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CASE REPORT

Isolated Hypophosphataemia Mimicking Cerebrovascular Accident

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ABSTRACT

Hypophosphataemia occurs in an abnormally low serum phosphate level. Three main mechanisms are postulated: decreased intestinal absorption, increased renal excretion, and extracellular shifts to intracellular compartments. It is potentially a fatal disease if not intervene. The management is merely treating the underlying disorder, giving phosphate supplement and requiring close biochemical monitoring. The incidence of symptomatic isolated hypophosphataemia is extremely rare. In this case report, a 33-year-old man presented with three days history of dysphagia, inability to complete sentences and generalized muscle weakness. He developed blurred vision especially upon exposure to bright light. He had a history of single parathyroidectomy for parathyroid adenoma 2 years ago. Physical examinations were unremarkable. Laboratory investigations were normal except for phosphate level of 0.30 mmol/L. Intravenous KH_2PO_4 with a dosage of 10 mmol was administered in slow bolus in 3 hours. His symptoms resolved slowly after correction. Although isolated hypophosphataemia is rare but need to recognize the symptoms and signs of hypophosphataemia and treat accordingly.

INTRODUCTION

Phosphate is the most abundant intracellular anion in the body. It is essential for several biological processes including energy production, formation of cellular structure, and tissue repair. Hypophosphataemia develops when there is an abnormally low level of phosphate in the blood. It is often missed due

to non-specific features which can lead to a considerable morbidity as well as mortality if neglected. It is imperative to obtain a thorough history with complete physical examination in achieving an accurate diagnosis. Although it is rare, the incidence of hypophosphataemia can occur in as many as 3% of hospitalized patients and as high as 30% of patients admitted to intensive care unit[1].

Mild and moderate hypophosphataemia are usually asymptomatic. Major clinical sequelae usually occur only in severe hypophosphataemia. Fortunately, this extreme form is very unusual especially in hospitalized patients. Hypophosphataemia in hospitalized patients has many causative factors. These include alcoholism, gram-negative sepsis, hyperalimentation, diabetic ketoacidosis, primary hyperparathyroidism, gastrointestinal malabsorption, as well as medications such as diuretics, insulin, corticosteroids, phosphate-binding antacid and epinephrine[2]. In this case report, we highlight the case of symptomatic isolated hypophosphataemia.

CASE PRESENTATION

A 33-year-old man presented to emergency department with three days history of dysphagia, inability to complete sentences and generalized muscle weakness. He developed blurred vision especially upon exposure to bright light. Upon further questioning, he was in a motivation camp a week prior to this presentation. He claims to develop reduced oral intake during the camp. He had a history of single parathyroidectomy for parathyroid adenoma 2 years ago. The surgery was eventful though it was complicated with transient hypocalcaemia. However, he defaulted his subsequent follow-up. Fortunately, he did not develop any electrolytes imbalance.

On examination, he was alert and conscious. His vital signs were stable. He had difficulty to speak which clinically was consistent with dysarthria. Surprisingly, the systematic reviews were unremarkable with intact neurological examinations. Arterial blood gases and electrocardiogram was normal. Laboratory investigations were unremarkable except for phosphate level of 0.30 mmol/L.

In view of his unusual presentation, he was planned for computed tomography (CT) of the brain to exclude cerebrovascular disease which was negative. A decision of phosphate correction was made due to isolated phosphate abnormality. Intravenous KH_2PO_4 with a dosage of 10 mmol was administered in slow bolus in 3 hours. His symptoms resolved slowly after correction. Repeat phosphate level was 0.51 mmol/L. He was discharged with early follow-up at nearby clinic for phosphate monitoring.

DISCUSSION

Hypophosphataemia usually occurs in response to the reduced function affected by systemic conditions. This most likely happened to the patient due to a decreased intestinal absorption caused by dietary restriction during camping that he went a week before onset. The categorization of hypophosphataemia can be determined biochemically with its level. It is divided into mild (0.6 – 0.8 mmol/L), moderate (0.3 – 0.6 mmol/L) and severe (<0.3 mmol/L) form[3].

It is important to consider the possibility of an isolated event in view of unequivocal findings. Body weakness is common but it can affect any specific muscles. Other symptoms include dysphagia, diplopia, and dysarthria. Although patient developed dysphagia, the short duration negates the possibility of mechanical or functional oesophageal diseases. Hence, a central neuromuscular cause is suspected. It is essential to remember that various presentations of neuromuscular dysfunction can occur. They include a simple

paraesthesia and profound mental alteration. Hypophastaemia has been reported involving respiratory muscle paralysis which mimics Guillain-Barre syndrome[4]. Another case reported a child presented with diabetic ketoacidosis (DKA) and seizure which was not resolved with optimization of the endocrine function. The child was proved to developed hypophosphataemia related seizure rather than being affected by the DKA status[5]. A systematic review by Ariyoshi N et al. reported a negative relationship between severe hypophosphataemia and left ventricular dysfunction leading to cardiomyopathy and arrhythmias. Consequently, patient may also present with failure symptoms which may posed a challenge especially in ischemic heart disease patient[6].

Hypophosphataemia does not automatically mean that replacement therapy is indicated. Recognizing and establishing the cause of the hypophosphataemia can give proper guidance on how we are going to treat the patient. Sometimes treating the underlying causes can automatically correct the phosphate level[7]. Asymptomatic patient with moderate hypophosphataemia may be considered for oral phosphate supplementation provided that the enteral route is feasible. Oral phosphate supplement can be given at the usual dose of 500 mg BD. Intravenous phosphate medication is requisite in patients with severe hypophosphataemia, symptomatic moderate hypophosphataemia, and none feasible enteral route. Either potassium phosphate or sodium phosphate injection may be used for replacement. The recommended regime involves administration of 0.08 mmol/kg per body weight over 6 hours for severe hypophosphataemia without obvious clinical manifestation while 0.16 mmol/kg per body weight infused over 2 – 6 hours in life threatening condition[8]. Oral therapy is safer compared to intravenous route. Rapid correction is safe but the magnitude of response can be unpredictable. The faster the therapy, the more likely the side effects will occur.

CONCLUSION

Although it is rare, recognizing symptoms and signs of hypophosphataemia is crucial after ruling out other alternative diagnoses. In this patient, we have decided to treat the hypophosphataemia, with resultant resolution of symptoms.

CONFLICT OF INTEREST

The authors declare that they have no competing interests in publishing this case.

CONSENTS

Written informed consent was obtained from the patient to publish the case. A copy of written consent is available for review by the Chief Editor.

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