



EMC 360 Acute Fluid and
Respiratory Disorders

10 Phosphorus

K pp. 98 - 107
B p. 292
C p. 46



Objectives

- Upon completion of this lecture the learner should:
 - Be familiar with the functions, physiology, and pathophysiology of phosphorus.
 - Be able to identify etiologies and risk factors predisposing to phosphorus emergencies.
 - Be familiar with some signs, symptoms, and clinical features of phosphorus emergencies .
 - Discuss the prehospital concerns regarding a probable phosphorus emergency.



Distribution

- Intracellular
 - Bone structure (80%)
 - HPO_4^{2-} is the most abundant intracellular anion. Many and varied intracellular functions.
- Extracellular
 - 1% ECF (3rd space + serum)
 - Serum Ca^{++} and phosphate are inversely are inversely proportional.
 - Free anion - with unusual variations



Phosphorus HPO_4^{--}

- Significant shifting from extracellular to intracellular occurs in response to:
 - Injury (burns)
 - Acid-base imbalance (alkalosis)
 - Catabolic states (malnutrition)
 - Glucose or insulin excess



Balance

- Absorption is proportional to dietary intake
 - 70 % is absorbed via passive transport
 - 30 % via active transport
 - dependent on vitamin D
- Excretion
 - renal
 - reabsorbed in the proximal tubules
 - Increased reabsorption
 - hypovolemia



Function

- Cellular
 - Bones, teeth
 - Cell wall
- Intracellular
 - Metabolism
 - Energy transformation and storage (ATP)
 - Utilization of CBH, protein, fat
 - Oxygen delivery (2,3, DGP)
 - Genetic (nas / chromosomes)
- Extracellular
 - Buffer



Hypophosphatemia Causes

- Like hypokalemia causes
- Losses due to intracellular shift
 - Alkalosis
 - Drugs
 - Insulin
- GI losses
 - Vomiting
 - Diarrhea



Hypophosphatemia Pathophysiology

- p. 101
- Nutrition
 - Vitamin D deficiency
- Alcoholism (hypomagnesemia)
- Acid-base and glucose abnormalities
- Diuretics
 - rarely



Hypophosphatemia Pathophysiology

- Neuromuscular dysfunction
- Cellular
 - RBC malfunctions
 - Hb - decreased O₂ - off loading
 - WBC malfunctions
 - Platelet malfunction



Hypophosphatemia Clinical Presentation

- Neuromuscular Sx
 - Weakness
 - Paresthesias
- ABCs
 - Hb - decreased O₂ - off loading (bicarb-like)
 - Hypoxia
 - Platelet malfunction
 - Bleeding (e.g., GI bleed)



Hyperphosphatemia Pathophysiology

- Etiology
- Renal failure to excrete HPO₄⁻
- Movement out of cells into the ECF
- Excessive intake of vitamin D
- Physiologic and clinical effects
- Renal failure
- Hypo - Ca⁺⁺



Treatment of Phosphate Emergencies

- Correct the underlying disorder
- CO₂M₃EBIG
 - O₂ - Hb - Hypoxia
 - Large bore IV (2) - Bleeding (e.g., GI bleed)



Treatment of Renal Failure Patients

- For hypocalcemia
 - Give oral calcium (Tums®)
- For hyperphosphatemia
 - Give oral phosphate binders (i.e., aluminum antacids Amphogel®)
- Avoid Mg^{++} (magnesium antacids, Maalox®)



Summary

We have discussed:

- Functions and homeostasis of Phosphorus HPO_4^{-}
- Regulators of HPO_4^{-} balance
- Pathophysiology and causes of HPO_4^{-} abnormalities
- Clinical presentations and associations
- Treatment of associated conditions
 - DKA
 - Alcoholism
 - Renal failure
