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Case Report

Clay ingestion induced hypokalemic paralysis: a rare case report in an elderly female with severe iron deficiency anemia

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ABSTRACT

Hypokalemic paralysis is a rare neuromuscular disorder characterized by painless muscle weakness resulting from low potassium levels, most commonly presenting during adolescence. In this case report, we detail the presentation and management of an 80-year-old female who experienced sudden onset bilateral limb weakness. Initial clinical assessment revealed the absence of reflexes and hypotonia in all four limbs without any gastrointestinal or neurological symptoms. Comprehensive diagnostic evaluation identified severe iron deficiency anemia and an unusual history of clay ingestion due to pica as the underlying causes of her hypokalemic paralysis. Treatment included intravenous and oral potassium supplementation, as well as packed cell volume transfusions, leading to a significant improvement in her symptoms and normalization of her potassium levels. This case highlights the need to consider atypical etiologies, such as pica-induced clay ingestion, in the differential diagnosis of hypokalemic paralysis, particularly in elderly patients with nutritional deficiencies. Early recognition and appropriate management are critical for favourable outcomes.

Keywords: Hypokalemic paralysis, Clay ingestion, Pica, Iron deficiency anemia, Elderly patients

INTRODUCTION

Hypokalemic paralysis is a rare neuromuscular disorder characterized by transient episodes of muscle weakness due to decreased serum potassium levels. It commonly presents in late childhood or adolescence and is often associated with triggers such as heavy exercise, fasting, or high-carbohydrate meals.[1,2] We report a unique case of hypokalemic paralysis induced by clay ingestion in an elderly female with severe iron deficiency anemia.[3-5]

Despite the importance of potassium effects on peripheral nerve and nerve excitability, the biophysical basis of these effects is only partly understood. Since the resting potential depends primarily on the selective permeability of the axolemma to potassium ions, it is expected that hyperkalemia will cause membrane depolarization with a consequent increase in potassium permeability and membrane conductance, and thereby a 'fanning-in' of threshold electrotonus. This behaviour is well accounted for by a model of nerve excitability, in which myelinated axons are represented by two linked compartments (node and internode), with different assortments of ion channels following Hodgkin-Huxley equations.[6]

CASE REPORT

An 80-year-old female presented to the department of medicine with a three-day history of sudden onset bilateral limb weakness. She denied any preceding symptoms or significant medical history. On examination, she was found to have absent reflexes and hypotonia in all four limbs, with no bowel or bladder involvement, altered sensorium, vomiting, or diarrhea. Further history revealed a long-standing habit of clay ingestion due to pica, likely contributing to the electrolyte imbalance.

Table 1: Investigations of patient during hospitalization.

Investigations	Day-1	Day-2	Day-3	Day-5
Hemoglobin (12.0-15.0 g/dl)	5.9		7.5	8.3
White blood cells (4000-1000 cells/cumm)	10,200		9,800	9,200
Platelet (150,000-450,000/cumm)	420,000		380,000	394,000
Mean corpuscular volume (MCV 80-100 fl)	68			
Red-cell distribution width (RDW 11.5-14.5 %)	19.3			
Peripheral smear		Microcytic hypochromic red cell, normal white cell and platelet series		
Serum ferritin (24–336 ng/ml)	5.4			
Serum sodium (135-145 meq/liter)	136.8	140.6	138.6	144.4
Serum potassium (3.5-5.0 meq/liter)	1.9	2.4	3.6	4.2
Serum calcium (8.5-10.5 mg/dl)	8.9			
Serum magnesium (1.7-2.2 mg/dl)	1.89			
Spot urine sodium and potassium	Within normal limits			
Thyroid stimulating hormone (TSH 0.4-4.0 mu/l)		2.3		
Immunofluorescence ANA (normal <1:160 titer)			Negative	
Arterial blood gas	Ph: 7.37, bicarbonate: 22.7 meq/l			

Treatment and outcome and follow up

The patient was initially treated with parenteral potassium supplementation (40 meq in 500 ml normal saline infusion over 4 hours, 12 hourly) and 2 packed cell volume transfusions to address the underlying severe iron deficiency anemia. This was followed by oral potassium citrate syrup (POTCLOR 10 ml 8 hourly). Over the course of three days, the patient's muscle weakness gradually improved, and serum potassium levels normalized. Additionally, iron supplements and folic acid, oral B12, tablets were prescribed upon discharge to address the underlying iron deficiency anemia.

At a follow-up visit one-month post-discharge, the patient reported no recurrence of muscle weakness. Laboratory tests showed stable potassium levels and improved hemoglobin levels. The patient's adherence to dietary recommendations and supplementation regimen was reinforced, and she was counselled on the risks of pica and the importance of addressing underlying nutritional deficiencies.

DISCUSSION

Clay ingestion-induced hypokalemia is a rare but potentially serious cause of hypokalemic paralysis.[4,7,8] In this case, the patient's longstanding habit of clay ingestion, secondary to pica, exacerbated by severe iron deficiency anemia, led to electrolyte disturbances and subsequent muscle weakness.[9-11] Prompt recognition and appropriate management of both the electrolyte imbalance and the underlying iron deficiency anemia were crucial in achieving a favourable outcome. Clay causes increased intestinal excretion of potassium as the clay binds to potassium ions.[12]

Long term effect of Pica includes stress or anxiety, negative behaviour during childhood, nutritional deficiency, and mental health issues.[13]

CONCLUSION

This case emphasizes the importance of considering unusual etiologies, such as clay ingestion induced by pica, in the differential diagnosis of hypokalemic paralysis, particularly in elderly patients with concomitant nutritional deficiencies. Early recognition and intervention are essential for effective management and prevention of complications associated with this rare condition.

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