bow due to rigidity
- Softened bones at the base of the skull may lead to PLATYBASIA= descent of cranium into the cervical spine

- Skin over affected bones feel warm to touch


NORMAL BONE
Spongy bone
Magnified view
Compact bone
Cortex
Marrow/
medullary
cavity
PAGETIC BONE
Bowing, hypertrophy
Bowing of long
weight-bearing bone
Thickening of
cortical bone
Fracture
Increased vascularity and
fibrous tissue in marrow
Resorption of bone with
irregular reformation


PAGET'S DISEASE
Normal
lumbar vertebra
a Periosteal apposition
Normal endosteum
b Periosteal apposition
Endosteal resorption
c Periosteal apposition
Endosteal apposition
d Focal Periosteal Apposition
(pumice-stone)

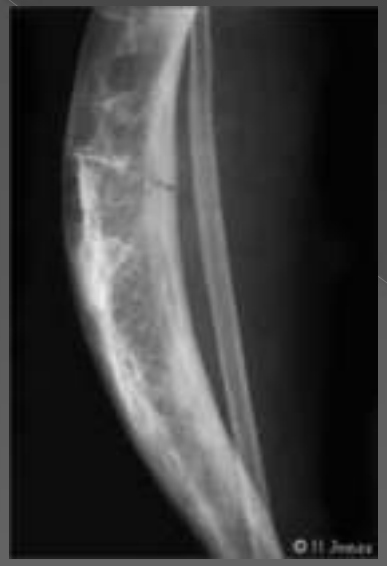

Normal
bones
Paget's Disease
of Bone

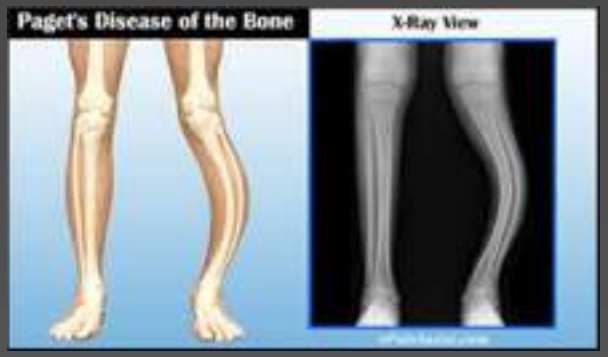
Oral manifestations

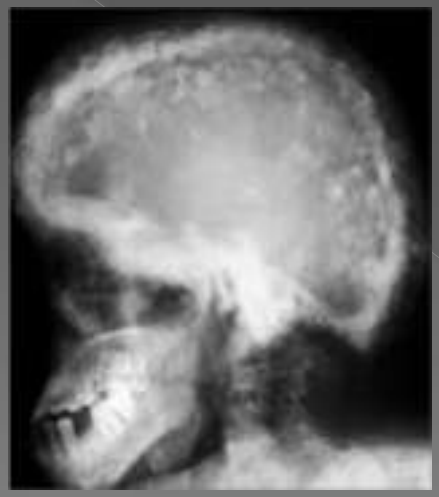
- Both jaws commonly affected
- Higher incidence in maxilla
- Migration of teeth common
- Widening of alveolar process and flattening of palate
- Denture wearing patients complain of ill fitting dentures
- As disease progresses mouth may remain open

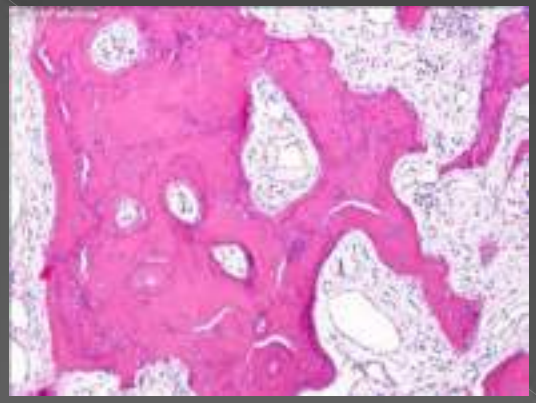
Radiographic features

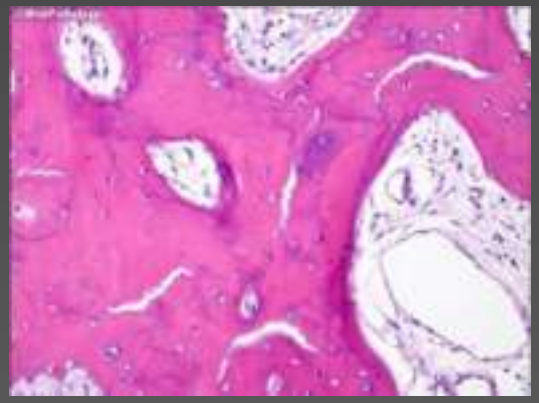
- Osteoclastic phase
- Phase of bone formation
- Osteoblastic phase
- Early phase shows lytic lesions
- Isolated lesions of skull called as osteoporosis circumscripta

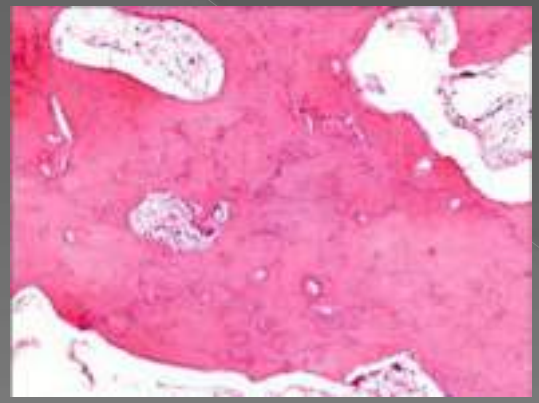



Paget's Disease of the Bone
X-Ray View







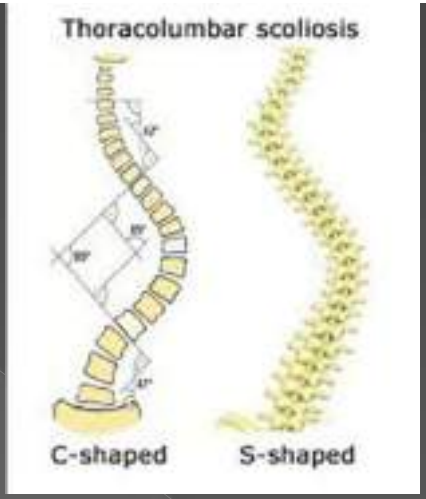


Lab findings

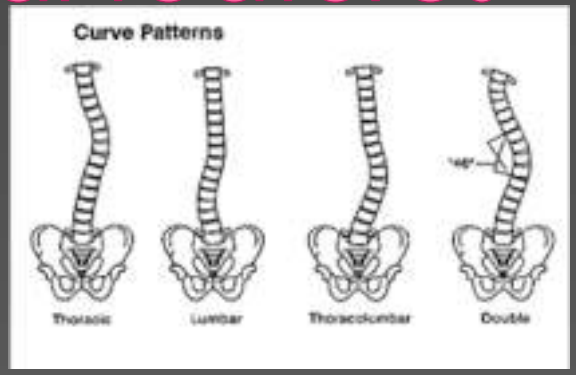
- Increased alkaline phosphatase(250 Bodansky units)
- Serum calcium, acid phosphatase and phosphorus normal
- Urinary hydroxy proline elevated-due to increased osteoclastic activity

Marfens syndrome

- Heritable genetic defect
- Autosomal dominant
- President Abraham Lincoln
- FBN1 gene, chromosome 15, band q15-q23
- Codes for connective tissue fibrillin
- Causes defective fibrillin production



Clinical features



- Arachnodactyly, dolichostenomelia(long limbs relative to trunk)
- Thoracolumbar scoliosis
- Shape of skull and face long and narrow
- Hyper extensibility of joints, habitual dislocations, kyphosis and flat feet
- Aortic dilation, aortic regurgitation,aneurysms
- Ocular findings-myopia, cataract, retinal

Oral manifestations

- High arched palatal vault
- Bifid uvula
- Multiple odontogenic cysts
- TMJ dysarthrosis

