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Case Report

Hypophosphatemia in Intensive Care Unit: An Unusual Cause of Respiratory Failure (About a Case)

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Abstract

Hypophosphatemia is frequently observed in critically ill patients and it is related to increased mortality and morbidity. A 31-year-old patient was admitted to our ICU for ketoacidosis, she developed an acute respiratory distress and tetraparesia despite of normal glycemic values. The unusual cause of respiratory failure was a severe hypophosphatemia 0.32 mmol/l. The correction of the metabolic acidosis by insulin therapy resulted in intracellular penetration of phosphate causing severe hypophosphatemia responsible for acute respiratory distress and nervous dysfunction. This case provides an opportunity for reviewing the main causes and consequences of hypophosphatemia, and for emphaising the value of monitoring serum phosphate levels and providing supplemental phosphate in ICU patients at risk for phosphate depletion.

Keywords: *Hypophosphatemia; Intensive Care Unit; Respiratory Failure*

Introduction

Hypophosphatemia is a metabolic disorder that is commonly encountered in critically ill patients. It is defined as plasma phosphate level below 0.80 mmol per litre (mmol/L), and can be further divided into subgroups of mild (a plasma phosphate of 0.66 to 0.79 mmol/L), moderate (plasma phosphate of 0.32 to 0.65 mmol/L) and severe (plasma phosphate of less than 0.32 mmol/L).Inadequate intake, redistribution of phosphate into cells and loss of phosphate from the body are the main mechanisms related to the occurrence. Phosphate has many roles in physiological functions thus the depletion of serum phosphate could lead to impairment in multiple organ systems, which include respiratory system, cardiovascular system, neurological system, and muscular system. In critically ill patients, factors such as sepsis, trauma, fluid therapy, refeeding syndrome, acid-base disorders, medications and renal replacement could contribute to the occurrence of hypophosphatemia. We report the case of a diabetic patient, who presented an acute respiratory distress and tetraparesis secondary to hypophosphatemia, requiring intubation and mechanical ventilation.

Case Report

A 31-year-old woman known as an insulin-dependent diabetic, admitted to our unit for diabetic ketoacidosis. at admission, she was obnibulated with a Glascow score at 13, she was hemodynamically stable, but tacchycardia at 110 beats per minute, she was polypnic at 40 cycles per minute, the saturation under 3litres of oxygen was 98 %. glucosuria and ketonuria were present. the laboratory test showed a blood glucose level of 65.3 mmol/l, a natremia of 122 mmol/l (natremia corrected to 140 mmol/l), a potassium content of 3.20 mmol/l and an osmolarities of 330 mosmol/l. gasometry showed plasma pH at 7.10 (PaO_2: 90 mmHg, $PaCO_2$: 48 mmHg, HCO 3 -: 16 mmol/l). Cytobacteriological examination of the urine had noted leukocyturia at 30.10 ^ 3 with candida albicans. the diagnosis

of acidocetotic decompensation promoted by the urinary tract infection was taken. The initial management involved a 6-liter filling of crystalloids in 12 hours with a potassium chloride injection in an electric syringe at a rate of 1 g per hour, with insulin therapy according to the capillary glycemia, and fluconazole (400 mg all 12 hours) intravenously. the evolution after 24 hours of hospitalization was marked by a neurological improvement and glycemic equilibrium. On the third day of her hospitalization, the patient suddenly had a respiratory distress with tetraparesis requiring intubation and mechanical ventilation, using catecholamines following hemodynamic instability. Gasometry noted respiratory acidosis (ph 7.20, PaO_2: 90mmhg, $PaCO_2$: 52 mmhg, hco3: 22 mmol/l). The blood ionogram then showed a blood glucose level of 18 mmol/l, a kaliemia of 2.5 mmol/l, a calcemia a 3.5 mmol/l and a phosphoremia at 0.32 mmol/l and then below the detection threshold 8 hours after intubation. KPCs were normal. The hypothesis of deep hypophosphoremia associated with hypokalemia at the origin of respiratory decompensation was then most likely. The patient died 12 hours after intubation following a cardiac arrest that failed to respond to resuscitation measures.

Discussion

Symptoms of severe hypophosphatemia may mimic symptoms of associated underlying disease and therefore may not be recognized in a critically ill patient. Common manifestations include nausea, weakness, malaise, numbness, irritability, cardiomyopathy, confusion, convulsions, respiratory failure, rhabdomyolysis, and hemolytic anemia [1]. The phosphate-depletion neuromuscular syndrome in man was first described in 1968. Furthermore, in patients who develop respiratory insufficiency, hypophosphatemia adversely affects diaphragmatic function [2]. The muscle weakness may arise from derangements seen in other systems. In the red cells, as the phosphate decreases, the concentrations of ATP and [3,4] diphosphoglycerate are all decreased, which may be implicated in muscle injury. The decreased level of [3,4] diphosphoglycerate causes a leftward shift of the oxygen dissociation curve and impairs oxygen release.

Hypophosphatemic patients often are hypokalemic and hypomagnesemic and these disorders should be corrected as well. Severe hypophosphatemia is commonly seen in patients receiving treatment for diabetic ketoacidosis. Some patients even develop hypophosphatemia before treatment as described earlier. Multiple studies show an association between hypophosphatemia and increased mortality [5]. Severe hypophosphatemia has been reported to predict up to eightfold increased mortality rate in sepsis patients [5].

With the high prevalence of hypophosphatemia in critically ill patients, as well as their susceptibility to life-threatening symptoms, frequent laboratory monitoring is recommended, especially in previously mentioned high-risk groups. It is generally recommended to correct hypophosphatemia in hypophosphatemic patients with associated symptoms [3].

Correction of hypophosphatemia is possible via oral or intravenous routes. Intravenous administration of phosphate is not without complications, though. Phosphate may precipitate with calcium. Large intravenous doses of phosphate may result in hyperphosphatemia, hypomagnesemia, hypocalcemia, and hypotension. It is therefore necessary to know when intravenous therapy is indicated, and how much and how fast phosphate should be supplied [6-12].

Conclusion

Critically ill patients have a high prevalence of hypophosphatemia because of the presence of multiple causal factors. Hypophosphatemia may lead to a multitude of symptoms, but most often remains asymptomatic. Additional studies must be required to address the current approach to hypophosphatemia in critically ill patients, as well as the association of hypophosphatemia with morbidity and mortality, and the effect of the correction of this electrolyte disorder.

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