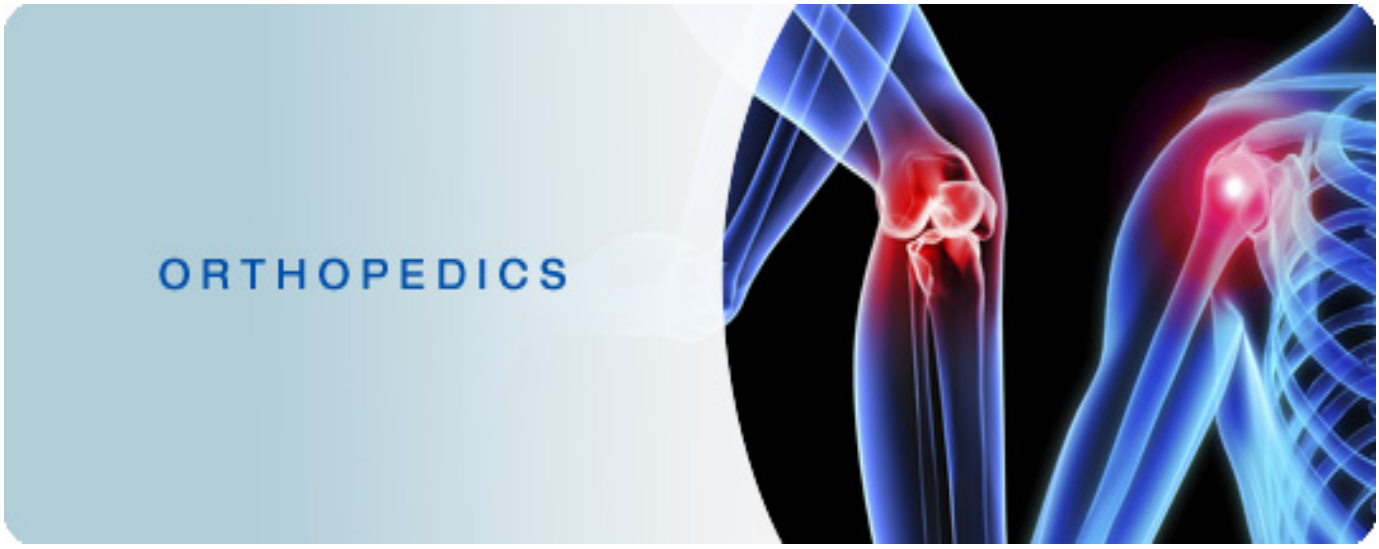
Isn't it funny how someone can say "I believe in Allah " but still follow the Satan who by the way also, " believes " in Allah…

430 ORTHOPEDICS TEAM



Lecture: Metabolic Bone Disorders

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Team Leader:

Ayedah Al-Ruhaimi.

-The slides were provided by the doctor.

-Important notes in **Red**

-Copied slides in **Black**.

-Doctor's notes in **green**.

Orthopedic Surgeons and Bone:

- Orthopedic surgeons have to deal with all types of bone: healthy or diseased; and that's why they have to know about bone metabolism.
- Bones in the body protect vital organs eg: skull protects brain, ribs protect lungs and heart.
- Bones give support to muscles and tendons.
- Bone may become weak in certain conditions.

Bone is a living structure:

- There is a continuous activity in bone during all stages of life
- There is continuous bone resorption and bone formation as well as remodeling.
  While osteoblasts are forming a new bone, osteoclasts are removing the dead aged ones. This process accelerates with aging and when estrogen levels drop (menopause) with decreased rate of formation and increased rate of bone loss. Opposite to that is happening in childhood where bone is formed in higher rates than loss, the age period from 18-21 years is the period of equilibrium.
- That means bone is not only for protection and support but its contents play an important part in blood homeostasis
- Many factors are involved in this process

Bone Metabolism:

Bone metabolism is controlled by many factors:

- Calcium
- Phosphorus
  regulated by:
- Parathyroid gland
- Thyroid gland

- Estrogen
- Glucocorticoid hormones
- Intestinal absorption
- Renal excretion
  Patients with malabsorption condition like celiac disease or sprue or having a kidney disease like tubular necrosis or glomerular nephropathy will not

benefit from the dietary or supplemental calcium and vitamin D intake till this condition is corrected.