Treatment

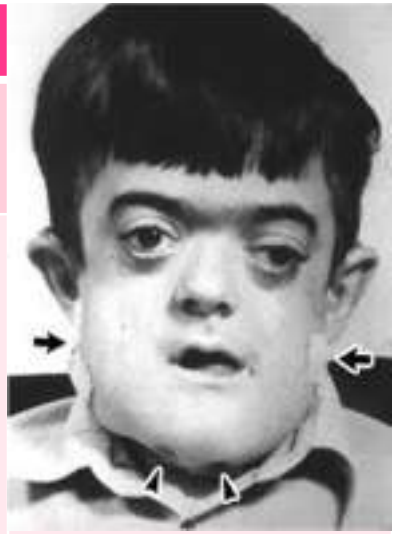
- none

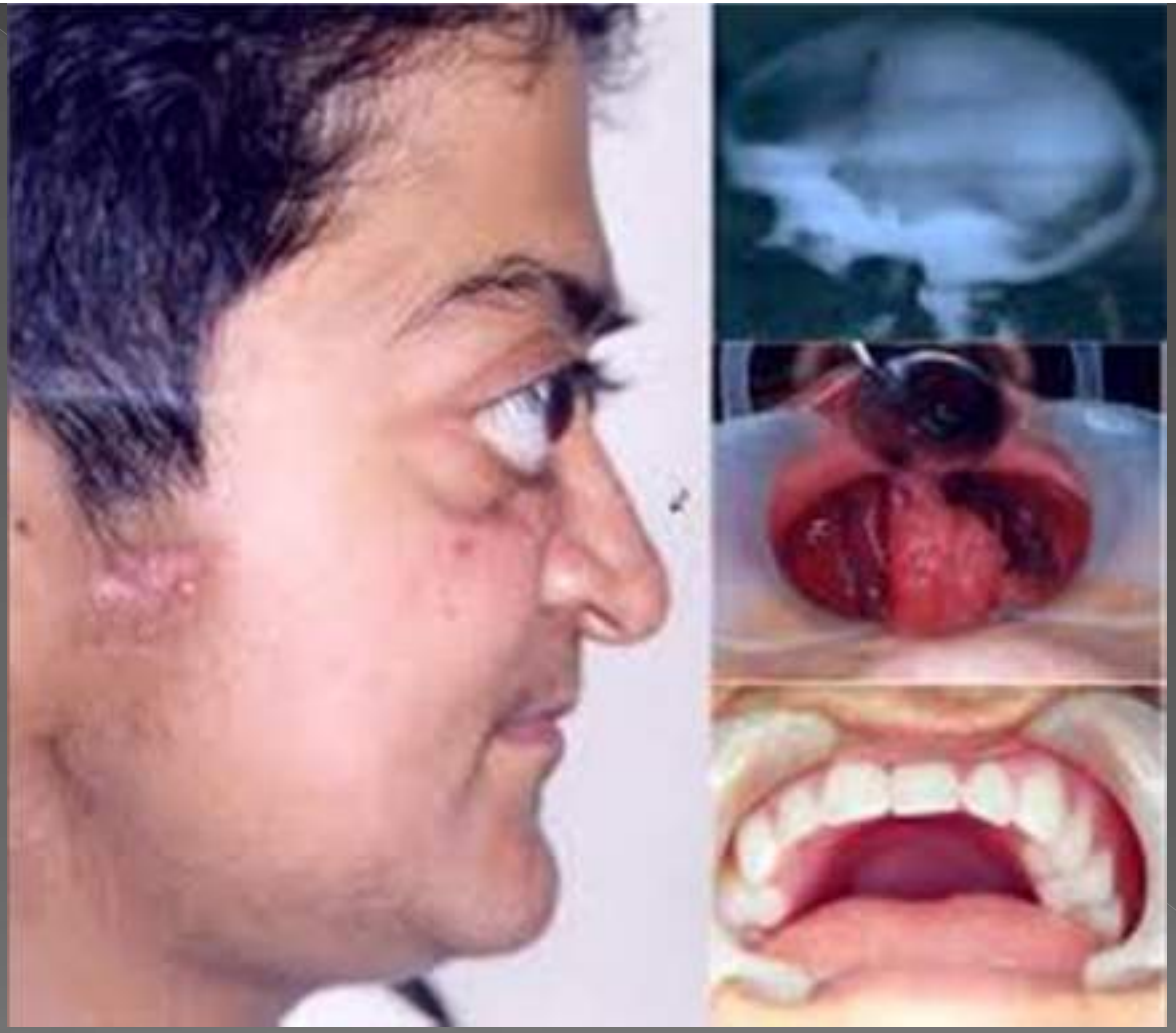
Ostepetrosis

- Marble bone disease
- Rare heriditary disorder
- Failure of osteoclastic activity leads to increase in bone mass
- Thickend sclerotic bones with poor mechanical properties
- Increased bone fragility

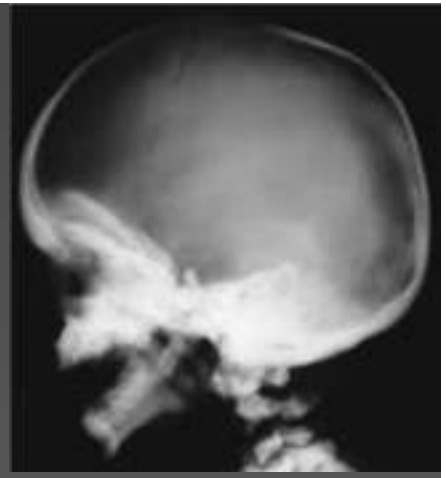
Clinical features

- 3 types: adult onset (OR), infantile (OD) and intermediate (OD)
- If untreated, infantile form results in death by first decade due to anemia, bleeding or infection

	Infantile	Intermediate	Adult	
Diagnosed	Early life		Late adolescence	
Symptoms	•Failure to survive & growth retardation •Nasal stuffiness due to mastoid and paranasal sinus malformation •Deafness •Proptosis, hydrocephalus		Asymptomatic, accidentally detected Short stature, frontal bossing, large head, hepatomegaly,	
Bone defects	+nt Cranial nerve entrapment - neuropathies		Common Bone pain, Carpal tunnel syndrome Osteoarthritis Cranial nerve entrapment - neuropathies	



X-ray features



- Gen osteosclerosis
- Bones appear club like
- Show bone within bone appearance
- Sinuses small and under pneumatized
- Vertebrae radio dense
- Bone may show alternating sclerotic and lucent band showing rugger jersey sign

Lab findings

- Anemia due to marrow displacement
- Hypocalcemia may cause rickets
- PTH increased
- Acid phosphatase and creatinine kinase (CK-BB) increased due to defective osteoclasts

Treatment

- Calcitriol stimulates dormant osteoclasts
- Erythropoietin to correct anemia
- Cortecosteroids to treat anemia
- Gamma interferon

Rickets

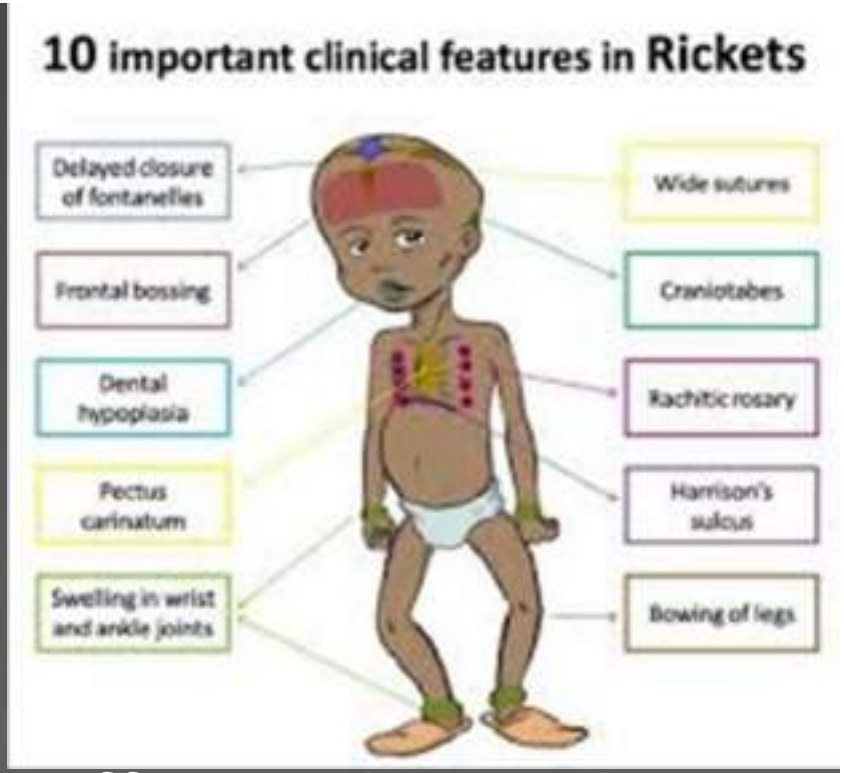
- Derived from word **wricken=to bend**
- Decreased mineralization at the level of bone plates resulting in growth retardation and delayed skeletal development
- Found only in children before epiphysis plate closure
- Osteomalacia found in adults

