Hypophosphatemia

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Risk

- Incidence: 1% of population, 5–20% of hospitalized pts

Perioperative Risks

- Acute respiratory or cardiac failure, generalized weakness, confusion, seizures

Worry About

- Periop respiratory or cardiac failure.
- Too rapid correction can cause hypocalcemia or Ca^{2+} deposition in tissues.

Overview

- Of total body phosphorus, 90% is distributed in bone, 10% intracellularly, and <1% in extracellular fluid.
- Normal ionized phosphorus (Pi) is 2.7–4.5 mg/dL. It may fall by 30% after administration of carbohydrates/insulin. Higher in childhood and in postmenopausal women.
- Serum concentration does not correlate closely with body stores.
- Normal requirement is 1 mmol/kg/d.
- Primary absorption of Pi is in the duodenum and jejunum, stimulated by vitamin D.
- Kidney: Primary filtration in the kidney and primary reabsorption in the proximal tubules, with only 10% reabsorption in the distal tubules. Regulated by PTH, cortisol, high dietary intake, and calcitonin. Increased Pi excretion with volume expansion.
- Functions: Phosphates provide the primary energy bond in ATP and creatine phosphate. Severe Pi depletion can cause cellular energy depletion, lack of cAMP; Pi is also important for cellular structures as phospholipids, nucleic acids, and cellular membranes. As part of 2,3-DPG, phosphates promote release of O_2 from Hgb.

Etiology

- Decreased intake, increased loss, redistribution, occasionally genetic
- Decreased absorption and/or intake: Malnutrition, malabsorption syndromes, Crohn disease, celiac disease, inadequate replacement in TPN, hemodialysis, Mg^{2+} and aluminum antacids, sucralfate, vitamin E deficiency
- Increased losses: Rapid volume resuscitation, steroids, pancreatitis, burns, alcoholism, dialysis, hyperparathyroidism, diuretics
- Redistribution: Shift from serum into cells (hyperglycemia, glucose infusion, hormonal effects), catecholamines, insulin, glucagon, calcitonin
- Respiratory alkalosis, leukemic blast cell crisis

Usual Treatment

- Prefer oral over parenteral because of risk of resultant hypocalcemia or calcification of tissues. Suggested dose of K-PHOS is 2–5 mg/kg per d.
- If parenteral therapy is required, administer 10–45 mmol of IV Na^+ or K^+ phosphate over 6–12 h. Important to monitor Ca^{2+}, K^+, and Mg^{2+} levels.

Assessment Points

System	Effect	Result
CV	Depressed ATP, impaired response to norepinephrine/angiotensin	Heart failure
HEME (WBC)	Impaired phagocytic, migratory, and bactericidal activity	Sepsis
(Platelets)		Thrombocytopenia, impaired clot retraction
(RBC)	Reduced RBC 2,3-DPG	Increased Hgb-O_2 affinity
CNS	Neurologic dysfunction	Seizures, coma, hyperreflexia, paresthesia, dysarthria
MS	Respiratory failure, motor weakness	Proximal > distal, rhabdomyolysis, myoglobinuria

Key References: Bugg NC, Jones JA: Hypophosphatemia. Pathophysiology, effects and management on the intensive care unit, *Anaesthesia* 53(9):895–902, 1998; Ianov I, Wilkerson DL: Hypophosphatemia and acute postoperative respiratory distress, *J Ark Med Soc* 106(11):265–266, 2010.

Perioperative Implications

- Potential need for postop ventilation in pts with hypophosphatemia.
- Correction of severe hypophosphatemia should be done slowly over several hours to days to prevent severe hypocalcemia and vascular and interstitial Ca^{2+} precipitation.
- Consider hypophosphatemia in the pt who is difficult to wean off the ventilator, as this might be the cause.

Hypopituitarism

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Risk

- Incidence: 45.5:100,000.
- 30% of pituitary macroadenomas (>10 mm) cause one or more hormone deficiencies.
- About 4.2 years after pituitary radiation therapy, some 50% of pts have hypopituitarism.
- Less common causes include empty sella syndrome, head trauma, infiltrative disease, and expansive internal carotid artery aneurysm.

Perioperative Risks

- If hormone replacement is adequate, surgery presents no increased risk.
- If due to secreting tumor, there is an increased risk of Cushing disease, acromegaly, SIADH, or hyperthyroidism.

Worry About

- Concerns regarding manifestations of disease process: Cushing disease (hypercortisolism secondary to an adrenocorticotropic hormone–secreting adenoma), acromegaly (secondary to a growth hormone–secreting adenoma), and hyperthyroidism in the setting of thyrotropic adenomas.
- Operative risks: Bleeding, DI, and SIADH.
- GH-secreting adenoma predisposing to acromegaly and subsequent airway abnormality and OSA.
- Hypoglycemia.
- Altered volume status due to increased urinary losses.
- Adequacy of adrenal function.
- Increased risk of CV disease.
- Possible increase in ICP.

Overview

- Partial or complete disruption of pituitary gland secretion. Symptoms result from end-organ hypofunction or dysfunction. Organs affected include adrenal and thyroid glands, reproductive system, and liver (glucose production) and kidneys.
- Pt may manifest cortisol deficiency, hypothyroidism, amenorrhea, infertility, insulin-induced hypoglycemia, and/or DI.
- Pituitary apoplexy is the abrupt destruction of pituitary tissue resulting from infarction or hemorrhage. Symptoms include sudden loss of pituitary function with hypotension, eye pain, blindness, and ophthalmoplegia.

Etiology

- 61% secondary to tumors of the pituitary gland
- 9% due to other types of lesions
- 19% due to other causes (radiation, hemorrhage, infarct, head trauma, infiltrative diseases)
- No cause identified in 11%

Usual Treatment

- Surgical resection of adenoma with appropriate hormonal replacement therapy for ACTH: Prednisone or cortisone PO; for TSH: thyroxine PO; for LH and FSH: estrogen and progesterone PO for women, testosterone esters IM for men; for ADH: intranasal desmopressin.