- Diet
- Vitamin D
- Sun exposure
- Exercise

Bone Structure:

Bone is formed by

- Bone matrix : which consists of
  - 40% organic : collagen type1 (responsible for tensile strength)
  - 60% Minerals: mainly Calcium hydroxyapatite, Phosphorus, and traces of other minerals like zinc
- Cells in bone : osteoblasts, osteoclasts, osteocytes

Plasma levels:

- Calcium : 2.2-2.6 mmol/l
- Phosphorus : 0.9-1.3 mmol/l
  you should remember those values only if you are intern or resident
  Both absorbed by intestine and secreted by kidney in urine
- Alkaline phosphatase : 30-180 units/l shows the activity of bone metabolism
  Is elevated in bone increased activity like during growth or in metabolic bone disease or destruction
  in adult it should not exceed 180 units/l, but in children it is considered normal up to 400 units/l because of growth
- Vitamin D level : 70-75 nmol/l
  this value is controversial, because here in Saudi Arabia all women are less than 75, they still don't know if it is normal to have less than that in our population or that all Saudi females are truly vitamin D deficient. Most probably it shouldn't be less than 75 even in our population :/
  Vitamin D is not only important for human skeleton, it also gives immunity against cancer and several diseases. Recently they discovered that all Alzheimer patients are vitamin D deficient.

Parathyroid Hormone (PTH):

Production levels are related to serum calcium levels:

- It increases calcium levels in the blood by increasing its release from bone, this is a fast action that increases ca levels in blood secondary to disease as a compensatory mechanism.
- increase absorption from the intestine

- and increase reabsorption from the kidney (also increase secretion of phosphorus)

Hyperparathyroidism:

This part and the last part of the lecture which also about hyperparathyroidism are not included in the syllabus and you are not going to ask about, it is only to help you in DDx.

- Primary: Adenoma of the gland very rarely carcinoma.
- Secondary: as a result of low calcium "Chronic" malabsorption, low calcium diet, kidney excrete more Ca .
- Tertiary: as a result of prolonged or sustained stimulation = hyperactive nodule or hyperplasia

Calcitonin:

- Is secreted by C cells of thyroid gland
- Its secretion is regulated by serum calcium
- Its action is to cause inhibition of bone resorption and increasing calcium excretion by this it causes lowering of serum calcium

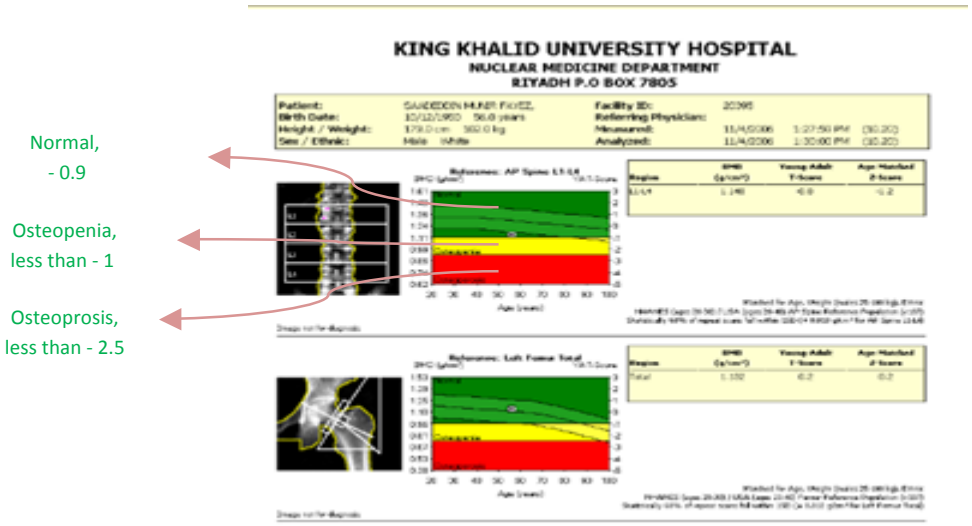
Bone Strength:

- Bone strength is affected by mechanical stress which means exercise and weight bearing, those who were exercising since childhood or farmers have very strength bone.
- Bone strength gets reduced with menopause and advancing age
- Reduced bone density on X rays is called Osteopenia
- Osteopenia is also a term used to describe a degree of reduced bone density, which if advanced becomes Osteoporosis