Etiology

- Deficiency or abnormal metabolism of Vitamin D
- Abnormal metabolism or excretion of inorganic phosphate
- Rachetic metaphysis-increased number of cells resulting in width and thickness of hypertrophic zone

Findings

- Reaction of periostium
- Indistinct cortex
- Coarse trabaculation
- Knees, wrist and ankles affected
- Epiphyseal plate widened and irregular
- Increased metaphysis



Vitamin D resistant rickets

- Refractory rickets
- Phosphate diabetes
- Low serum phosphate
- Vitamin D produces no effects
- Mutation in PEX gene-Xlinked dominant form- located on Xp22.1 locus, autosomal dominant form-12p13 chromosome

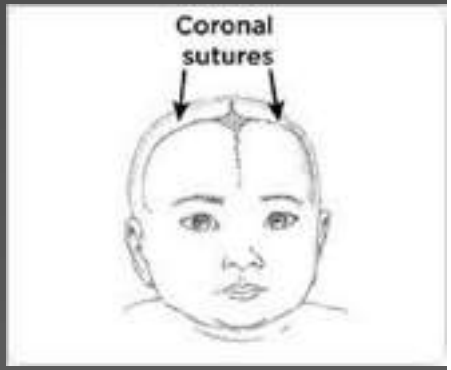
Clinical features

- Present since conception
- Infants born with normal weight but show growth retardation
- Serum phosphate levels decreased
- Widened joint spaces, flaring of knees
- When child walks , bending of weight bearing bones seen
- Dentition absent or eruption delayed

Lab findings

- Calcium below or slightly below normal
- Alkaline phosphatase raised
- Serum parathyroid levels normal, calcitrol levels low
- Urinary loss of phosphate increased

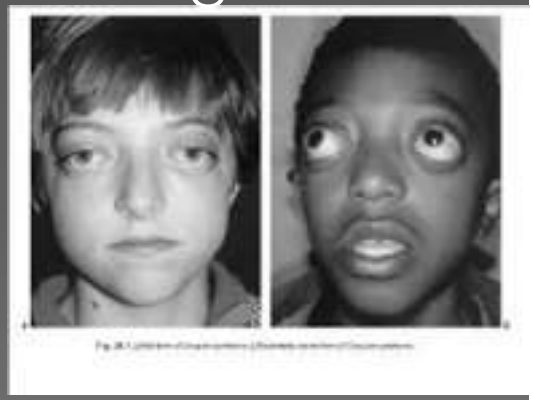
Craniofacial dysostosis



- Crouzon disease or syndrome
- Type of craniosynostosis syndrome
- Premature craniosynostosis
- Crouzon's diseases is most common craniosynostosis syndrome without Syndactyly,
- Apert's syndrome is most common craniosynostosis syndrome with Syndactyly
- anomalies of hands and feet may or may not be seen
- Occurs due to premature obliteration of sutures especially coronal and saggital

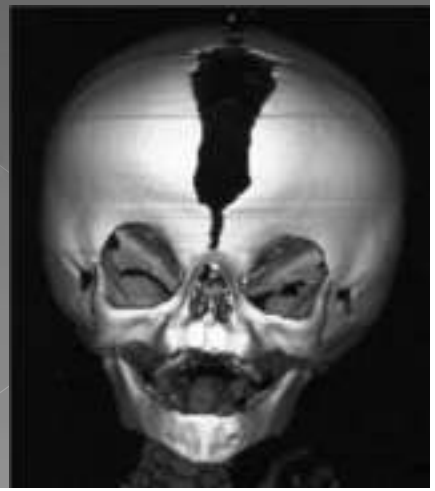
Etiology & Clinical Features

- Mutation in FGFR-2 gene
- Facial deformity observed at birth
- Fontanels remain open and pulsating for a long time
- AP diameter smaller than transverse
- Forehead high and wide
- Wide face and pseudoprognathism
- Deviation of nasal septum
- Narrow or obliterated nares
- Wide beaked nose
- Hypertelorism, divergent squint , upper eyelid frog face
- Upper lip short and cleaved
- Progressive optic nerve atrophy leads to blindness



•Malocclusion, malposed teeth and dysphagia

patients report headache, convulsions and mental retardation seen



Xray

- Radiographs necessary to confirm diagnosis
- Skull x ray reveals obliterated sutures, shallow eye sockets,/ exopthalmos
- Under developed lateral nasal sinus
- Spine shows bifid spine

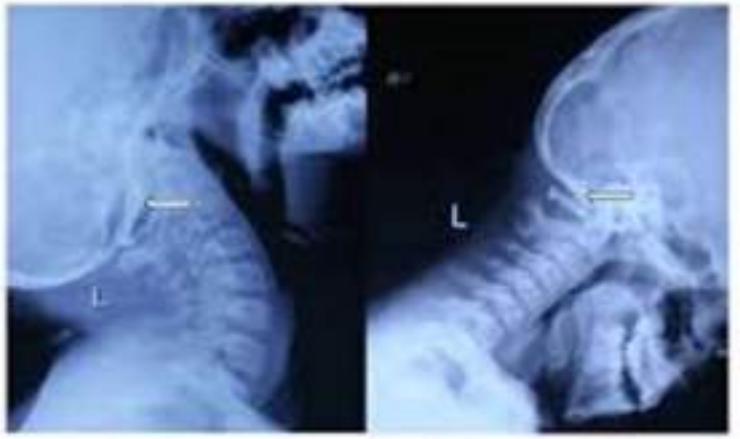


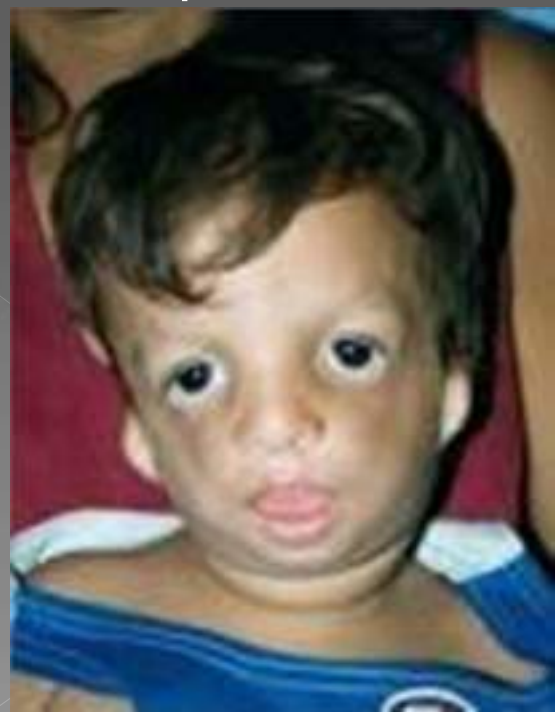
Figure 6: Lateral extension and flexion radiograph of the cervical spine demonstrating asymmetric partial assimilation of the atlas into the occiput.

Treatment

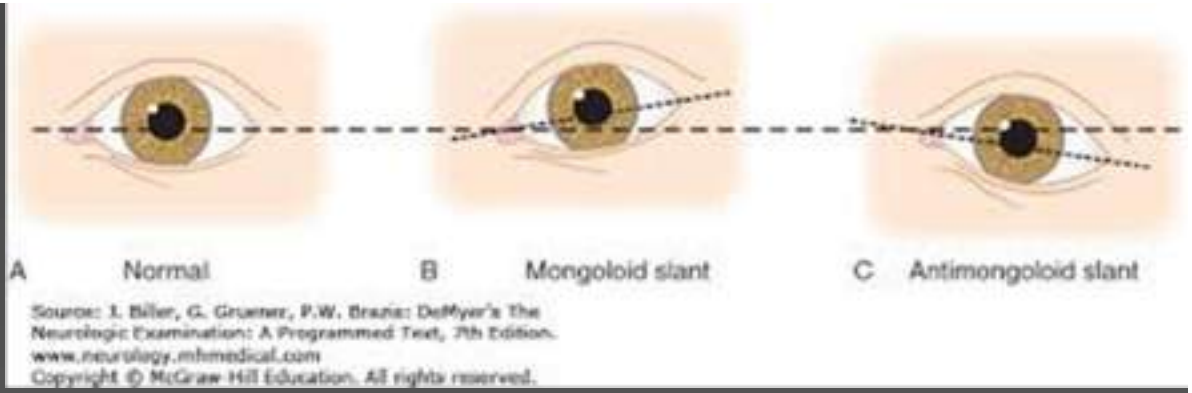
- Surgery to decrease intracranial pressure
- Plastic surgery for facial deformity

Mandibulofacial dysostosis

- Teacher collinf-s-Franceschetti syndrome
- Heriditary, dominant charecteristic
- Gene mapped to chromosome 5q32-q33.1



