Managing Phosphorus & Magnesium Disorders

Adesola Odunayo, DVM, MS, DACVECC
University of Tennessee

You have asked…

What role do phosphorus and magnesium play in the health of veterinary patients?

The expert says…

Phosphorus is an important component of nucleic acids, phospholipids, and phosphoproteins and has a role in the metabolism of protein, fat, and carbohydrate and in processes requiring energy from adenosine triphosphate (ATP). Magnesium, meanwhile, is important for energy production, vascular smooth muscle tone, and protein synthesis; helps maintain appropriate intracellular potassium concentration; and regulates cytoplasmic calcium concentrations.

Phosphorus

Approximately 85% of total body phosphate is inorganic hydroxyapatite in bone and about 15% is in soft tissues. Phosphorus levels are regulated by the GI tract and kidneys under the influence of calcitriol, calcitonin, and parathyroid hormones.

Hypophosphatemia

Hypophosphatemia occurs when serum phosphorus concentrations are less than 2.5 mg/dL, although reference ranges vary (see **Causes of Hypophosphatemia**). Hypophosphatemia decreases ATP concentrations in RBCs, increasing erythrocyte fragility and leading to hemolysis (generally not observed until phosphorus concentration is <1 mg/dL). Hypophosphatemia

ATP = adenosine triphosphate

MORE ▶

Causes of Hypophosphatemia

Decreased intake	■ Phosphate binders ■ Vitamin D deficiency
Increased loss	■ Hyperadrenocorticism ■ Primary hyperparathyroidism ■ Renal tubular disorders
Laboratory error	
Redistribution from extracellular fluid to intracellular space	■ Hypothermia ■ IV dextrose administration ■ Refeeding syndrome (associated with enteral or parenteral nutritional supplementation after starvation or malnutrition) ■ Respiratory alkalosis ■ Treatment of diabetic ketoacidosis with insulin administration

may also cause impaired oxygen delivery, shortened platelet survival time, weakness, pain (associated with rhabdomyolysis), vomiting, and proximal tubule bicarbonate wasting.

Hypophosphatemia should be anticipated in susceptible patients (ie, receiving insulin for diabetic ketoacidosis, at risk for refeeding syndrome). In these patients, phosphorous levels should be monitored closely or supplemented.

When hypophosphatemia develops, phosphorus should be supplemented in symptomatic patients *or* asymptomatic patients at risk for symptomatic hypophosphatemia. Phosphorus is best replaced by potassium phosphate CRI at 0.01 to 0.06 mmol/kg/hr IV mixed in saline or dextrose. Discontinue phosphorus supplementation when serum concentration normalizes. Monitor serum phosphorus concentration closely after discontinuing supplementation. Levels should be rechecked q6h to avoid oversupplementation, which may lead to hypocalcemia, soft tissue mineralization, hyperphosphatemia, or acute kidney injury. Oral phosphorus supplementation may be provided, although it is generally ineffective in vomiting patients or those with diarrhea. The underlying cause of hypophosphatemia should be investigated and addressed.

Hyperphosphatemia

Hyperphosphatemia occurs when serum phosphorus concentrations are greater than 6.5 mg/dL, but reference ranges may vary; like hypophosphatemia, the causes can vary (see **Causes of Hyperphosphatemia**). Increases in phosphorous concentration usually lead to decreased calcium concentration. Hyperphosphatemia may lead to hypocalcemia and soft tissue mineralization. Risk for soft tissue mineralization is higher when the calcium phosphorous ($Ca \times PO_4$) product is greater than 70.

IV fluid therapy usually decreases circulating phosphorus levels by increasing the glomerular filtration rate, hence phosphorus excretion, preferred for vomiting and/or anorexic patients. All oral sources of phosphorus should be evaluated and adjusted. Dietary phosphorus restriction should be initiated (usually with a protein-restricted diet [also low in phosphorus]). In patients with chronic renal insufficiency, intestinal phosphate absorption should be reduced by administration of oral phosphate binders with a meal. Common phosphate binders include aluminum hydroxide, calcium carbonate, and calcium acetate.

