Metabolic Bone Disorders

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Orthopedic Surgeons and Bone

- Orthopedic surgeons have to deal with all types of bone: healthy or diseased; so they have to know about bone metabolism
- Bones in the body protect vital organs and give support to muscles and tendons



 Bone may become weak in certain conditions

Bone is a living structure

 Continuous activity in bone during all stages of life

 Continuous bone resorption and bone formation as well as remodeling

 Bone is not only for protection and support but its contents play an important part in blood homeostasis



Bone Metabolism

- Calcium
- Phosphorus
- Parathyroid gland
- Thyroid gland
- Estrogen
- Glucocorticoid hormones

- Intestinal absorption
- Renal excretion
- Diet
- Vitamin D
- Sun exposure

Bone Structure

Matrix

- 40%
- collagen type1
- responsible for tensile strength
- Cells in bone: osteoblasts, osteoclasts, osteocytes

Minerals

• 60%

 mainly Calcium hydroxyapatite,
Phosphorus, and traces of other minerals like zinc

Parathyroid Hormone (PTH)

- Production levels are related to serum calcium levels
- PTH secretion is increased when serum calcium is low
- PTH increases calcium levels by:
- ➢ increasing its release from bone
- increase reabsorption from the kidney (also increase secretion of phosphorus)



Increased concentration of calcium in blood

increase absorption from the intestine

Calcitonin

Secreted by C cells of thyroid gland

Secretion is regulated by serum calcium

 It decreases serum calcium by inhibition of bone resorption and increasing calcium excretion

Bone Strength

 Bone strength is affected by mechanical stress; exercise and weight bearing

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 by a test done at radiology department called DEXA scan (Dual Energy X-ray Absorptiometry).

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Bone Density

- Normal or increased bone density does not always mean increased bone strength
- Brittle bone disease (Osteogenesis imperfecta): bone density is normal but bone is not strong and fragile and may break easily
- Marble bone disease (osteopetrosis): bone is extremely dense, hard but brittle.





Disorders to be discussed

Rickets

Osteoporosis

Osteomalacia

• Hyperparathyroidism

Rickets & Osteomalacia

• Different expressions of the same disease due to Inadequate mineralization

 Rickets affect areas of endochondral growth in children

 Osteomalacia: all skeleton is incompletely calcified in adults

Rickets & Osteomalacia

Causes

- Calcium deficiency
- Hypophosphataemia
- Defect in Vitamin D metabolism nutritional underexposure to sunlight intestinal malabsorption liver & kidney diseases

- Child is restless, babies cry without obvious reason
- Failure to thrive
- Muscle weakness
- In severe cases with very low calcium: tetany or convulsions



Craniotabes



Pigeon chest deformity (Pectus Carinatum)



Pectus Carinatum



 Rickety (Rachitic) rosary :enlargement of the costochondral junctions Rachitic rosary



lateral indentation of the chest (Harrison's sulcus or groove)



- Joint thickening especially around wrists and knees
- Deformity of limbs, mostly genu varum or genu valgum





Rickets: X-ray

- Growth plate widening and thickening
- Metaphyseal cupping
- Long bones deformities



Rickets: X-ray

- Growth plate widening and thickening
- Metaphyseal cupping
- Long bones deformities



Rickets & Osteomalacia: Biochemistry

Hypocalcaemia, Hypocalciuria

High alkaline phosphatase

Osteomalacia: symptoms and signs

- Bone pain, mainly backache
- Muscle weakness



Osteomalacia: X-ray

Reduced bone density

 Vertebral changes: Bi-concave vertebra, vertebral collapse, kyphosis

 Insufficiency fractures: Loosers zones in scapula, ribs ,pelvis, proximal femur







Rickets & Osteomalacia: Treatment

Rickets

*Adequate Vitamin D replacement *Sun exposure *Correct residual deformities Osteomalacia *Vitamin D + Ca *Fracture management *Correct deformity if needed

Osteoporosis

 Decreased bone mass: decreased amount of bone per unit volume (reduced density)

Mineralization is not affected

 Mainly post-menopausal and age related



Honey Comb Pattern In Normal Bone

Honey Comb Pattern With Big Holes In Osteoporosis



Osteoporosis: Primary and Secondary

Primary Osteoporosis:





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



Post-menopausal Osteoporosis: Risk Factors

- Race
- Hereditary
- Body build
- Early menopause
- Smoking/ alcohol intake/ drug abuse
- ? Calcium intake

Senile Osteoporosis

- Usually by 7th to 8th decades
- Steady loss of at least 0.5% per year
- Part of physiological manifestation of aging
- Risk factors in Senile Osteoporosis :
- Dietary: less calcium and vitamin D and protein
- Muscle weakness
- Reduced activity



Osteoporosis: Clinical Features

Silent disease

 Serious due to possible complications: mainly fractures





Osteoporosis: Clinical Features

 Causes gradual increase in dorsal kyphosis and loss of height

 Osteoporosis is not osteoarthritis; but the two conditions may co-exist



Osteoporotic Fractures

Pathological fractures

 Most common is osteoporotic vertebral compression fracture (OVC)

Vertebral micro-fractures occur unnoticed (dull ache)

Most serious is hip fractures

Secondary Osteoporosis

Drug induced: steroids, alcohol, smoking, phenytoin, 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 are helpful

Osteomalacia vs. osteoporosis

Osteomalacia

Unwell Generalized chronic ache Muscles weak Looser's zones Alkaline phosphatase increased Serum phosphorus decreased

Osteoporosis

Well Pain only after fracture Muscles normal No Looser's zones Normal

Normal

Prevention of Osteoporosis

Prevention 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 wome; but now this is discontinued

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)

 Drugs which enhance osteoblast activities: bone stimulating agents like Teriparatide (FORTEO), strontium ranelate (PROTELOS)

Exercise in Osteoporosis

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





Management of Fractures in Osteoporosis

 Use of load-shearing implant (nail) in fracture internal fixation instead of load-bearing implant (plate)





Vertebral Osteoporotic Compression Fracture: Management

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



vertebroplasty

- Injection of bone cement into the collapsed vertebra
- under X ray control
- Results in immediate pain relief and helps to prevent further collapse
- Possible complication is leakage of cement into spinal canal (nerve injury) or venous blood (cement embolism)



Kyphoplasty

- 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



Hyperparathyroidism

Primary: Adenoma of the gland

Secondary: as a result of low calcium

 Tertiary: as a result of prolonged or sustained stimulation; hyperactive nodule or hyperplasia

Hyperparathyroidism

- Leads to increased bone resorption, subperiosteal erosions, osteitis manifested by fibrous replacement of bone
- Significant feature is hypercalcemia
- In severe cases: osteitis fibrosa cystica and formation of Brown tumors



Hyperparathyroidism: Radiological changes

- Generalized decrease in bone density
- Sub-periosteal bone resorption (scalloping of metacarpals and phalanges)
- Brown tumors
- Chondrocalcinosis (wrist, knee, shoulder)







Hyperparathyroidism: Management

- Treatment of the underlying 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
- Extreme care should be applied after surgery to avoid hypocalcaemia due to hungry bones syndrome

Thank you