SydPath
ST VINCENT'S PATHOLOGY

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CALCIUM INFORMATION SHEET

PHYSIOLOGY

Calcium is a divalent cation with vital roles in bone structure, coagulation and signalling in excitable membranes. The average dietary intake is about 25 mmol/day, of which 10 mmol is absorbed and 5 mmol/day returned to gastrointestinal tract in biliary, pancreatic and intestinal secretions. Serum concentrations are very tightly controlled by a feedback loop between free (ionised) calcium and parathyroid hormone with vitamin D regulating calcium absorption from the gut. Intracellular calcium concentrations are much lower than plasma at about 0.1 µmol/L in the cytosol and 0.1-10 mmol/L in mitochondria. Total body calcium stores are about 35 moles, of which over 99% is in bone. In the circulation 45% of calcium is protein bound (35% to albumin, 10% to globulins) and the remainder is in the filterable fraction, of which 48% is ionised and the rest bound to anions such as phosphate, citrate, or carbonate. About 250 mmol of calcium is filtered at the glomerulus each day and over 95% is resorbed, leaving less than 7.5 mmol/d of calcium excreted in the urine.

HYPERCALCAEMIA

SYMPTOMS: Polyuria (decreased ADH effect), polydipsia, anorexia, nausea, fatigue, mental clouding, dysphoria, bone pain, pathological fractures, pancreatitis, ulcer (stones, bones, psychic moans, abdominal groans)

CAUSES OF HYPERCALCAEMIA:

- Factitious (prolonged use of tourniquet, note elevated albumin measurement)
- Hyperparathyroidism (primary or tertiary)
- Hypercalcaemia of malignancy (especially breast, lung, kidney, prostate, haematological malignancies)
- Hypervitaminosis D
- Milk alkalai syndrome (antacids)
- Drugs (thiazides, lithium)
- Immobilisation
- Granulomatous disease (sarcoid, TB)
- Endocrine causes (thyrotoxicosis, acromegaly, hypoadrenalism)
- Familial hypocalciuric hypercalcaemia (defective PTH receptor)

HYPOCALCAEMIA

CAUSES OF HYPOCALCAEMIA

- Factitious (EDTA contamination, low serum albumin, drip-arm collection)
- Hypoparathyroidism, Pseudohypoparathyroidism
- Hyperphosphataemia (renal failure, phosphate supplements)
- Magnesium deficiency

- Hypovitaminosis D (low intake, malabsorption, decreased hydroxylation, nephrotic syndrome, dilantin, alcohol, glutethimide)
- Medullary carcinoma of the thyroid (increased calcitonin)
- Acute pancreatitis
- Drugs (mithramycin, EDTA, frusemide, ethacrinic acid, citrate)

CALCIUM CORRECTED FOR ALBUMIN CONCENTRATION:

As about half of the total albumin in the circulation is bound to albumin, the total calcium concentration will vary with changes in albumin concentration even if the free calcium remains constant. It is possible to make an approximate correction for this effect and adjust the measured total calcium to give a value as though the albumin was normal. The usual reference intervals for calcium can then be applied. A common equation is as follows:

Ca(corrected) = Ca(measured) + (40-Alb) x 0.02