Causes of Hyperphosphatemia

Decreased excretion	■ Renal insufficiency ■ Hypoparathyroidism ■ Prerenal or postrenal disease
Increased intake	■ Hyperthyroidism phosphate enemas ■ IV phosphorus supplementation ■ Vitamin D intoxication
Laboratory error	
Physiologic cause	■ Growing animals
Translocation	■ Hemolysis ■ Metabolic acidosis ■ Rhabdomyolysis ■ Tumor lysis syndrome
Physiologic cause	■ Growing animals

Magnesium

Magnesium is an intracellular cation found primarily in bone and muscle; only about 1% is located extracellularly. Extracellular magnesium is present in 3 forms: an ionized form that is biologically active, a protein-bound form, and a complex form. Magnesium is regulated in the GI tract and kidneys under the influence of parathyroid hormone and 1,25-dihydroxycholecalciferol. Increased morbidity and mortality can occur in critically ill patients with altered serum magnesium concentrations.

Hypomagnesemia

Although the diagnosis of hypomagnesemia remains controversial and challenging, total and ionized magnesium levels can help evaluate patients with low magnesium concentrations. Total magnesium evaluation is widely available in reference laboratories, while ionized magnesium analysis is less common. Low total serum magnesium is usually significant in patients at risk for hypomagnesemia. However, total serum magnesium concentrations can be normal in patients with total body magnesium deficits.

Hypomagnesemia, commonly caused by depletion of total body stores of magnesium (see **Causes of Hypomagnesemia**), has been documented in critically ill dogs and associated with prolonged hospitalization and increased mortality.[1] Low magnesium may lead to cardiac arrhythmias (atrial fibrillation, supraventricular tachycardia, torsade de pointes), cardiac necrosis, systemic hypertension, dysphagia, and tetany. Hypomagnesemia may also cause refractory hypokalemia, hyponatremia, and hypocalcemia. Patients with concurrent refractory electrolyte abnormalities magnesium supplementation.

Causes of Hypomagnesemia

Changes in distribution	■ Administration of insulin ■ Idiopathic ■ Massive blood transfusion ■ Pancreatitis ■ Sepsis ■ Shock
Increased intake of magnesium	■ Inadequate nutrition
Increased loss of magnesium	■ Administration of aminoglycosides ■ Chronic diarrhea ■ Diabetic ketoacidosis ■ Hyperthyroidism ■ Inflammatory bowel disease ■ Postobstructive diuresis ■ Renal tubular acidosis
Laboratory error	

Supplementation should be considered in at-risk patients with total magnesium concentrations of less than 1.5 mg/dL or if any signs consistent with hypomagnesemia are present. Patients requiring magnesium supplementation are typically treated with parenteral infusion of magnesium salts (magnesium chloride or sulfate) at 0.3 to 1 mEq/kg/q24h IV. The dose should be reduced in patients with underlying renal disease and cardiac conduction abnormalities.

Hypermagnesemia

Hypermagnesemia, not as clinically significant or as common as hypomagnesemiais is commonly caused by renal insufficiency and pre- or postrenal azotemia. Recommended doses for magnesium supplementation are safe and rarely cause hypermagnesemia in veterinary patients. However, all patients supplemented with magnesium should be observed for changes in blood pressure or alterations on ECG.

Clinical signs associated with hypermagnesemia in humans include hypoventilation, hypotension, cutaneous flushing, and alterations in cardiac contractions. Hypermagnesemia, relatively easy to diagnose, is characterized by increased total serum magnesium or ionized magnesium concentration. Any value higher than laboratory reference range for magnesium suggests increased total body magnesium concentration.

Patients with hypermagnesemia should have discontinued parenteral magnesium supplementation, IV diuresis should be initiated (0.9% NaCl), and dialysis may be considered in patients with significant renal insufficiency. Calcium gluconate 10% (0.5–1.5 mL/kg IV over 10–15 min) may be administered to antagonize some of the effects of hypermagnesemia in patients with cardiac abnormalities. ■ cb

See **Aids & Resources**, back page, for references & suggested reading.