Calcium Ca^{++}

- K pp. 74 - 87
- C pp. 43-44

Objectives

- Upon completion of this lecture the learner should be able to:
  - Discuss the functions, physiology, and pathophysiology of calcium .
  - Discuss the clinical settings or risks for calcium emergencies .
  - Identify abnormal signs,symptoms, and clinical features of calcium emergencies .

Objectives (cont.)

- Correlate abnormal findings with other (e.g., acid-base) clinical conditions of patients.
- Appreciate the need for rapid intervention in patients with certain hypocalcemic complications.
- Discuss the prehospital managements of complications that may occur with a hypocalcemic emergency .

Priorities - review

- When fluids and electrolytes are altered, they should be corrected in the following order:
  - Volume
  - pH
  - Potassium, magnesium, and ***calcium***
  - Sodium and chloride

Calcium - distribution

- Intracellular
  - 98-99% stored in skeleton and teeth
  - Ca^{++} is the least abundant intracellular cation
- Extracellular
  - 1% ECF (3rd space + serum)
  - Roughly 50% is protein-bound
    - In malnutrition ECF (serum) Ca^{++} remains steady, even if total body Ca^{++} is depleted

Calcium Balance

- Significant and rapid Ca^{++} shifting from
- intracellular to extracellular occurs in response to :
  - Excessive production
  - Acute loss
    - binding to albumin
  - Chronic
    - inadequate intake

Calcium - function

- Muscle
  - smooth and skeletal
- Cardiac
  - conduction
  - contraction
- Clotting

Calcium - pathophysiology

- Hypocalcemia
- Muscular
- Fractures
- Bleeding disorders
- Acid-base abnormalities
- Hypercalcemia
- Cardiac
  - conduction abnormalities (HB, arrhythmias)
- Fractures

Calcium Abnormalities

- Hypocalcemia (less than 8.5 mEq/L)
- Hypercalcemia (greater than 10.5 mEq/L)
- Excessive losses or retention

Hypocalcemia

- Causes
- Acute loss
  - Alkalosis
    - Binding of Ca^{++} to albumin
  - Salt (PO4), soap (pancreatitis)
  - Shift
    - From ECF into the cell
      - Cell damage (sepsis)

Hypocalcemia - Clinical Presentation

- Neuromuscular :
  - Painful cramps
  - Tetany
- CNS
  - Weak and dizzy
  - ALOC
  - Paresthesias

Hypocalcemia - Treatment

- CO_2M_3E $BI_{NS}G$
- Correct volume status
- Treat complications (seizures, arrhythmias)
- Correct acid-base status (resp. alkalosis)
- Mg^{++} IV or PO
  - i.e., non-urgent
  - often this alone will correct low Ca^{++}
- CaCl 10 mL of 10% solu.
- Slow IV, over 10 minutes

Hypercalcemia

- Causes
- Malignancies (increased resorption)
  - known, or occult
- Immobility
- Excessive intake / potassium load
- Decreased excretion
- Drugs
  - excessive vitamin D

Hypercalcemia - Clinical Presentation

- 911 :
  - pain
  - fracture
  - psychosis
- Mnemonic :
  - stones
  - bones
  - moans
  - groans

Treatment of Hypercalcemia

- CO2M3E $BI_{NS}G$
- Correct volume
  - IV NS 5 - 10 L
- Furosemide 80 mg IV
- Methylprednisolone 125 mg IV

Summary

- We have discussed :
- Functions of and homeostasis of Ca^{++}
- Regulators of Ca^{++} balance
- Pathophysiology and causes of Ca^{++} abnormalities
- Common clinical presentations of Ca^{++} abnormalities
- Treatment of probable hypocalcemia and documented hypercalcemia