# CALCIUM METABOLISM
# CLINICAL DISORDERS OF CALCIUM METABOLISM

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# INTRODUCTION

- Dietary intake: 1g/day
- Most is lost in stool and **150 mg** is absorbed into circulation
- Calcium absorption in the GIT is influenced by predominantly vitamin D
- The bulk (99%) of calcium in the body is stored in the skeletal system.
- The remaining 1% is distributed between circulation (900mg) and in the intracellular (muscle predominantly) compartment.

# CONT.

- Calcium in circulation determines the physiologic function of calcium. The 3 major functions of calcium include:
  1. Major mineral in bone (offers strength); bone acts as a storage compartment for bone
  2. Neuromuscular conduction and muscular contraction
  3. Signal transducer in inter - and intracellular communication
- Calcium in circulation is transported albumin bound. Therefore calcium can either be albumin bound or unbound.
  - Perturbations in albumin levels affect calcium levels thus correction has to be done when determining calcium levels e.g. in a wasted patient, one with liver conditions or nephrotic syndrome

# CONT.

- Normal range for calcium: 2.25 mmol/L – 2.5 mmol/L (on a background of normal albumin levels)
- The key hormones involved in calcium regulation:
  1. PTH
  2. Vitamin D
  3. Calcitonin

# PTH

- 84 amino acid chain produced by the parathyroid gland
- Effects
  - Bone: demineralization
  - Kidney: stimulates 1 - alpha hydroxylase at the proximal convoluted tubule
    - This promotes 1 - hydroxylation of the 25 - hydroxylated vitamin D (in the liver) resulting in the production **1, 25 – dihydroxy – vitamin D**
    - Clinical application: metabolic bone disease in chronic kidney disease
      - Failure of vitamin D activation → reduced intestinal absorption of calcium → hyperparathyroidism → bone demineralization and concomitant softening

# VITAMIN D

- Exists as 7 – cholecalciferol under the skin where it is activated by sun light. It is 1 - hydroxylated in the liver and 25 – hydroxylated in the kidneys.
- Effects: Acts on the intestines to facilitate absorption of calcium.

# CALCITONIN

- Produced by the para - follicular cells of the thyroid
- Mechanism is being researched on

## OTHER HORMONES

- Thyroxine
  - Clinical application: thyrotoxicosis → hypercalcemia (the converse is true)

# CLINICAL DISORDERS OF CALCIUM METABOLISM

1. HYPERCALCEMIA
2. HYPOCALCEMIA

# 1. HYPERCALCEMIA

- Definition: a state where the serum calcium level is above 2.5 mmol/L
- Manifestations: (Depend on how high the levels go)
  - Neuromuscular/ Neurologic:
    - Confusion/ cognitive impairment → abnormal sensory manifestations ('things crawling under my skin'; EXCLUDE ORGANIC DISEASE BEFORE DISMISSING SUCH A PATIENT)
  - Musculoskeletal: Profound lethargy ('sina nguvu')
  - GIT: Anorexia, nausea, vomiting (due to altered GIT motility)

# CONT.

- Cardiovascular: calcified blood vessels (with chronic hyper - calcemia $2^0$ to metastatic calcification)
  - NB: Metastatic calcification is predicated on normal tissue in the setting of hyper - calcemia; dystrophic calcification is predicated on injured tissue
- Renal manifestations
  - Functional: polyuria, polydipsia
  - Structural:

# ETIOLOGY

- Major cause: Parathyroid adenoma seen in MEN syndromes.
- Other causes:
  - Malignant hyper - calcemia e.g. multiple myeloma, breast Ca with bone involvement.
    - Metastatic tumors that cause bone demineralization $2^0$ to osteolysis
  - Vitamin D excess (take a drug history esp. herbal medication)

# INVESTIGATION

- Calcium levels corrected with albumin
- Imaging
  - U/S, CT scan, Skeletal survey (look for metastatic bone disease)
- Technetium ($Tec^{99}$) bone scan

# MANAGEMENT

- Relieve symptoms
  - Lower calcium by giving saline (oral saline water or IV) → paramount
  - Loop diuretics (furosemide)
  - Steroids
  - Anti - neoplastics (depending on the underlying cause)
- Address the underlying cause

# 2. HYPOCALCEMIA

- Definition: a state where the serum calcium level is below 2.2 mmol/L
- Commonest cause: hypo - parathyroidism $2^0$ to surgical excision of the thyroid gland etc.
- Other causes:
  - Congenital absence (presents in childhood)
  - Post – irradiation

# MANIFESTATIONS

- Neuromuscular: calcific lesions in the brain → movement disorders, frequent seizures if epileptogenic areas are involved; carpo - pedal spasm (trousseau's sign), chvostek sign, tetany

# TREATMENT

- IV calcium gluconate
- Active vitamin D (alpha D3) → helps with absorption

# ASSIGNMENT (MUST KNOW)

- OSTEOPOROSIS: Reduced bone matrix and mineralization
- OSTEOMALACIA: Predominantly reduced bone mineralization

# TYPED BY EFFIE NAILA

NOTHING EVER GOES AWAY UNTIL IT HAS TAUGHT YOU WHAT YOU NEED TO KNOW.

HANG IN THERE.

JESUS LOVES YOU AND HE KNOCKS AT THE DOOR OF YOUR HEART TODAY.

LET HIM IN. YOU WILL NOT REGRET IT.