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Case Report

Severe and rapidly changing hypophosphatemia in cannabinoid hyperemesis syndrome: a case report

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Abstract

The increasing prevalence of cannabis worldwide requires awareness of a potential, less recognized, paradoxical entity, the cannabinoid hyperemesis syndrome (CHS). This includes cyclic episodes of nausea, vomiting, and compulsive hot water bathing for alleviation in individuals with chronic cannabis use. An 18-year-old male with daily and prolonged cannabis use has excessive nausea and vomiting, is diagnosed with CHS, and is further complicated by severe and rapidly fluctuating hypophosphatemia. He was successfully managed with intravenous (IV) antiemetic (metoclopramide) and IV normal saline in the emergency department. Hypophosphatemia was treated with IV phosphorous. Although hypophosphatemia in CHS is a rare encounter, the authors share their experience to promote broader recognition and insight into successful management.

Keywords: cannabinoid hyperemesis syndrome, vomiting, cannabis, marijuana, hypophosphatemia, intravenous phosphorous, case report

INTRODUCTION

Cannabis is the most widely used illicit substance in the United States and worldwide. The 2008 World Health Organization World Mental Health Surveys estimated the cumulative lifetime incidence of cannabis use to be 42.4% in the US population [1]. The medical community has well-articulated the positive effects of the antiemetic and appetite-stimulatory properties of cannabis. However, prolonged heavy use of the same has been deemed a potential, less well-recognized, paradoxical entity known as cannabinoid hyperemesis syndrome (CHS). First described in 2004 by Allen et al., CHS is characterized by paroxysmal and recurrent episodes of nausea, vomiting, colicky abdominal pain with long-term cannabis use, and compulsive hot-water bathing, which resolve upon cannabis abstinence [2, 3]. Symptomatic relief with hot water baths is a specific sign of this condition (91%–92.3% of cases) [2]. With the widespread use and/or abuse of marijuana and growing legalization, the authors aim to promote broader recognition and further understanding of CHS through their unique experience. The authors discuss the case of an 18-year-old male who had excessive nausea and vomiting after prolonged use of cannabis and had been diagnosed with cannabinoid hyperemesis syndrome, further complicated by severe and rapidly fluctuating hypophosphatemia. This derangement in phosphate level is a rare presentation in a user of cannabis with CHS.

CASE PRESENTATION

An 18-year-old male with a past medical history of asthma, bipolar disorder II, anxiety, Mallory-Weiss tear, and cannabinoid hyperemesis presented to the emergency department (ED) with complaints of nausea and vomiting for one day without fever. He complained of multiple episodes of intractable vomiting, acute in onset, non-bilious, non-blood-stained, and containing food particles. He had not been able to tolerate food by mouth and also complained of abdominal pain and two episodes of diarrhea. There is a positive history of occasional alcohol intake, and the patient has been smoking cannabis regularly for the past four years and denies the use of vaping or any unprescribed medications. He has had similar episodes for the past two years and has been treated accordingly, with periods of remission in between.

On examination, his vital signs were insignificant except for bradycardia. Systemic examinations were normal. Laboratory investigation was significant for low phosphate levels (<0.3 mg/dL) (normal range 2.5–4.5 mg/dL) and elevated lactic acid of 6.1 mmol/L. His ECG, serum urea and creatinine, and urinalysis remained normal. A 15 mmol Kphos dose of intravenous (IV) phosphorus was administered over four hours along with normal saline and IV antiemetic at the ED.

The patient was discharged after five hours at the ED, and his phosphorus level was 3.9 mg/dL at that time. Following

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seven hours of discharge, vomiting again started, and the patient returned to the ED. In the ED, vital signs were stable, but laboratory results were significant for low phosphate (0.6 mg/dL) and elevated lactic acid. Subsequently, intravenous phosphorous was replenished along with normal saline and an anti-emetic, which was followed by oral phosphate.

DISCUSSION

Cannabinoid hyperemesis syndrome is a paradoxical entity described in detail by a few reports [3]. The clinical diagnosis of CHS is often difficult due to its vague presentation as well as faltering, misinformation, and hesitancy with obtaining a history of cannabis intake from the patient. Diagnosis is of exclusion and is established in the background of prolonged cannabis intake with cyclical vomiting, vague abdominal pain, and resolution of these symptoms by hot water bath, in absence of organic etiology [3, 4]. Our patient had similar episodes for the past two years, which makes our diagnosis of CHS plausible. Cannabis is often used therapeutically in patients with nausea and vomiting induced by a variety of conditions, including chemotherapy-induced nausea and vomiting (CINV). This paradoxical presentation is thought to be due to chronic overstimulation of endocannabinoid receptors and the transient receptor potential vanilloid-1 (TRPV1) nociceptor system in the peripheral nervous system [5].

Hypophosphatemia occurring in CHS is a rare encounter described in a handful of reports [6–8]. The possible mechanisms of hypophosphatemia include enteric malabsorption or loss, excessive urinary excretion, or redistribution within the body (or transcellular shift) [6, 9]. It can be caused by similar mechanisms in respiratory alkalosis, excess catecholamine, and insulin secretion (re-feeding syndrome) [6], which were ruled out in our case.

Although scarce, the available literature demonstrated rapid self-correction of hypophosphatemia, usually occurring within a few hours [6, 7]. Our case is likely an exception. The rapid correction of phosphate within hours and spontaneity suggest that transcellular shifts and redistribution are the major culprits rather than total body depletion [10].

When the total body reserve for phosphorus is depleted, severe manifestations such as ataxia, depressed myocardial contractility, diaphragmatic weakness (phosphate levels < 2 mg/dL), and rhabdomyolysis (phosphate levels < 1 mg/dL) occur and are associated with poorer outcomes [11]. However, hypophosphatemia in CHS is asymptomatic; as in our case, the patient remained asymptomatic, which justifies a transcellular shift of phosphorus with a shift correction.

Our patient received intravenous antiemetic (metoclopramide) and IV normal saline in the ED.

Hypophosphatemia was tackled with IV phosphorous replenishment and serial measurements. Treatment of CHS includes supportive management for abdominal pain, fluid and electrolyte replacement, treatment of vomiting, and education for cannabis abstinence. Temporarily, hot-water baths have been effective in acute hyperemesis, which is often customary in patients experiencing CHS, while cannabis sobriety is essential to a permanent cure and preventing a recurrence [3, 12]. To optimize clinical outcomes, physicians should conduct urine toxicology screening for cannabinoids and serum electrolytes, particularly phosphate, at the initial point of care [8].

With the expanding legality of marijuana around the world, the authors expect a rise in the number of cases of CHS. Clinicians should therefore be aware of this fairly avertible, alarming affair among cannabis users and evaluate for concealed hypophosphatemia, which may be overlooked.

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CONFLICT OF INTEREST STATEMENT

The authors declare that they have no conflicts of interest.

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AUTHORS' CONTRIBUTIONS

Prakash Acharya contributed to the management of the patient, the study concept, the draft, and the review of the manuscript. Aakash Mishra and Sandip Kuikel drafted the original manuscript and revised it. Aman Mishra, Robin Rauniyar, Kunjan Khanal, Amit Sharma Nepal, and Sahil Thapaliya reviewed and revised the manuscript and provided valuable insights. All authors certify that they contributed sufficiently to the intellectual content. Each author has reviewed the final version of the manuscript and approved it for publication.

ETHICAL APPROVAL

The Institutional Review Board deemed the study exempt from review.

CONSENT

Written informed consent was obtained from the patient.

GUARANTOR

Dr. PA.

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