Clinical features



- Antimongoloid palpebral fissures
- deficiency of eyelashes /sometimes eyelids
- Hypoplasia of facial bones, especially malar bones and mandible
- Malformation of external ears, sometimes middle and internal ears
- Macrostomia, high arched palate, malocclusion

Clinical features

- Blind fistulas between angle of ears and angle of mouth
- Atypical hairgrowth in shape of tongue shaped processes of hairline extending towards cheeks
- Other anomalies such as facial clefts and skeletal deformities
- Bird like of or fish like faces

Xray features

- Para nasal sinuses underdeveloped
- Auditory ossicles underdeveloped
- Hypogenesis or agenesis of condyle

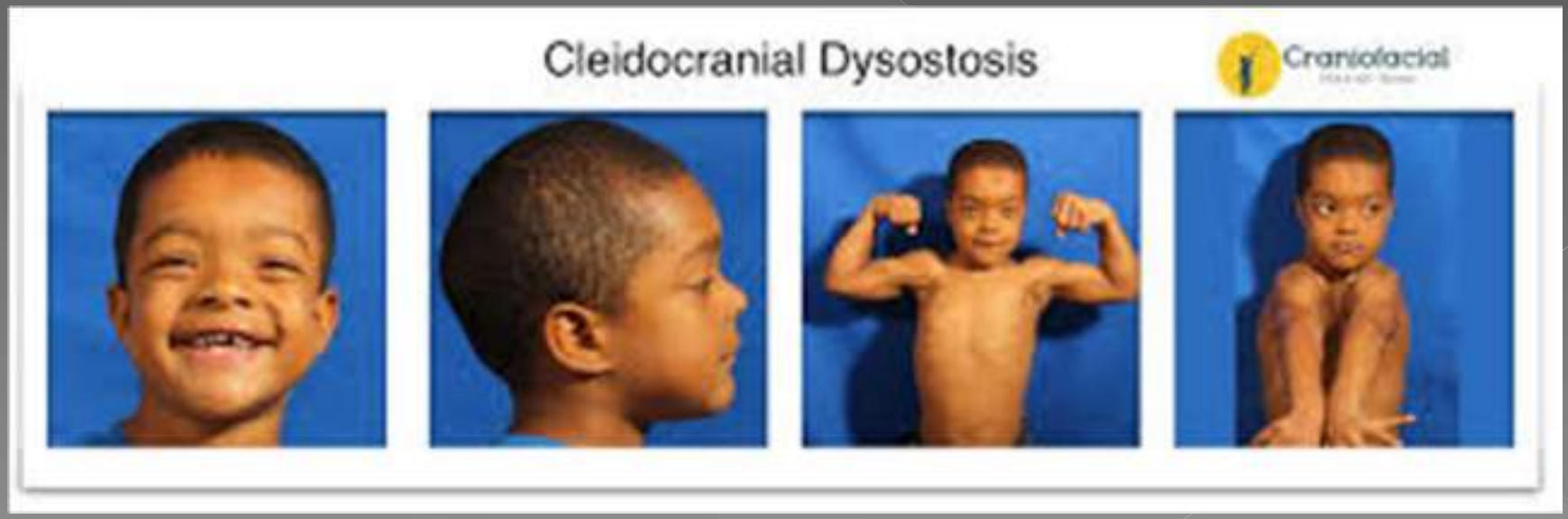
Treatment:

- None
- But patients have a good life span

Cledocranial dysostosis

- Congenital disorder
- Clavicular hypoplasia or agenesis with narrow thorax=allows apposition of shoulders
- Delayed ossification of skull, large frontanells, delayed closure of sutures
- Womarian bones
- Bossing of frontal, parietal and occipital bones

- Arnold head-large globular head with small face
- Syndrome transmitted as Autosomal dominant trait



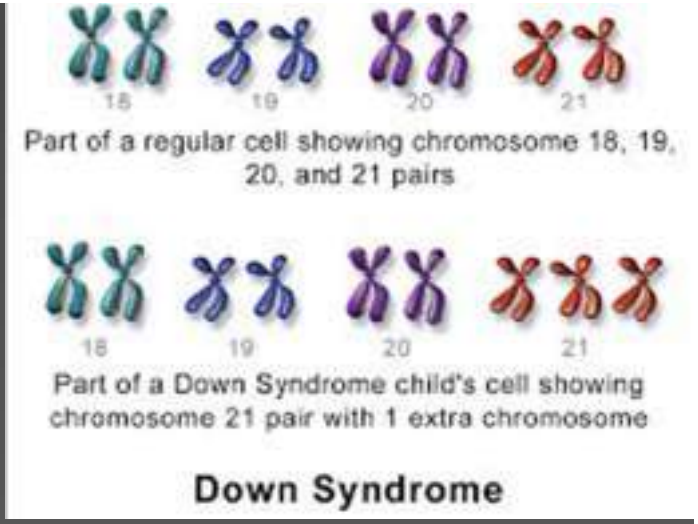
Oral manifestations

- High narrow, arched palate, cleft palate
- Absence or reduced cellular Cementum on roots of permanent teeth
- Numerous un-erupted teeth

Treatment

- No specific treatment

Downs syndrome



- Trisomy 21
- Three types of downs syndrome
- 1-typical trisomy 21 with 47 chromosomes
- 2- translocation type-46 chromosomes with extra material of 21st chromosome added to 14 chromosome or other chromosomes.
- 3- chromosomal mosaicism-different cells in an individual have different number or arrangement of chromosomes

Clinical features

- Mental retardation(IQ-25-50)
- Small head-brachycephaly
- Flat face with increased inter-ocular distance
- Depressed nasal bridge, flat occiput, broad an short neck
- Narrow, outward slanting palpabrel fissures
- Misshapen ears
- Short stature, broad and short hands, feets and digits
- Protuberent abdomen
- Congenital defects in heart

Oral manifestations

- Small mouth with protrusion of tongue
- Difficulty in eating speech, scrotal tongue, Hypoplasia of maxilla, delayed tooth eruption, partial anodontia, enamel Hypoplasia, cleft lip or palate
- Fissuring and thickening of lips with angular chelitis

Treatment:

none

Infantile cortical hyperostosis

- Caffey's disease
- Self limiting disorder affecting infants and causing bone changes, soft tissue swelling and irritability

Pathogenesis

- Inflammation of periostium and adjacent soft tissues
- Periostium remains thickened and subperiosteal immature lamellar bone
- Hyperplasia of lamellar cortical bone

Types

Sporadic

- Higher incidence in males
-

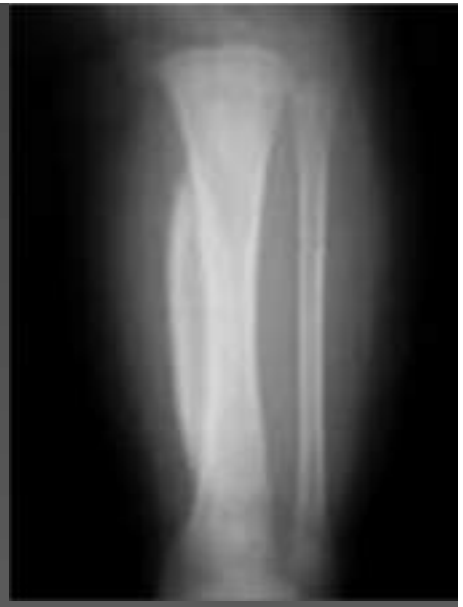
Famillial

- Earlier onset
- Male involvement rare
- Involvement of lower extremity common
- Tibia most common

Clinical features

- Triad- irritability+ swelling+bone lesions
- Swelling appears suddenly, is deep seated and tender
- Mandible and clavicle most commonly affected
- Mostly affecting ramus and angle
- Other signs- pseudoparalysis, pleurisy, anemia, leukocytosis, increaed ESR, serum alkaline phosphatase