Bone Density:

- Bone density is diagnosed at current time by a test done at radiology department called : DEXA scan which tests 3 areas: vertebrae, hip and wrists.
- DEXA is (Dual Energy X ray Absorbtionometry)
- Increased bone density does not always mean increased bone strength, as sometimes what is called Brittle bone which is a dense bone is not a strong bone but fragile bone which may break easily

Dexa Scan: for example, if bone is dense and we send 100 rays, 20 will go back and thin means that 80 are absorebed. But if bone is osteopenic 50 will go back so only half is absorbed which means less density.



Disorders to be discussed:

- Rickets
- Osteomalacia
- Osteoporosis
- Hyperparathyroidism (not included)

Rickets & Osteomalacia:

Different expressions of the same disease

➔ Inadequate **mineralization**

- Rickets: Affects areas of endochondral growth in children (wrist, knee, shoulder, hip)
  We can diagnose rickets by looking at knees (inspection) or wrist x-ray
- Osteomalacia: Affects all skeleton is incompletely calcified in adults

Causes

- Calcium deficiency
- Hypophosphataemia
- Defect in Vitamin D metabolism
  - Nutritional (poor, war zoon, …)
  - underexposure to sunlight
  - intestinal malabsorption (ex. Celiac disease)
  - liver & kidney diseases, (renal tubular acidosis , Glomerular nephropathy) collect 24 hour urine Ca (normal : 500 mg of Ca)

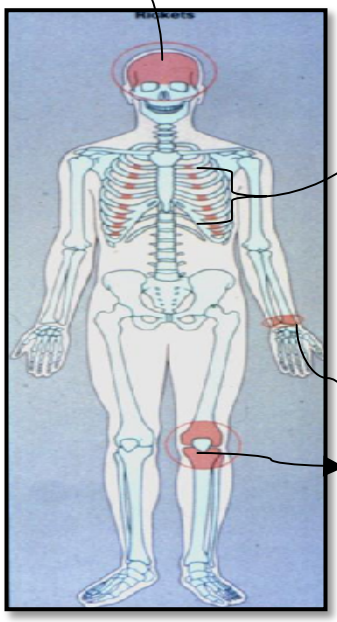
Rickets: Symptoms and Signs:

Knees are the first one to be affected then wrists then costochondral

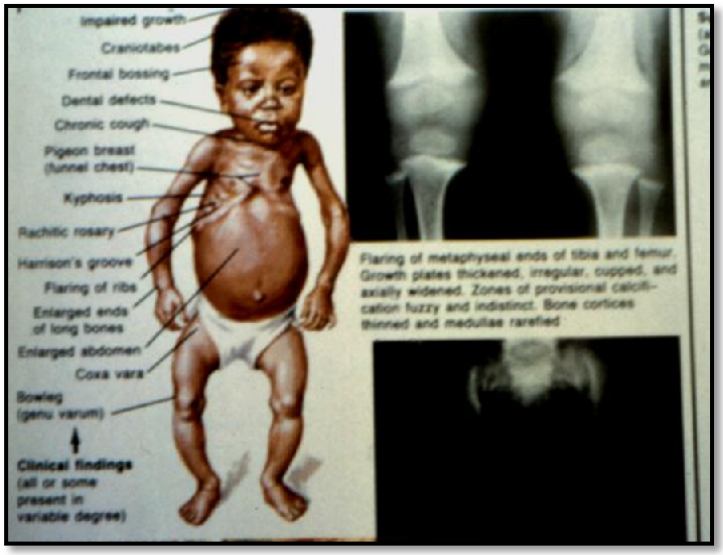
- Child is restless, babies cry without obvious reason
- Failure to thrive
- Muscle weakness
- In severe cases with very low calcium: tetany or convulsions
- Joint thickening (hypertrophy) especially around wrists and knees
- Deformity of limbs, mostly Genu varum (bowl legs) or Genu Valgum in late presentation
- Pigeon chest deformity, Rickety Rosary (beads like), craniotabes

