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.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
HEENT	Mandibular and oral soft tissue hyperplasia in acromegalics		Airway exam Check ring size	
CV	Hypovolemia Catecholamine resistance		Orthostatic hypotension	Give steroid replacement and observe effect on BP
GI	Hypoaldosteronism	Anorexia, N/V, weight loss, abdominal pain		Hyperkalemia, hyponatremia, hypocalcemia, hypovolemia
ENDO	Decreased ACTH	Fatigue, fever, stress-induced hypotension, and hyponatremia	Fever, hypotension, wt loss, mental status	Morning cortisol level, rapid ACTH stimulation test, insulin tolerance test
	Decreased LH, FSH	Decreased libido and sexual function, amenorrhea	Regression of secondary sexual characteristics	FSH, LH serum levels, serum estradiol and testosterone
	Decreased GH	Fatigue		Insulin-induced hypoglycemia, serum IGF-I
	Decreased TSH	Wt gain, cold intolerance, depression, constipation, hair loss	Myxedema, hyporeflexia	TSH, T_4
	Increased prolactin	Lactation, amenorrhea	Galactorrhea	Serum prolactin
MS	Increased GH in acromegalics		Large hands, feet, mandible, tongue	
RENAL	Increased vasopressin Decreased vasopressin	Excessive thirst Increased UO and thirst	 Hypovolemia Hypotension	Hyponatremia Hypernatremia Dilute urine

Key References: Nemergut EC, Dumont AS, Barry UT, et al.: Perioperative management of patients undergoing transsphenoidal pituitary surgery, *Anesth Analg.* 2005;101(4):1170–1181; Bajwa SJ, Kaur G: Endocrinopathies: the current and changing perspectives in anesthesia practice, *Indian J Endocr Metab.* 2015;19(4):462–469.

Perioperative Implications

Preoperative Preparation

- Ensure adequacy of hormone replacement therapy.
- Check serum Na^+, Ca^{2+}, and K^+ and correct if necessary.
- Determine volume status and adequacy of fluid replacement.
- In acromegalics, careful airway assessment and cardiac workup for possible cardiomyopathy.
- Consider steroid supplementation (hydrocortisone 100 mg/70 kg tid).
- Clinically assess for signs of increased ICP (N/V, papilledema, headache, blurry vision).

Monitoring

- Consider arterial line if severe CV compromise, central venous pressures if indicated by inadequate preop correction of fluid status
- Monitor lytes frequently if hyponatremia or hypernatremia is not corrected preop.
- Consider glucose monitoring.

Airway

- Acromegalic pts with normal airway exams may be difficult to intubate. Have LMA, fiberoptic, or glidescope available.
- Difficult airways in acromegalic pts due to macroglossia, hypertrophy of soft tissues of oropharynx, enlargement of soft palate, epiglottis, and aryepiglottic fold.

Induction

- Little risk of increased ICP with pituitary adenomas.
- No special technique if hormone replacement and volume status are adequate.

Maintenance

- Maintain normocarbia for pituitary surgery.
- Titrate narcotics and benzodiazepines carefully in pts with OSA secondary to GH-secreting tumors.

Extubation

- Routine (for nonpituitary surgery). May need CPAP postop if pt requires use at home for possible OSA.

Adjuvants

- Intraop DI treated with vasopressin 5 to 10 IU SQ or IM every 4–6 h

Postoperative Period

- Polyuria and polydipsia with dilute urine may indicate development of DI.
- Postop hypopituitarism may require steroid replacement therapy.

Anticipated Problems/Concerns

- Acromegalic pts should be treated as having difficult airways.
- Pts with GH deficiency may manifest hypoglycemia.
- Electrolyte abnormalities (K^+, Na^+, Ca^{2+}) and possible hypovolemia, predisposing to arrhythmias and CV instability.

Hypoplastic Left Heart Syndrome

Gianluca Bertolizio

Risk

- Frequency: 0.163–0.184 per 1000 live births
- 7–9% of all congenital heart diseases
- Approximately 1000 infants/y in USA
- Male to female: Up to 2:1
- 15% is associated with genetic syndromes, such as Turner syndrome, Jacobsen syndrome, trisomy 13, trisomy 18, and Smith-Lemli-Opitz syndrome

Perioperative Risks

- 60% prenatally diagnosed (18–22 wk)
- 90% mortality within the first month without operation
- 70% overall survival to adulthood
- 30% mortality within 30 d after stage I
- 10–15% interstage mortality

Worry About

- Premature closure of the DA and the presence of a restrictive PFO/ASD will cause a rapid decompensation of pt after birth.
- Severe hypoxemia due poor intracardial mixing or increased PVRs.
- Pulmonary edema due to pulmonary overcirculation and high LA pressure.
- Systemic and coronary hypoperfusion due to runoff (mainly diastolic) to the pulmonary circulation.
- High risk of myocardial ischemia.
- Aortic atresia + mitral atresia/stenosis subtypes have higher mortality.
- Possible presence of left ventriculocoronary fistulae.

Overview

- Secondary to a severe hypoplasia of the left heart structures (MV, AV, LA, LV, ascending aorta, aortic arch), which leads to different degrees of obstruction of LA and/or LV outflow tract. It is defined as single ventricle physiology, which has the following characteristics:
  - Complete mixing of the Qp and Qs.
  - Ventricular output = Qp + Qs.
  - Existence of two parallel circulations: The distribution of systemic and pulmonary blood flow is dependent on the relative resistances to flow.
  - $SaO_2 = SpaO_2$.
- The goal is to manage the PVR/ SVR ratio to maintain the Qp:Qs~1.
- Qp:Qs is calculated as the following = $(SaO_2 - SvO_2) / (SpvO_2 - SpaO_2)$.
- SvO_2 = mix venous saturation and $SpvO_2$ = pulmonary venous saturation. $SpvO_2$ = 100% if there is no lung disease.

Etiology

- It is a ductal-dependent circulation with complete mixing of blood through PFO or ASD.
- In a normal fetus systemic circulation, 90% of circulating blood flow is guaranteed by FO and DA. After birth, both DA and FO close. In the neonate with HLHS, blood cannot flow into the LV (MV atresia) or into the aorta (AV/aortic atresia); therefore it crosses the atrial septum through the PFO/ASD,