ASIAN JOURNAL OF PHARMACEUTICAL AND CLINICAL RESEARCH

INNOVARE ACADEMIC SCIENCES
Knowledge to Innovation

Vol 9, Issue 4, 2016

Online - 2455-3891
Print - 0974-2441

Case Report

A CASE REPORT ON ENTERIC FEVER INDUCED HYPOPHOSPHATEMIA

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Received: 23 March 2016, Revised and Accepted: 09 May 2016

ABSTRACT

Hypophosphatemia is critical ill patients are a common entity which causes unnoticed most of the time. This can lead to considerable morbidity and in some cases contribute to mortality. A 20-year-old female patient presented to Emergency Medicine with complaints of diarrhea, breathing difficulty, and tachypnea. Her routine electrolyte investigation showed low phosphate level. Once hypophosphatemia was corrected with intravenous phosphorous, patient became symptomatically better. In this study, we encounter that enteric fever can also be a factor for symptomatic hypophosphatemia and it should be corrected even if it is mild. Therefore, this case emphasis on importance of correcting symptomatic hypophosphatemia in critically ill patients.

Keywords: Enteric fever, Hypophosphatemia, Emergency medicine.

INTRODUCTION

Phosphorus is an essential element, around 80% is found in bone and teeth. It has an important role in acid-base hemostasis, cellular and subcellular metabolism, and maintaining cell structure [1]. Phosphate is the source of adenosine tri phosphate which fuels cell functioning and controls the level of 2,3-diphosphoglycerate present in the red blood cell [2]. The normal value of phosphorus is 2.5-4.5 mg/dl. Hypophosphatemia is defined as phosphorus level <2.5 mg/dl, and the overall incidence of hypophosphatemia is about 2-3%. Hypophosphatemia can be due to three mechanism – decreased intestinal absorption, increased renal excretion or internal redistribution of inorganic phosphate [3,4]. In certain conditions like alcoholism and diabetic ketoacidosis, hypophosphatemia was observed because of shift of phosphorus within the body.

Hypophosphatemia is classified by severity as mild, moderate and severe. Mild hypophosphatemia is a phosphorus level of 2-2.5 mg/dl. Moderate hypophosphatemia is a level of 1.0-2.0 mg/dl and severe hypophosphatemia is a level less than 1.0 mg/dl [5]. Mild to moderately severe hypophosphatemia is usually asymptomatic. If severe hypophosphatemia is present for longer than 2-3 days, serious complications can be seen including rhabdomyolysis, respiratory failure, acute hemolytic anemia, and fatal arrhythmias [6]. It is, therefore, necessary to correct its level by administration of phosphate. Phosphate can be administered either orally or intravenously. Oral administration in critically ill patients would be ineffective due to malabsorption associated with the illness. Therefore, preferred route is intravenously. It is also evident that addition of phosphate to the replacement and dialysate solution during continuous renal replacement therapy could help to reduce the incidence of hypophosphatemia [7].

CASE REPORT

A 22-year-old female with no known comorbidities came with complaints of diarrhea since 1-week after a food intake from outside along with fever and multiple episodes of vomiting. Initially, she passed greenish watery loose stools 3-4 times a day for which she took medications, but symptoms aggravated and had more than 11 episodes of loose stools for 2 days. She had no history of hemoptysis, hematemesis, melena, and abdominal distension. Subsequently, she developed breathlessness. She was admitted in a local hospital. Culture report showed growth of salmonella typhi and was started on ceftriaxone there. She was referred to our center for further management.

On admission, the patient was tachypneic with a saturation of 88% in room air. Her clinical investigation showed P/A: Soft, generalized tenderness+, Respiratory system: Within normal limits, and partial pressure of oxygen (PaO_2)/Fraction of inspired oxygen (FiO_2)=250. Arterial blood gas showing mild respiratory alkalosis and hypoxia. Her initial blood investigation showed C-reactive protein: 193 mg/l, altered liver function test with serum glutamic oxaloacetic transaminases/serum glutamic pyruvic transaminases: 116.3/510.6 IU. Widal and leptospiral immunoglobulin M antibody test were positive. On her electrolyte investigations her serum phosphate level was found to be low (1.6 mg/dl).

This was corrected by giving intravenous phosphorus 12 mmol in 200 ml normal saline over 4 hrs as per guideline on the post-admission day 1 and 2. Her serum phosphate level improved on the 3rd day. Once hypophosphatemia was corrected her breathing difficulty and tachypnea resolved.

As the patient was symptomatically better on the 5th day, she was shifted to ward. She was later discharged on a stable condition and was advised to take phosphorus rich foods including milk and green leafy vegetables (Table 1).

DISCUSSION

Enteric fever is an infection caused by either typhoid bacteria (*Salmonella enterica* serotype typhi) or paratyphoid bacteria. Typhoid is a febrile illness with onset of symptoms 5-21 days after ingestion of the causative microorganism contaminated food or water. The disease condition varies depending on host factors such as age, gastric acidity, and immunological status. Here, the patient had watery diarrhea along with enteric fever which led to probable malabsorption. Decreased intestinal absorption due to secretary diarrhea can probably explain hypophosphatemia in this patient. Hypophosphatemia behaves like a general marker of illness severity and not as an independent predictor of intensive care unit or in hospital mortality in critically ill patients [8]. Here, the only reason for tachypnea was hypophosphatemia after ruling out all other possibilities. Once this hypophosphatemia was corrected she improved and her respiratory rate also became normal. This highlights the importance of phosphorus which resulted in respiratory muscle weakness and fatigue. In most of the settings, hypophosphatemia

Table 1: Serum phosphate level on post admission days

Days	Serum phosphate (mg/dl)
1st post admission day	1.6
2nd post admission day	1.73
3rd post admission day	2.1
5th post admission day	3.5

goes undetected and even if it is detected it is not corrected. Since this patient is having malabsorption, oral route phosphate absorption plays no role. Therefore, intravenous correction is done.

CONCLUSION

Hypophosphatemia is a common problem in critically ill patients and even more prevalent in those who are given specialized nutritional support. With the existing studies conducted, we noticed that hypophosphatemia is associated with sepsis, trauma, fluid therapy, severe respiratory disease, etc. Through this study, we encounter that enteric fever can also be a factor for hypophosphatemia. Therefore even mild hypophosphatemia, if it is symptomatic, it should be corrected. In critically ill patients, intravenous corrections have shown promising results over oral corrections.

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