Skull enlarge =Delayed sutures closure

Between costochondral junction in rib cage



Chondral hypertrophy especially in growing bone

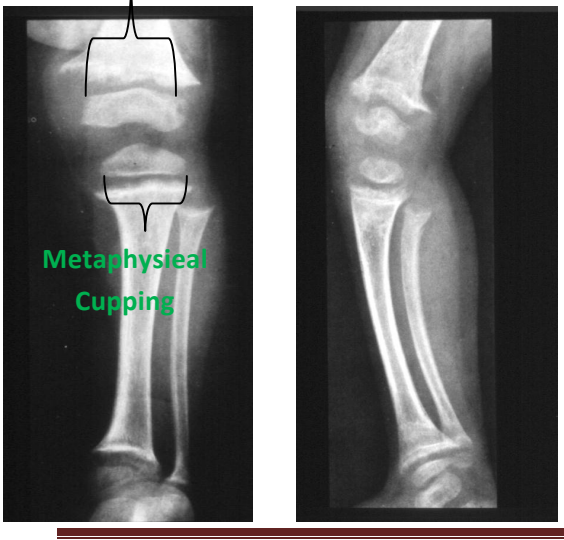


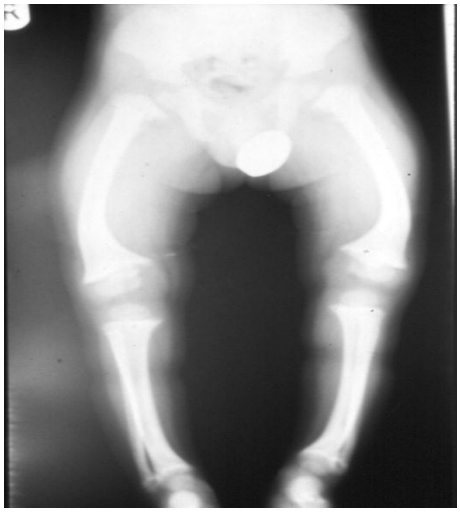
X Ray Findings in Rickets:

Growth Plate& Metaphysial Changes:

Long Bones Deformities:

Epihyseal Widening

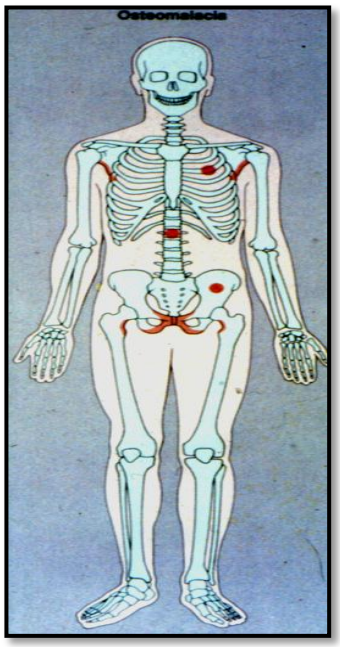




Biochemistry:

Hypocalcaemia, Hypocalciuria, High alkaline phosphatase (try to form too much bone)

Osteomalacia Symptoms and Signs:



- Generalized bone pain, mainly backache
  why back? Because back bear body during walking
- Muscle weakness

Because muscles actions depend on Ca
➔ low Ca = pain and weakness

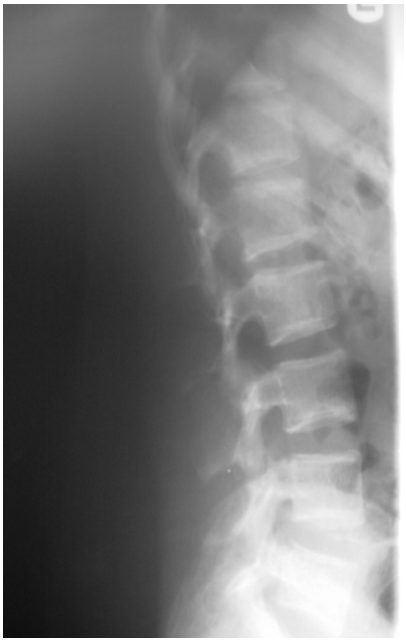
- Reduced bone density
- Vertebral changes : Bi-concave vertebra, vertebral collapse , kyphosis

When bone become soft the convex shape of intervertebral disc will be concave then any press on vertebra with this shape will cause fracture

