**UNIVERSITY OF NAIROBI**

**SCHOOL OF MEDICINE**

**DEPARTMENT OF CLINICAL CHEMISTRY**

**Wednesday 17th February, 2010 Dr. Elizabeth Odera**

**CALCIUM AND PHOSPHATE METABOLISM**

* Co exist in relatively fixed form proportions in mineral phase of hard tissues – bone dentin and enamel
* Calcium ions and inorganic phosphate depend in part on balance between bone mineral deposition and bone resorption
* Regulation of calcium ions and inorganic phosphate – Parathyroid Hormone, Vitamin D and Calcitonin

**Calcium**

* Most is extracellular – 99% is in hard tissues exists in crystalline hydroxyapatite form
* Exists in plasma in 3 physiochemical states;
* Approximately 45% is protein bound, most 80% to albumin and 20% to globulin
* Approximately 45% ionized form and is physiologically active form
* Approximately 10% complexed with citrate, lactate, phosphate and bicarbonate
* The ionized form is in dynamic equilibrium with other forms; is affected by pH and plasma proteins
* Acidosis leads to dissociation hence increase in ionized calcium

**Physiological Functions of Calcium**

Intracellular calcium plays a vital role in;

* Regulating cell functions especially adenylate cyclase and phosphodieterase
* Function as plasma membrane and regulates membrane permeability and affects neuromuscular release
* Regulation of secretions of endocrine glands – parathyroid hormone, calcitonin
* Important in cell coagulation

**Phosphate**

* Distributed equally between extracellular and intracellular compartments
* Intracellular phosphate; component of structural organic – macro molecules, phospholipids and phosphoproteins called organic phosphate
* Extracellular phosphate occurs in organic form as hydroxyapatite
* Plasma of serum phosphate – most occurs in the inorganic form (Pi) as mono or dihydrogen forms
* 15% of plasma phosphate is protein bound and the rest in complexed and free form

**Causes of Hypercalcimia**

* Primary hyperparathyroidism
* Malignancy
* PTH producing tumors
* Skeletal metastasis of tumor
* Multiple myeloma
* Vitamin D toxicity
* Drug induced hypercalcemia – thiazides
* Sarcoidosi – absorption of calcium in GIT; secondary to vitamin D hypersensitivity
* Thyrotoxicosis
* Immobilization

**Hyperphosphatemia**

* Poisoning – phosphate containing laxatives
* Respiratory acidosis
* Chronic renal failure
* Hypoparathyroid state
* Acromegally

**Hypophosphatemia**

* Intake; deficiency of dietary phosphate
* Redistribution – glucose infusion and respiratory alkalosis
* Renal causes;
* Specific phosphate transport defects are x-linked dominant hypophosphatemia
* Multiple renal tubular transport defect e.g. idiopathic fanconi syndrome and galactosaemia

**Laboratory findings depends on the cause**

* Changes (serum) of calcium, inorganic phosphate, 25 – OH D and 1 α25 (OH)2D vary with different disorders
* Vit D deficiency – serum calcium is normal or low
* Phosphorus and 25OH-D – characteristically low
* Renal tubular disorders – normal calcium but low phosphorus (inorganic)
* Chronic renal failure;
* Hyperphosphatemia
* Hypocalcemia
* Normal 25(OH)D
* Low 1,25(OH2)D
* Radiological investigations are important