Case Report / Olgu Sunumu

A Rare Cause of Paresthesia: Hypophosphatemia

Nadir Bir Parestezi Nedeni: Hipofosfatemi

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Abstract

Phosphate is a structural molecule for cells and also is used as coenzyme or as seconder messenger. Renal or gastrointestinal loss of phosphate, diabetes mellitus, chronic alcoholism, hyperparathyroidism, sepsis, increased glucocorticoid, diuretics and antacids may cause hypophosphatemia. Muscle weakness, paresthesia, confusion, convulsion, tremor and coma are neurological symptoms of hypophosphatemia. Main clinical signs occur due to deterioration oxygen distribution and reduced intracellular adenosine triphosphate. In the treatment of hypophosphatemia identification of underlying causes is important. In this article, a 26-year-old young male patient with paresthesia that is caused by hypophosphatemia due to D vitamin deficiency is reported. Clinicians must be on the alert about phosphate imbalance which is seen more rare than other electrolytes when investigation of patients with paresis and/or paresthesia.

Keywords
Hipofosfatemi, parestezi, D vitamin eksikliği

Anahtar Kelimeler
Hypophosphatemia, paresthesia, D vitamine deficiency

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Öz

Fosfor hücreler için yapısal bir molekül olup ayrıca koenzim ya da ikincil haberci olarak kullanılmaktadır. Fosfatın renal ve gastrointestinal kayıpları, diyabetis mellitus, hiperparatiroidizm, kronik alkolizm, sepsis, glukokortikoid fazlalığı, diüretikler ve antiasitler hipofosfatemiye neden olabilir. Kas güçsüzlüğü, parestezi, konfüzyon, nöbet, tremor ve koma hipofosfateminin nörolojik bulgularındandır. Temel klinik bulgular dokulara oksijen dağılımındaki bozulmaya ve hücre içi adenozin trifosfatın azalmasına bağlı olarak gelişir. Hipofosfateminin tedavisinde altta yatan nedenin belirlenmesi önemlidir. Bu yazıda D vitamini eksikliğine bağlı hipofosfateminin neden olduğu parestezili 26 yaşındaki genç erkek hasta sunulmuştur. Klinisyenler parezi ve/veya parestezili hastaları incelerken diğer elektrolitlere göre daha nadir görülen fosfor bozuklukları hakkında dikkatli olmalıdır.

Introduction

Phosphate is an electrolyte playing a role in the regulation of calcium levels, arrangement of carbohydrate and lipid metabolism, and maintaining acid-base balance. Hypophosphatemia may develop as a result of renal loss, chronic alcoholism, diabetes, burns, gastrointestinal loss, diuretics, and antiacids. Muscle weakness, paresthesia, confusion,

seizure, tremor, and coma are neurological symptoms of hypophosphatemia. Identification of the underlying cause is an important step for determining the etiology of hypophosphatemia. The treatment includes oral or intravenous replacement of phosphate. In this paper a 26-year-old male patient who was admitted to emergency with paresthesia due to vitamin D deficiency induced hypophosphatemia.

Case Report

A 26-year-old man who presented with numbness in all four extremities was evaluated. The symptoms began at the distal of extremities and added upwards and with increasing intensity in the last 2 weeks. He additionally had myalgia. He had no history of smoking or alcohol use, nor he had any systemic disease. He also had no familial history of any disease. On neurological examination he was conscious, and had orientation, cooperation. His pupils were isocoric; his light reflexes were bilaterally normal; his eye movements were unrestricted in every direction; he had no nystagmus or facial asymmetry; and he had intact lower cranial nerves and motor function. However, he had diffuse patchy paresthesia on sensory testing; his deep tendon reflexes were hypoactive; and his plantar responses were bilaterally flexor. In laboratory tests, serum glucose, hemoglobin, biochemical parameters (urea, creatinine, sodium, potassium, calcium, chloride, magnesium, ALT, AST and thyroid function tests, and vitamin B12 and folic acid levels) were within normal ranges.

The phosphate level was 1.3 mg/dL (normal range 2.5-4.5 mg/dL). A search for the cause of hypophosphatemia found normal parathormone and glucocorticoid hormone levels and a low vitamin D level (5.7 ng/dL; normal range 20-120 ng/dL). Vitamin D deficiency induced hypophosphatemia was considered as the primary diagnosis. The patient's neurological examination completely returned to normal within 72 hours of phosphate replacement. A search for the cause of vitamin D deficiency revealed no signs of symptoms of nutritional deficiency, intestinal malabsorbtion (celiac disease, crohn disease, cystic fibrosis, gastric or intestinal perforation), hepatic or renal disorders, abnormal hepatic or renal function tests, or any malaborbtion syndrome affecting vitamin B12 or folic acid levels. He also lacked any history of the use of medications preventing vitamin D absorbtion, including corticosteroids, orlistat, cholestiramine, or phenytoin. There was also no history of sunlight avoidance causing deficient sunlight exposure. The patients was also administered treatment against vitamin D deficiency.