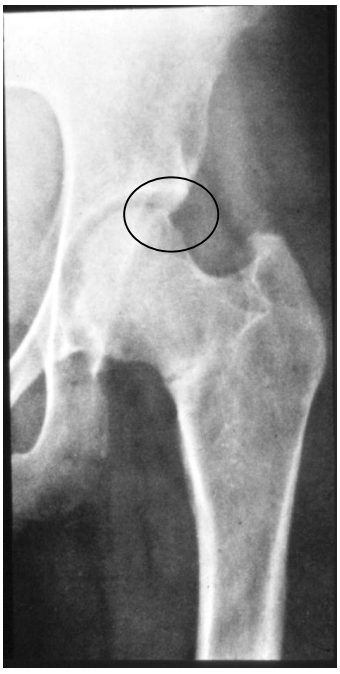
- Stress fractures: (transverse fracture on x-ray) Loosers zones in scapula, ribs ,pelvis, proximal femur. It can happen from only walking up from chair not even a fall

Bi-concave vertebra

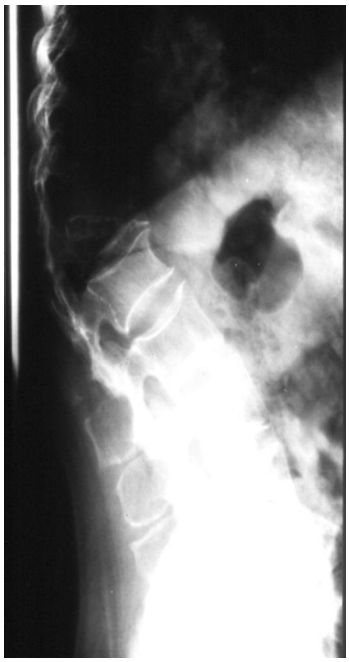
(from above and below) . Any fall can cause compression fracture (looser zoon)



Reduced bone density femoral head stress fracture



Kyphosis



Treatment:

easily treated unless the patient is having intestinal or kidney disease. (should be treated first. because there is no point of the treatment without treat the primary cause).

Vitamin D deficiency

- Rickets

  - adequate Vitamin D replacement
  - sun exposure
  - correct residual deformities

- Osteomalacia

  - Vitamin D + Ca (milk, eggs, fish)
  - fracture management
  - correct deformity if needed

Osteoporosis:

loss of bone unit unlike osteomalacia which affects bone mineralization

- Decreased **bone mass**: decreased amount of bone per unit volume (and this causes reduced density) ➔ osteoclast activity
- Mineralization is not affected
- Mainly post-menopausal (drop Estrogen level) and age related
  Normally we lose 0.3% of bone but in osteoporosis will be about 3% per year

Osteoporosis: Primary and Secondary

- Primary Osteoporosis:
  - Post menopausal
  - Senile process happen in every men and women

Post menopausal Osteoporosis:

- Due to rapid decline in estrogen level
- This results in increased osteoclastic activity
- Normal bone loss usually 0.3% per year
- Post menopausal bone loss 3% per year

-Osteoporosis is painless disease unless it cause fracture (neck of femur).
- Hx. Of F# (high risk group)

Risk Factors in Post menopausal Osteoporosis:

- Race: Caucasian (fair skin)
- Hereditary
- Body build (thin people)
- Early menopause
- Smoking/ alcohol intake/ drug abuse
- ? Calcium intake (low Ca)

Senile Osteoporosis:

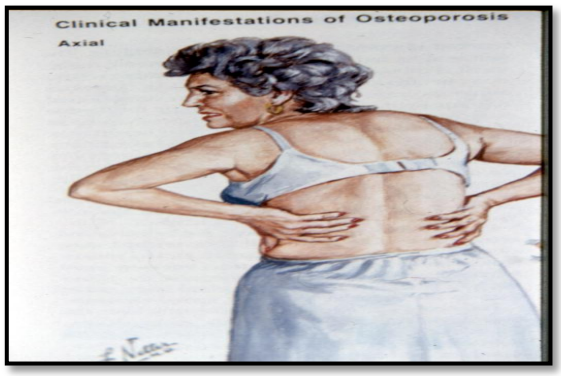
- Usually by 7th to 8th decades there is steady loss of at least 0.5% per year female after 50 years and male after 70 years
- It is part of physiological manifestation of aging

