



Case Report

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A Case of Severe Acute Hyperphosphatemia Secondary to Ingestion of Phosphoric Acid Solution Treated with Continuous Renal Replacement Therapy

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Abstract

We report a case of severe acute hyperphosphatemia with serum phosphate levels > 12.92 mmol/L (above laboratory detection levels) in a Filipino gentleman from ingestion of phosphoric acid solution. He also developed hypocalcemia, metabolic acidosis and acute kidney injury. He was treated with continuous renal replacement therapy. His hyperphosphatemia and electrolyte abnormalities improved with dialysis but he subsequently demised due to associated complications from severe caustic injury.

Keywords

Hyperphosphatemia, Continuous replacement therapy, Phosphoric acid solution ingestion

Introduction

Acute hyperphosphatemia due to phosphate load is typically attributed to sodium-phosphate laxatives, tumor lysis syndrome or rhabdomyolysis. It can lead to acute renal failure secondary to acute phosphate nephropathy and in addition cause hypocalcaemic tetany, cardiac arrhythmia and arrest. Treatment usually involves cessation of the offending agent, hydration to enhance renal excretion, oral phosphate binders, and hemodialysis if such measures are not successful [1].

We report a case of severe acute hyperphosphatemia with serum phosphate levels > 12.92 mmol/L (above laboratory detection levels) secondary to ingestion of phosphoric acid based cleaner in an attempt at deliberate self-harm. We are not aware of any cases of acute hyperphosphatemia of such severity in patients with normal renal function, treated with continuous renal replacement therapy that achieved resolution of the electrolyte and metabolic abnormalities.

Case Report

A 41-year-old Filipino gentleman presented to the emergency department (ED) with generalized abdominal pain after intentional ingestion of approximately 100-150 ml of Maintex Lime Go Restroom Cleaner, a phosphoric acid based cleaner, in an attempt at deliberate self-harm. He also reported throat pain with hematemesis. He did not have any significant past medical history and was not on any chronic medications.

On arrival to the emergency department, the patient was alert. On examination, he had a pulse rate of 97 beats per minute and a blood pressure of 123/88 mmHg. He was tachypneic with a respiratory rate of 29 breaths per minute. His posterior pharyngeal wall was noted to be erythematous. Abdominal examination revealed generalized tenderness without guarding or rebound. Bowel sounds were present but sluggish. Rectal examination revealed dark coffee ground stools. Cardiovascular and respiratory examination was unremarkable (Table 1).

The admission electrocardiogram (ECG) and chest X-ray were unremarkable.

Our clinical impression was that of acute hyperphosphatemia secondary to phosphoric acid solution ingestion with hypocalcemia, HAGMA from exogenous acid ingestion and lactic acidosis. There were also concerns of gastrointestinal bleed.

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Table 1: Early biochemical evaluation revealed the following results

	Value	Reference Range
Phosphate	> 12.92 mmol/L (above laboratory detection levels)	0.94-1.50 mmol/L
(Corrected) Calcium	1.98 mmol/L	2.09-2.45 mmol/L
Creatinine	102 µmol/L	50-104 µmol/L
Urea	3.5 mmol/L	2.7-6.9 mmol/L
Potassium	5.4 mmol/L (hemolyzed sample)	3.5-5.1 mmol/L
Sodium	139 mmol/L	136-146 mmol/L
Chloride	95 mmol/L	98-107 mmol/L
Bicarbonate	5.7 mmol/L	10-29 mmol/L
Lactate	3.3 mmol/L	0.5-2.2 mmol/L
Alanine Transaminase	14 U/L	6-66 U/L
Aspartate Transaminase	29 U/L	12-42 U/L
pH	7.007 (Anion Gap 38.3, Delta bicarbonate 26.3 suggestive of high anion gap metabolic acidosis (HAGMA))	7.35-7.45
pCO ₂ (Venous)	41.5 mmHg	35-35 mmHg
Prothrombin time (PT)	13.0s	9.9-11.4 seconds
aPTT	45.2s	25.7-32.9 seconds
International normalized ratio (INR)	1.26	
Haemoglobin	16.7 g/deciliter (dL)	14-18 g/deciliter (dL)
White blood cell	31.20 × 10 ⁹ /L (Predominant neutrophilia)	4-10 × 10 ⁹ /L
Paracetamol levels	Not detected (< 5 mg/L)	10-30 mg/L
Salicylate levels	Not detected (< 0.02 mmol/L)	0.22-2.17 mmol/L

We decided to initiate intermittent hemodialysis (IHD) in view of severe metabolic acidosis. Shortly upon admission to the intensive care unit (ICU), the patient developed bradycardia, which led to apulseless electrical activity (PEA) collapse. Cardiopulmonary resuscitation was initiated immediately. He was intubated and mechanically ventilated. He was started on continuous veno-venous hemodiafiltration (CVVHDF) without anticoagulation in view of hemodynamic instability.

His CVVHDF effluent dose was prescribed according to the international guidelines and divided equally between dialysate and replacement fluid. The filter clotted once because of high transmembrane pressure (TMP). His filtration fraction was also elevated. Regional citrate anticoagulation was not ideal in this situation in view of hyperlactemia and hypocalcemia. Hence, CVVHDF was changed to continuous veno-venous hemodialysis (CVVHD), to reduce the risks of further episodes of filter clotting that could potentially delay treatment.

After 4 days of CRRT (CVVHDF and CVVHD), patient's phosphate level dropped to 1.36 mmol/L (Figure 1). Hypocalcemia also resolved with a corrected calcium level of 2.21 mmol/L (Figure 2). His metabolic acidosis, predominantly secondary to lactic acidosis, attributed to hypoperfusion from hemodynamic instability and acute liver failure, improved after 5 days on CRRT. However, he continued to have anuric acute kidney injury, which was attributed to acute phosphate nephropathy, septic and ischemic acute tubular

necrosis and required CRRT throughout his admission.

Despite improvement in his electrolyte and metabolic abnormalities, he continued to be haemodynamically unstable requiring inotropic support. The patient subsequently demised from other medical issues including hemorrhagic shock secondary to a bleeding duodenal ulcer because of ischemic necrosis, and acute liver failure secondary to ischemic hepatitis. He also had possible intestinal ischemia and ventilator associated pneumonia (VAP) which may have contributed to his demise.

Discussion

Hyperphosphatemia attributed to an acute phosphate load occurs typically due to oral sodium-phosphate laxatives and has been well documented to cause acute phosphate nephropathy and nephrocalcinosis. Its proposed mechanisms include hypovolemia, a large phosphate load at the distal nephron with calcium phosphate precipitation [1]. The symptoms and signs associated with hyperphosphatemia are usually secondary to the associated hypocalcemia. This may include muscle cramps, tetany, paresthesia, seizures, coma, hypotension and arrhythmias. Additionally, acidosis can cause intracellular phosphate to shift extracellularly, increasing urinary losses, further increasing calcium phosphate precipitation.

Reported treatment strategies are varied. Acute extracellular fluid expansion with IV fluids can help increase

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