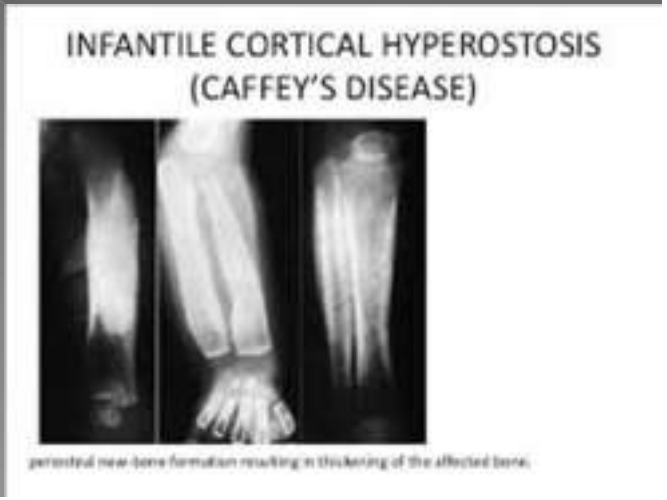
Xray



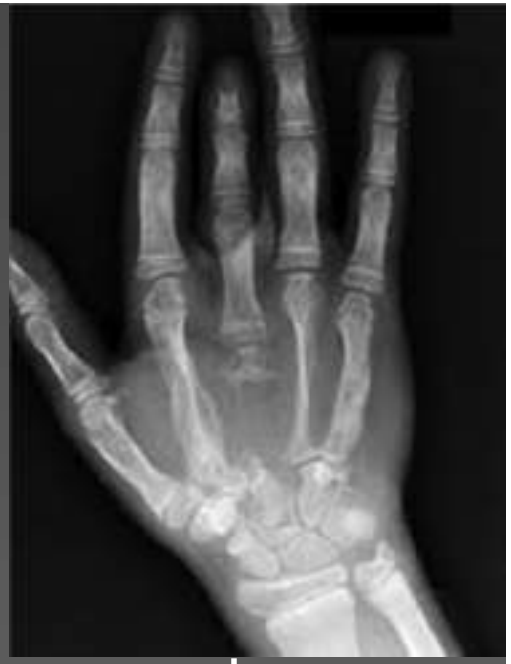
- Periosteal new bone formation, producing cortical thickening

Treatment:

- None
- Corticosteroids to alleviate pain
- NSAIDS



Massive osteolysis



- Vanishing bone disease
- Gorham syndrome
- Characterized by dissolution of a part or whole bone
- Cavernous angioma like permeation may be a prominent pathological feature of affected bone
- Clavicle, scapula, humerus, ribs, ilium and sacrum

Histology

- Bone replaced by connective tissue containing thin walled vessels or anastomosing vascular spaces line by endothelial cells
- Absence of osteoclasts

Treatment:

- No specific
- Radiation therapy helps

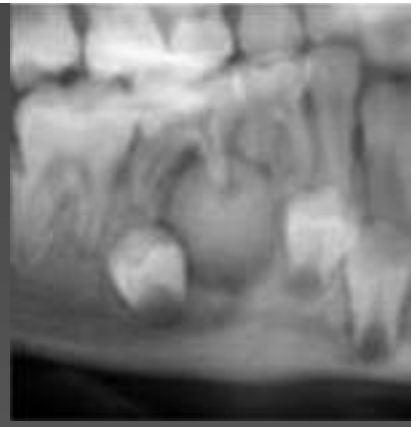
Cementoblastoma

- True cementoma
- True neoplasm of functional cementoblasts which form a large mass of cementum like tissue on tooth root
- Relatively uncommon

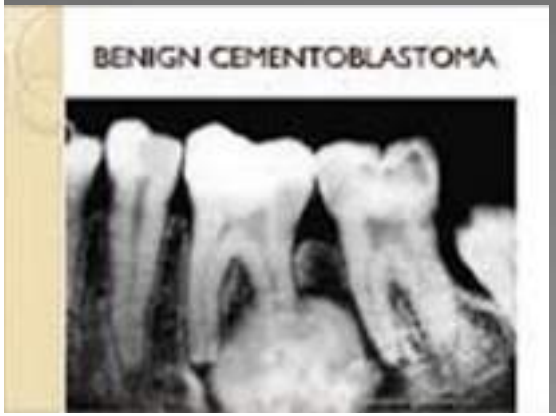
Clinical features

- Below 25 years
- No gender predilection
- Mandible affected 3 times more than maxilla
- Mandibular first permanent molar most affected tooth
- Slow growing lesion causing expansion of cortical plates of bone
- Asymptomatic
-

Xray features

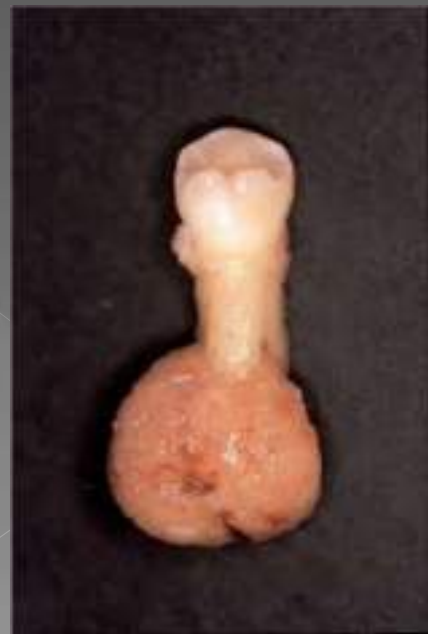


- Tumor mass attached to tooth root
- Appears as a well circumscribed dense radio-opaque mass surrounded by a thin radiolucent rim
- Outline of affected root obliterated due to resorption and fusion of mass to tooth



Histology

- Sheets of Cementum, sometimes in a globular pattern resembling giant cementicles
- Reversal lines can be seen through the tissue

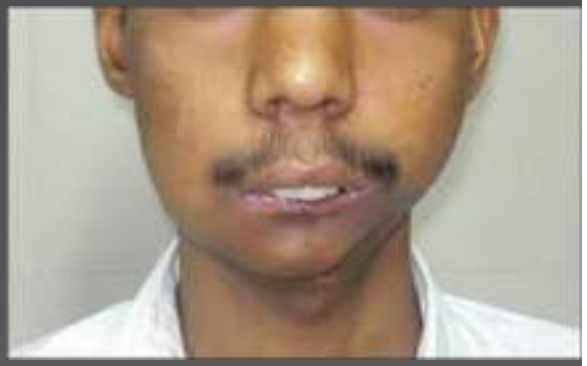


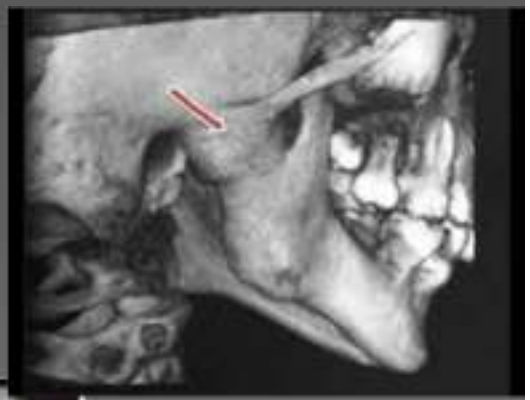
Developmental disturbances of TMJ

- Aplasia
- Hypoplasia
- Hyperplasia

Traumatic disturbances of TMJ

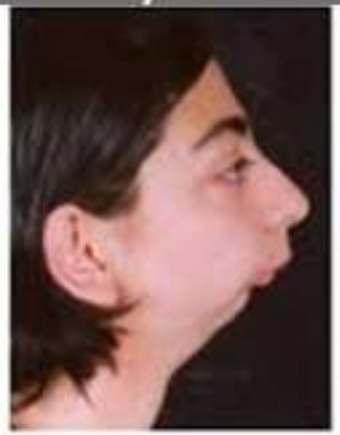
- Luxation & subluxation
- Ankylosis
- Injuries to articular disk/ meniscus
- Fracture of condyle





TMJ Subluxation

a



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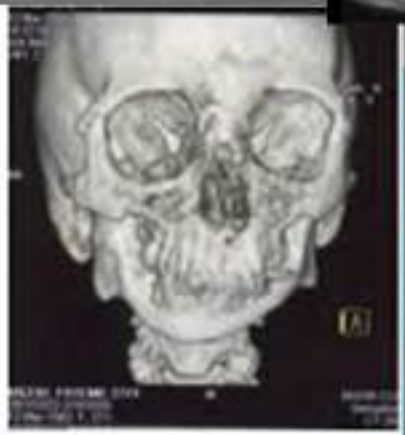