Risk factors in Senile Osteoporosis:

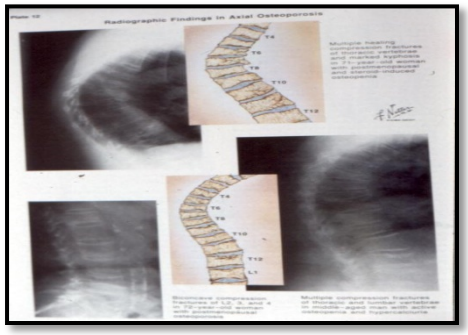
- Male menopause
- Dietary : less calcium and vitamin D and protein
- Muscle weakness
- reduced activity

Clinical Features of Osteoporosis:

- Osteoporosis is a Silent disease
- Osteoporosis is Serious due to possible complications :mainly fractures
- Osteoporosis does not cause pain usually
  pain comes with fracture especially femoral neck which can cause death, or vertebral fracture which starts with gradual back pain as a result of micro-fracture due to vertebral changes.
  Osteoporotic patients are 50% more prone to recurrent fractures.
- Osteoporosis causes gradual increase in dorsal kyphosis
- Osteoporosis leads to loss of height
- Osteoporosis is not osteoarthritis; but the two conditions may co-exist



How does kyphosis and loss of height occurs:



With osteoporosis the anterior part of vertebra narrows which leads to kyphosis and loss of height.

Can't be prevented

Osteoporotic Fractures:

- They are Pathological fractures (malignancies, cyst)
- Most common is osteoporotic compression fracture (OVC #s)
- Vertebral micro fractures occur unnoticed (dull ache)
- Most serious is hip fractures (increased vascularity ➔ bleeding)
- Also common in wrist fractures (Colles fracture)
  colles fractures are distal radius fractures, characterized by dorsal angulation, radial deviation and impaction.

Secondary Osteoporosis:

- Drug induced :steroids (change the dose after one year to minimize side effects), alcohol, smoking, phenytoin (for Epilepsy Pt.),heparin
- Hyperparathyroidism, hyperthyroidism, Cushing's syndrome, gonadal disorders, malabsorption, mal nutrition
- Chronic diseases : RA, renal failure, tuberculosis
- Malignancy : multiple myeloma, leukemia, metastasis

Disuse Osteoporosis:

- Occurs locally adjacent to immobilized bone or joint
- May be generalized in bed ridden patients
- Awareness of and attempts for prevention (by moving the immobilized limb from time to time) are helpful

Osteomalacia vs. Osteoporosis

Osteomalacia	Osteoprosis
Any age	Post-menopause, old age
Pt. ill	Not ill
General ache	Asymptomatic till
Weak muscles	Normal
Looser zones	Nill
Alkaline ph increase	Normal
PO4 decrease	Normal

Prevention of Osteoporosis:

- Prevention of osteoporosis should start from childhood
- Healthy diet, adequate sunshine, regular exercise, avoidance of smoking or alcohol, caution in steroid use

- At some time in the past there was a recommendation of HRT (Hormone replacement Therapy) for post menopausal women ? And men; but now this is discontinued because of HRT side effects

Management of Osteoporosis:

- Drugs
- Exercise
- Management of fractures

Drug Therapy in Osteoporosis:

- Estrogen has a definite therapeutic effect and was used extensively as HRT but cannot be recommended now due to serious possible side effects
- Adequate intake of calcium and vitamin D is mandatory
- Drugs which inhibit osteoclast activities : e.g. Bisphosphonates like sodium alendronate (FOSAMAX , BONVIVA)
- Drugs which enhance osteoblast activities: bone stimulating agents like PROTELOS, FORTEO. The problem in this type of medications is the risk of malignancies.

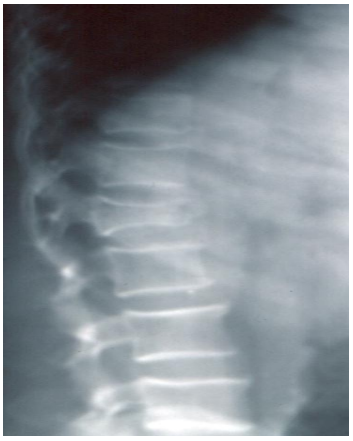
Exercise in Osteoporosis:

- Resistive exercises
- Weight bearing exercises
- Exercise should be intelligent to avoid injury which may lead to fracture

Management of Fractures in Osteoporosis:

- Use of load shearing implants in fracture internal fixation instead of plating.
  In osteoporosis the fractures can heal but not like normal bones.