Discussion

Phosphate is a structural molecule for human cells which is also used as a coenzyme or second messenger. Human body normally contains 600-700 mg phosphate, and approximately 1000 mg phosphate is consumed daily. Absorbtion of phosphate is the function of proximal tubule of the intestines. Phosphate is predominantly excreted in urine but some amount also in stool. Phosphate balance is regulated by vitamin D, parathormone, acid-base balance, and glucocorticoids (1). Healthy individuals have a phosphate level of 2.5-4.5 mg/dL.

The main mechanisms leading to hypophosphatemia (serum phosphate level <2.5 mg/dL) include phosphate shift from the extracellular compartment to the intracellular compartment, reduced phosphate intake, reduced phosphate absorbtion from the intestines, and increased urinary phosphate excretion (2). Hypophosphatemia may develop with glucose, fructose, and aminoacid infusion or intravenous hyperalimentation. Hypophosphatemia is seen a rate of 1-5% in hospitalization. Sepsis, diabetic ketoacidosis, liver failure, and post-renal transplant period are particularly risk for hypophosphatemia, and patients with these conditions should be more closely monitored. Our patient was, however, a young adult without any systemic disorder or a marked risk factor for hypophosphatemia.

Renal losses are among the most common causes of hypophosphatemia. Chronic alcoholism, malabsorbtion, burns, hypothermia, diabetes, renal losses, vitamin D deficiency, prolonged vomiting, glucocorticoid excess, hyperparathyroidism, antiacid, thiazide diuretic, and phosphate binding agent use are some major causes of hypophophatemia.

Hypophosphatemia is rarely symptomatic unless serum phosphate level is reduced below 2 mg/dL. Affected patients are typically asymptomatic, with signs and symptoms occurring with deep hypophosphatemia. Main clinical signs and symptoms emerge as a result of disturbed tissue oxygenization and reduced intracellular adenosine triphosphate

concentration (3-5). The patient's phosphate was 1.3 mg/dL which caused the symptoms.

The signs and symptoms may involve all organs and systems. The gastrointestinal effects (loss of appetite, nausea, vomiting, gastric atony), neurological symptoms (weakness, paresthesia, ataxia, tremor, seizure, coma), musculoskeletal signs and symptoms (myalgia, fractures, bone aches, osteomalasia), cardiac signs and symptoms (reduced myocardial contractility), and hematological disturbances (hemolytic anemia, thrombocyte dysfunction) may all be found. Mohseni et al. (6) defined acute hemiparesis in a patient with hypophosphatmeia. The patient's phosphate level was 1.3 mg/dL and displayed the symptoms of numbness, myalgia, and sign of diffuse paresthesia, and symmetrically reduced deep tendon reflexes. No sign related to other systems was noted.

A complete evaluation of phosphate level requires the measurement of parathormone, vitamin D, and glucocorticoid hormone levels. In cases with reduced phosphate intake vitamin D deficiency may cause hypophosphatemia by reducing gastrointestinal absorbtion. Our patient had normal parathormone and glucocorticoid hormone levels but reduced vitamin D level.

We considered that hypophosphatemia was caused by vitamin D deficiency. Appropriate phosphate replacement corrected phosphate level, and the correction of the underlying vitamin D deficiency prevented further renal phosphate loss.

Hypophosphatemia treatment should both aim at phosphate replacement and the identification of the underlying cause (7). We also started vitamin D along with phosphate replacement. In this paper we discussed the clinical and laboratory features of a young male patient with hypophosphatemia who presented to our clinic with diffuse paresthesia, dissussion with literature. We aimed to emphasize that calling to mind phosphate imbalance as a rare cause of paresia and paresthesia along with more common electrolyte disorders such as sodium, potassium, calcium, and magnesium deficiency.

Ethics

Informed Consent: The written informed consent was obtained from the patient for publication.

Peer-review: Internally peer-reviewed.

Authorship Contributions

Surgical and Medical Practices: Z.Ö.A., R.Ö., A.B., Concept: Z.Ö.A., R.Ö., A.B., Design: Z.Ö.A., R.Ö., A.B., Data Collection or Processing: Z.Ö.A., R.Ö., A.B., Analysis or Interpretation: Z.Ö.A., R.Ö., A.B., Literature Search: Z.Ö.A., R.Ö., A.B., Writing: Z.Ö.A., R.Ö., A.B.

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