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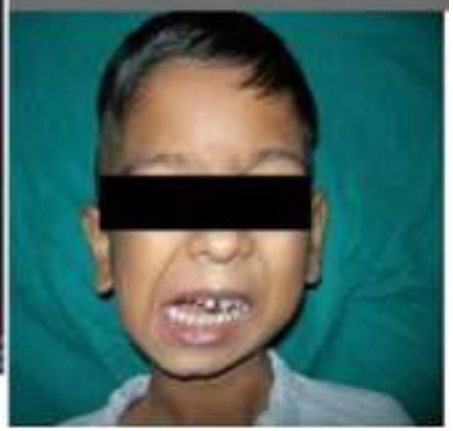
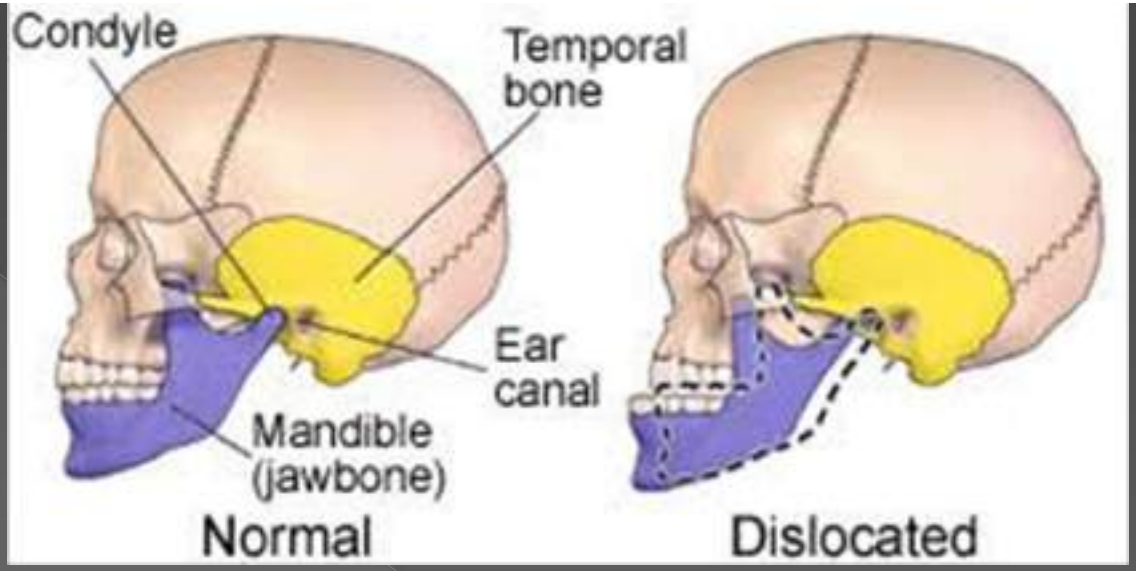
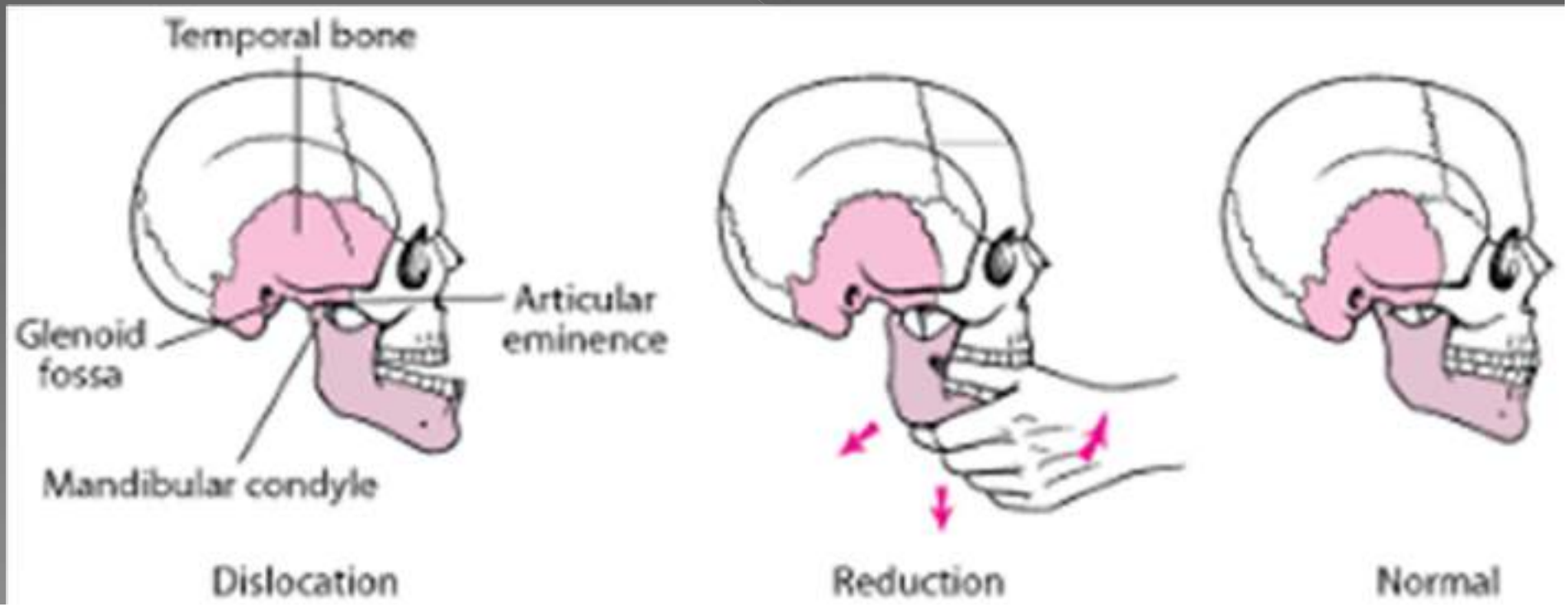



Condyle
Temporal bone
Ear canal
Mandible (jawbone)
Normal
Dislocated


Temporal bone
Glenoid fossa
Articular eminence
Mandibular condyle
Dislocation
Reduction
Normal