Vertebral Osteoporotic Compression Fracture:

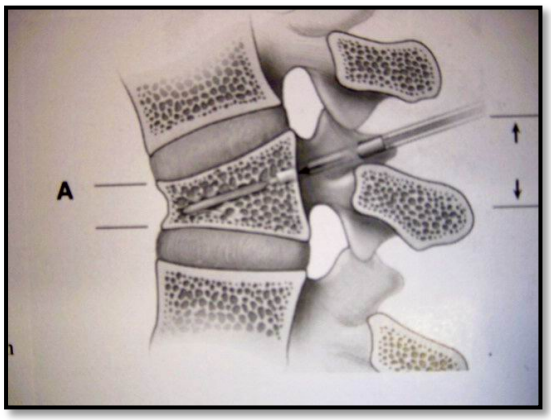


Management of OVC Fractures:

- Pain relief
- Prevention of further fractures
- Prevention of instability
- Vertebroplasty
- Kyphoplasty

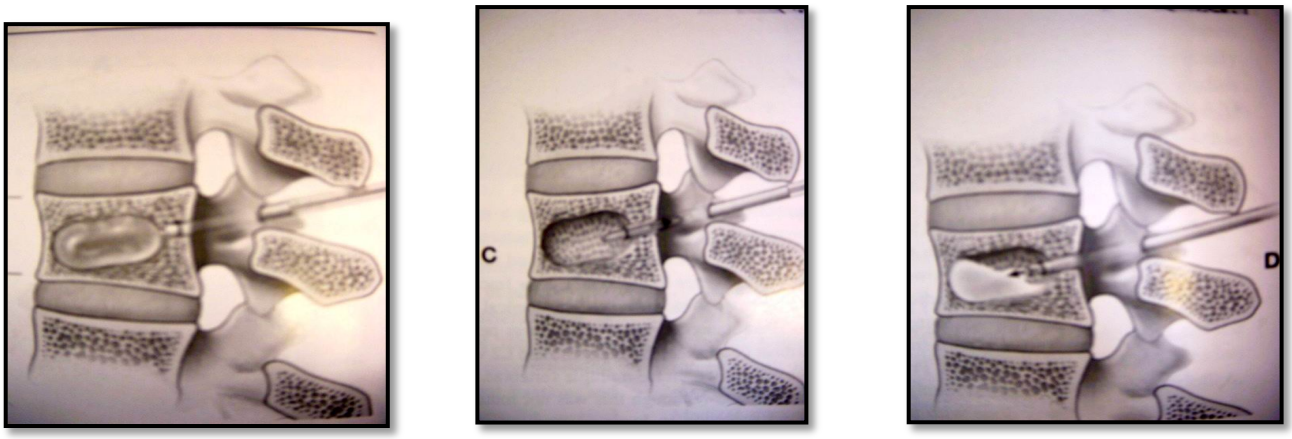
Vertebroplasty:

- Is the injection of bone cement into the collapsed vertebra
- The injection is done under X ray control (image intensifier) by experienced orthopedist or interventional radiologist
- It results in immediate pain relief
- It helps to prevent further OVF
- Possible complication is leakage of cement into spinal canal (nerve injury) or venous blood (cement PE)



Kyphoplasty:

- Is the injection of bone cement into the collapsed vertebra AFTER inflating a balloon in it to correct collapse and make a void (empty space) into which cement is injected
- It is possible that some correction of kyphosis is achieved
- It is safer because cement is injected into a safe void