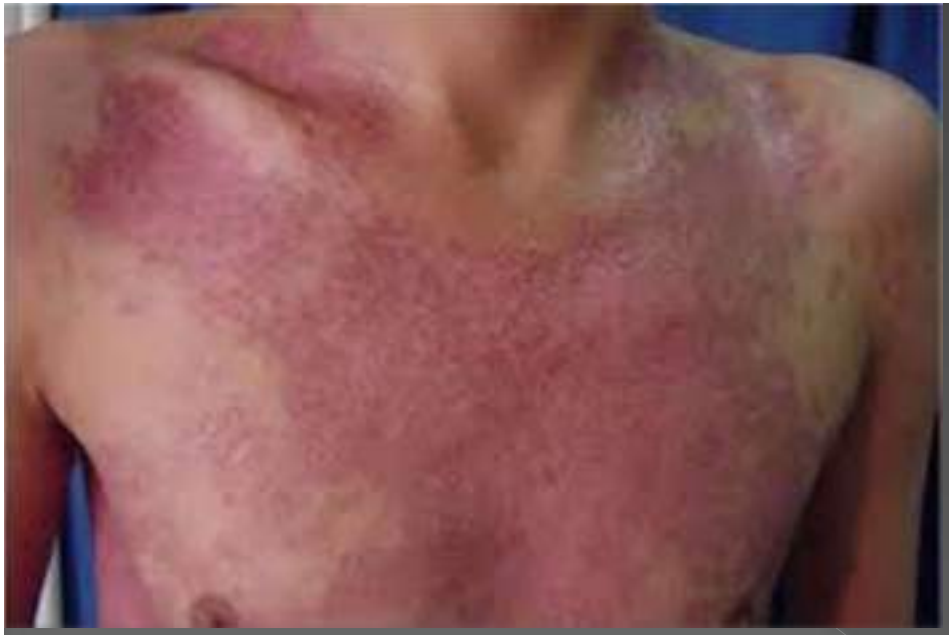
Inflammatory disturbances of TMJ

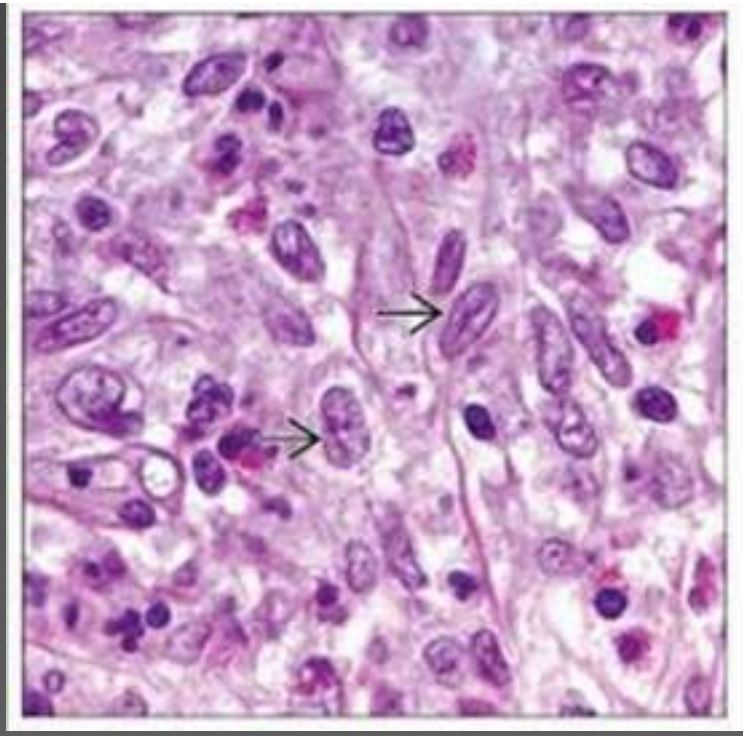
- Arthritis
- Osteoarthritis

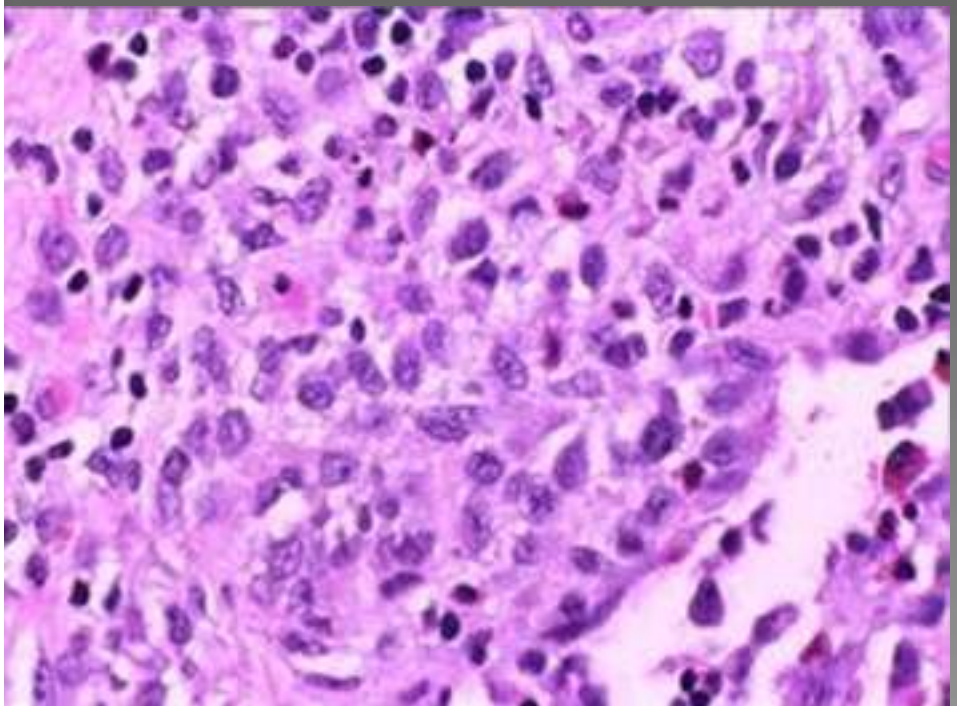
- **Histiocytosis**: Rare spectrum of disorders characterized by proliferation and accumulation of histiocyte in various lesions within the body.
- Lesions may include Langerhans cells, monocytes, and eosinophils.

Langerhan cell histiocytosis

- Affects bone and other system
- Presence of birbeck granules, CD1a antigen on the cell surface, HLA-DR positivity-confirms langerhan cell origin
- Probably a reaction to viral infection
- Includes 3 distinct clinical entity
- 1. letterer- Siwe disease(acute fulminant dessiminated disease involving skeletal and extraskeletal tissues)
- 2. eosinophillic granuloma(solitary, few indolent chronic lesions of bone)
- 3. Hand –Schuller christian disease(intermediate affects only skeletal

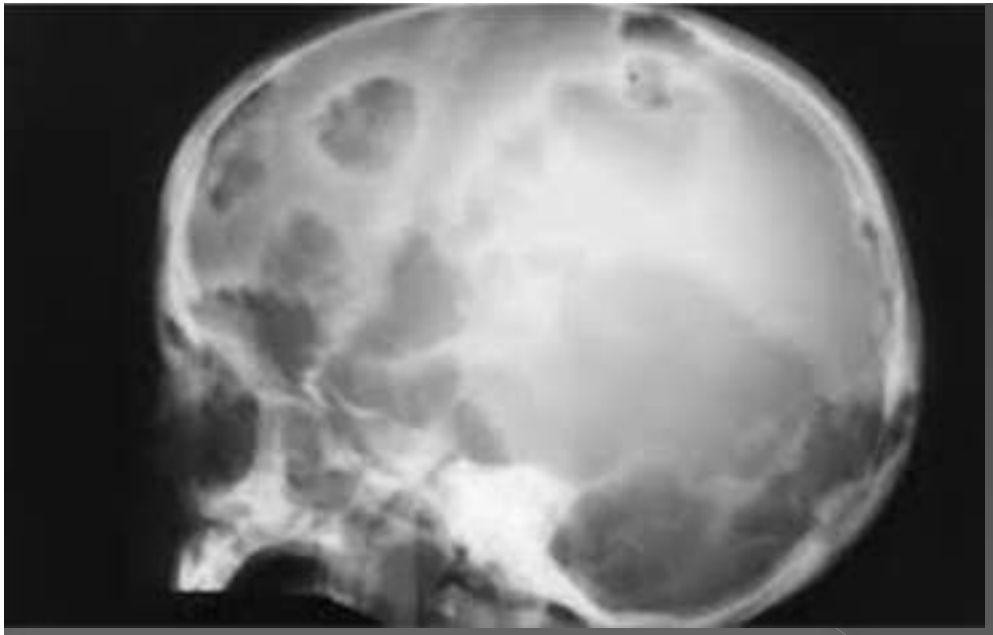


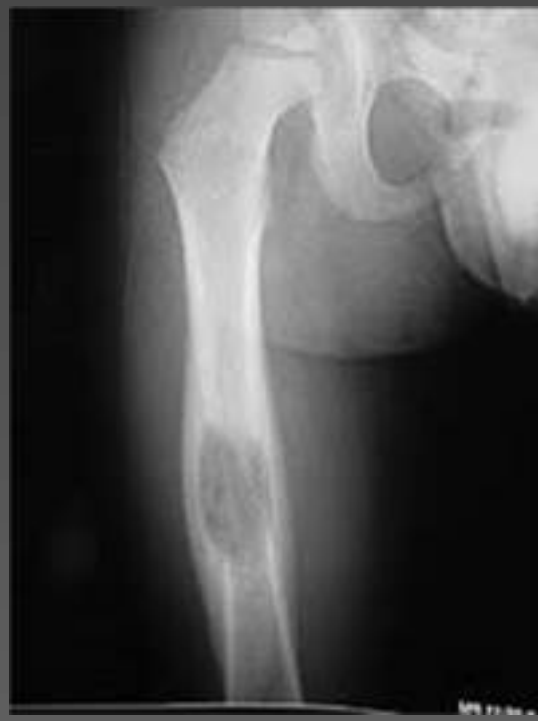


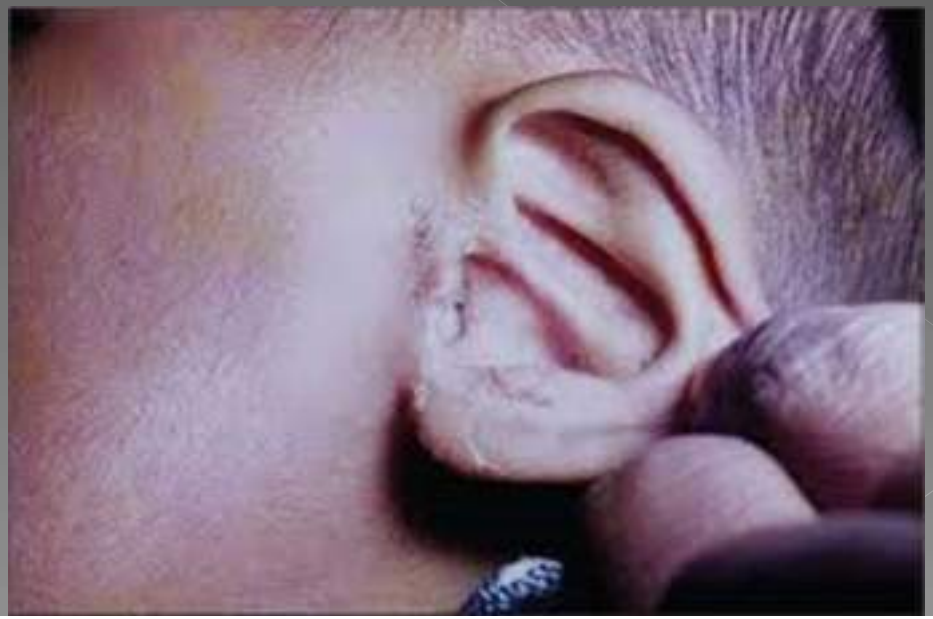


Hand –Schuller –Christian Disease

- Characterized by skeletal and extra skeletal lesions
- Chronic clinical course
- Occurs usually before 5yrs
- Triad-single/multiple punched out lesions of skull+unilateral/ bilateral exopthalmos+diabetes insipidus (with /without polyuria, dwarfism or infanti)







Oral manifestations

- Mouth sores, ulcerative lesions, halitosis, gingivitis, suppuration, unpleasant taste
- Loose and sore teeth with precocious exfoliation, failuer of healing of tooth sockets after extraction
- Loss of supporting alveolar bone – resembling advanced periodontal disease

Xray-

- Skull lesions sharply outlined
- Jaw lesions diffuse

Histology

- Four stages:
- 1. proliferative histiocytic phase with accumlation of collection of eosinophills with histiocytes
- 2. vascular granulomatous phase with histiocytes and eosinophills, sometimes lipid laden macrophages
- 3. diffuse xanthomatous phase with abundant foam cells
- 4.fibrous healing phase

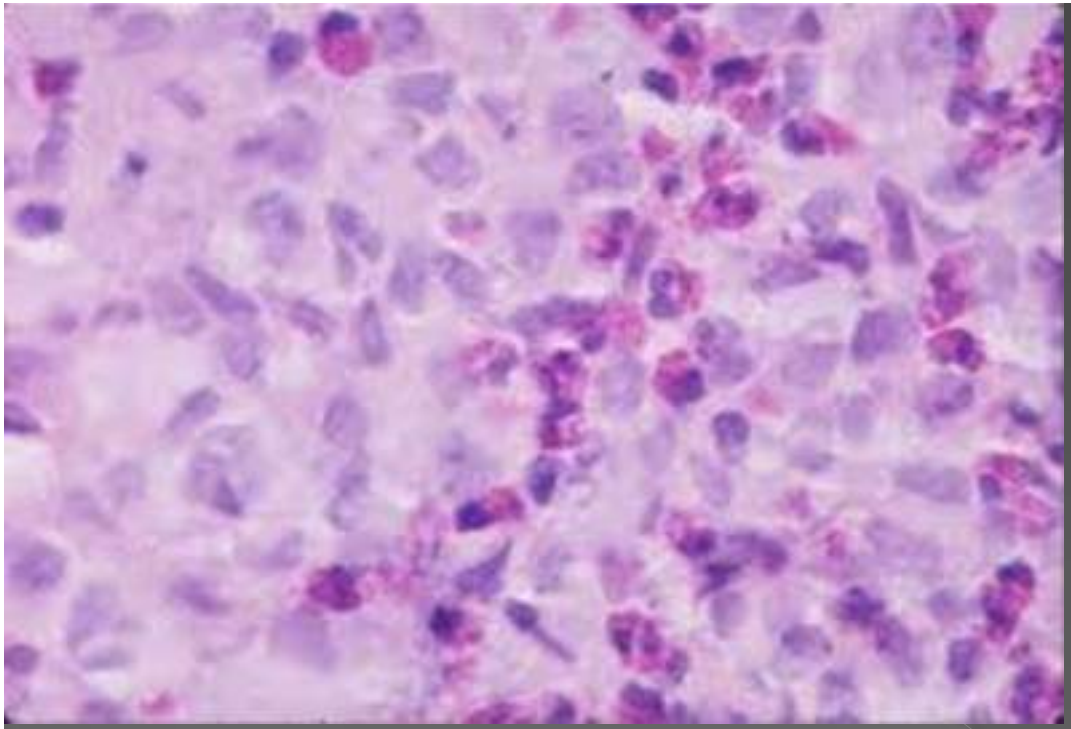
Lab findings

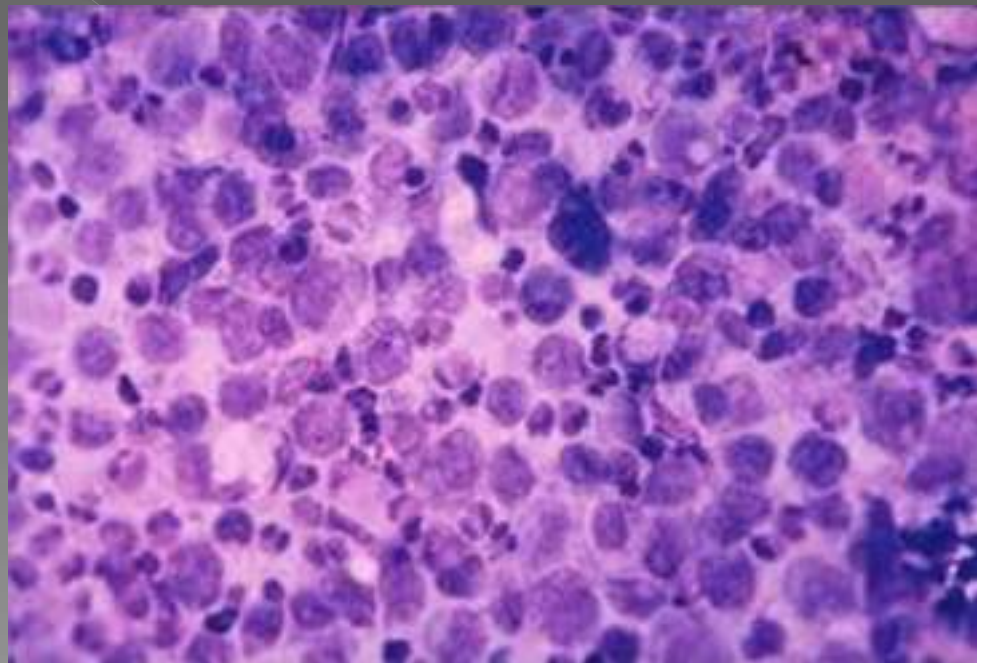
- Anemia
- Leukopenia
- Thrombocytopenia
- Serum cholesterol normal, tissue cholesterol raised
- Prognosis good- half patients undergo spontaneous remission over years
- Curettage / excision

Eosinophillic granuloma

- Introduced by Jaffe and Lichtenstein
- Histiocytic proliferation with abundant eosinophills, no intracellular lipid accumlation
- Sometimes found incidentally, other times associated with pain , swelling and tenderness
- Lesions are destructive, well demarcated and roughly oval in shape
- Area destroyed is replaced by soft tissue

- Xray-irregular radiolucent area involving superficial bone
- Cortex is destroyed, pathological fractures
- Histologically primary cell is histiocyte growing in sheet like collections
- On maturation number of eosinophills reduce or may disappear





- Prognosis excellent
- Curettage along with radiotherapy

Letterer–Siwe disease

- approximately 10% of **LCH** disease
- most severe form.
- Prevalence is estimated at 1:500,000
- disease almost exclusively occurs in children less than three years old.

- Physiologically, Langerhans cells detect non-self-antigens and present them to the cells of the immune system (T cells), thus allowing an appropriate immune response from the body.
- LC are normally found in the epidermis of the skin, but in LSD they spread to bone and other tissues and become associated with eosinophils.
- Lesions arise in many organs, including bone, skin, spleen, liver, lungs, lymph nodes, and brain.
- Granulomatous inflammatory lesions develop and may proliferate and become destructive.
- These lesions later become less cellular, necrotic, and fibrotic

- The hallmark of LSD is the presence of pathologic Langerhans cells in involved tissues.
- Specific histochemical, immunologic, and protein markers have been identified (Birbeck granules or positive S-100 beta protein and CD Ia antigen).
- These biochemical findings in addition to multiorgan involvement help make the diagnosis.

Clinical Features

- Predominantly affecting children age 2 months to 3 years.
- Clinical features include fever, anemia, thrombocytopenia
- manifestations of histiocyte proliferation, including skin disorders (seborrheic, eczematous, pustular or nodular lesions particularly on the scalp)
- lytic lesions of the bones, and splenogenic thrombocytopenia.
- The lungs can be involved (nonproductive cough, dyspnea, pleural effusion, interstitial pneumonitis, and spontaneous pneumothorax).
- Hypothalamic involvement resulting in diabetes insipidus (DI).
- Eye protrusion may be present. The clinical course is very variable, and spontaneous remissions have occurred.
- Prognosis is generally poor.
- Treatment may include use of glucocorticoids, chemotherapeutic agents, bone marrow transplantation

Cemento-osseous dysplasia

- Occur in tooth bearing area
- Excessive deposition of cementum like material
- Commonest fibro-osseous lesion in terms of frequency of occurrence

pathogenesis

- Due to the close proximity of lesions to the teeth and production of cementum like material some consider PDL to be responsible for it
- Defect in extra-ligamentary bone remodelling influenced by local and systemic factors

- Three types:
- Focal cemento- osseous dysplasia
- Periapical cemento- osseous dysplasia
- Florid cemento- osseous dysplasia

Take home message

- bone disorders include a wide variety of disorders ranging from genetic to acquired
- Identification of any bone related pathology must be done based on clinical, radiological and histological findings