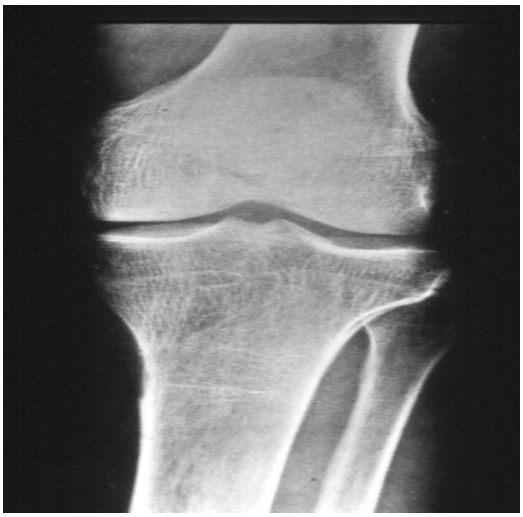
Balloon Kyphoplasty

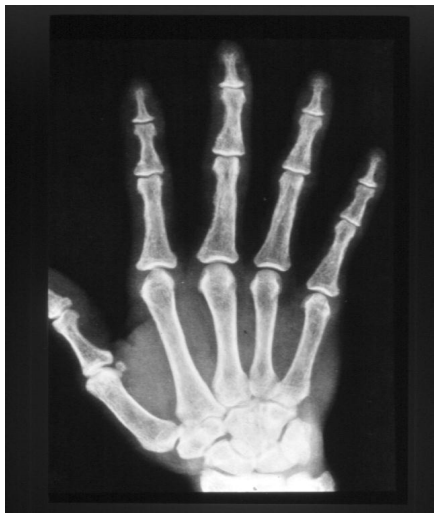
Hyperparathyroidism: (not included)

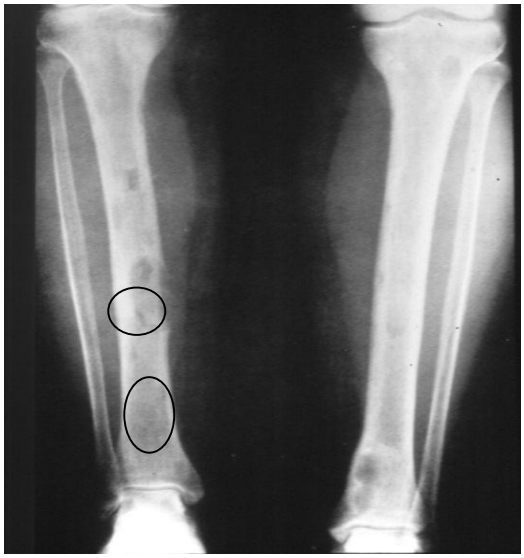
- Excessive PTH secretion : primary, secondary or tertiary
- Leads to increased bone resorption , sub periosteal erosions, osteitis manifested by fibrous replacement of bone
- Significant feature is hypercalcemia
- In severe cases : osteitis fibrosa cystica and formation of Brown tumours

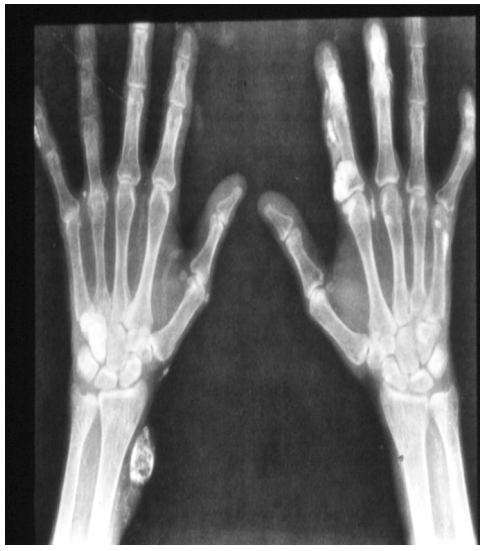
Radiological changes in Hyperparathyroidism:

- Generalised decrease in bone density
- Sub-periosteal bone resorption (scalloping of metacarpals and phalanges)
- Brown tumors. Too much bone reuptake causing areas of empty bone with bleeding, this blood will accumulate like paste forming what calls brown tumors.
  DDx: multiple myeloma, metastasized cancer.
- Chondrocalcinosis (wrist, knee, shoulder)









Brown Tumors

Management of Hyperparathyroidism:

By management of the cause :

- Primary hyperparathyroidism due to neoplasm (adenoma or carcinoma) by excision.
- Secondary hyperparathyroidism by correcting the cause of hypocalcaemia.
- Tertiary hyperparathyroidism by excision of hyperactive (autonomous)nodule, remove.
- Extreme care should be applied after surgery to avoid hypocalcaemia due hungry bones syndrome