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The man with the mysterious hypokalemia

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Abstract

A 57 year-old male, with a past medical history significant for hypertension, presented to the emergency room complaining of generalized weakness. He had run out of his antihypertensive, amlodipine 5mg,. The patient was found to have severe hypokalemia and was admitted for further evaluation and management. Despite replacement with multiple runs of potassium, the patient's hypokalemia failed to improve. During his hospital stay, it became apparent that he was experiencing fever along with cyclical shaking spells that in conjunction with subsequent drops in potassium. Despite no past medical history of malaria, and no recent travel to endemic areas, a parasite smear was ordered given his presentation. The study was positive for Plasmodia falciparum. Subsequently, the patient was started on antimalarial drugs; consequently, his potassium levels stabilized and his symptoms resolved.

Keywords

malaria; P. falciparum; hypokalemia; family medicine; inpatient medicine; infectious disease

Introduction

Malaria is a parasitic infection spread by the bite of the female anopheles mosquito [6]. There are four parasitic species, namely, *Plasmodium vivax, Plasmodium falciparum, Plasmodium ovale* and *Plasmodium malariae* that are known to infect humans [6]. Other modes of transmission can be congenital or via blood products [7]. Generally, malaria initially presents with symptoms typical of a viral illness with cyclical fevers [6]. The typical presentation of malaria is common only 50-70% of the time [7]. Early diagnosis in patients with P. falciparum is important in order to avoid a host of complications associated with malarial illness. Malaria is known to cause electrolyte abnormalities, due to its disturbance of water balance [6]. Other complications resulting from malarial infection include: renal failure, intravascular hemolysis, metabolic acidosis, disseminated intravascular coagulation(DIC), hypoglycemia, hepatic failure. One of the gravest complications of malaria includes cerebral malaria; this is characterized by altered mentation and seizures [5]. People living in areas where malaria is endemic already possess partial immunity to malaria; they, however, are at risk for repeated infections [6]. Many people carry the disease but have no clinical manifestations [6].

Case Report

Day 1: A 57 year-old African-American male presented to the emergency department with a two day history of generalized weakness and dizziness. He attributed these symptoms to noncompliance

with his antihypertensive medication, Amlodipine, since he had run out. He denied chest pain, shortness of breath, blurry vision, headaches, cough, fever, nausea, vomiting, diarrhea, dysuria, sick contacts, similar symptoms, weight loss, or night sweats.

His past medical history consisted of hypertension diagnosed at the age of 14 years. His medications at home were: Amlodipine 5mg daily, and Aspirin 81 mg. The patient's family history was significant for hypertension in nine out of ten siblings. His last hospitalization was in 2014 for a hypertensive crisis. His travel history was significant for a recent trip to Canada one week ago. His last travel to Africa was three years ago.

While in the emergency room, his vital signs were as follows: Blood Pressure – 172mmHg/127mmHg, Heart Rate – 79bpm, Oxygen Saturation – 99% on Room Air, Respiratory Rate 18, Temperature – 97.6 F. Additionally, the patient had laboratory workup, which included: severe hypokalemia at 2.4mEq/L, hypomagnesemia at 1.6mEq/L, Blood urea nitrogen/Creatininewas 21/1.3 and Creatine Kinase (CK) 1277U/L. Hemoglobin/Hematocrit was 12.0g/dl/37.6%, Platelets 102.x 1000 microliter. His Chest X-ray was negative for any acute cardiopulmonary pathology. His EKG was significant for an incomplete right bundle branch block. An eight-panel urine drug screen was negative. Urinalysis showed 1-5 red blood cells.

While in the emergency department, the patient received 60 mEq IV Potassium Chloride, 20meq oral potassium chloride, 2 grams magnesium Sulfate intravenously, normal saline bolus, as well as amlodipine 5 mg.

After the potassium was replaced, his Basic Metabolic Panel(BMP)was repeated two hours later, showing some improvement in his magnesium levels as well as creatine kinase (CK) levels. His magnesium had improved to 2.1mEq/Land the CK level trended down to 787U/L from 1277U/L.However, the potassium level remained low at 2.5mEq/L, despite replacement mentioned above. Given his hypokalemia and uncontrolled blood pressure, the patient was admitted to the hospital for further evaluation and management.

Day 2: The following morning, the patient's blood pressure was 147mmHg/92mmHgand his potassium had normalized to 3.8mEq/L. However, his magnesium had decreased to 1.6mEq/L, despite adequate replacement the night prior. His CK was 627U/L.

Later that afternoon, the patient had sudden onset of uncontrollable shivering, lasting approximately 10 minutes, associated with considerable weakness. Of note, his vital signs were stable. His blood pressure was – 128 mmHg /76 mmHg, Heart Rate – 84 bpm Oxygen Saturation – 99% on Room Air, Temperature – 96.8 F. Repeat Basic Metabolic Panel and ECG were ordered. BMP was significant for hypokalemia at 2.7mEq/L. His EKG findings were significant for Sinus tachycardia, slight up sloping and ST segment depression. Cardiology was consulted for abnormal EKG; the patient underwent a pharmacologic stress test, which was within normal limits.

At that time, the amlodipine 5mg was changed to lisinopril 20 oral daily. At this time he had received a total of 90 mEq potassium chloride intravenously and 80 mEq potassium chloride orally over the past 36 hours. Nephrology had been consulted for severe hypokalemia. Renal ultrasound was ordered as well; findings were significant for medical renal disease as well as a complex upper pole right

renal cyst; radiologist recommended an MRI study for further characterizing the lesion. The MRI study was significant for a Bosniak 2F cyst.

Diagnosis/Day 3: By the third day of admission, the patient had received aggressive potassium replacement: On day 1 of hospitalization, he Received 70meq of potassium chloride. The second day, he received 80meq, and on the third day, he received 50meq. The patient also had another episode of chills and shaking and this time was febrile.

Upon further questioning of the patient, he admitted to experiencing these shivering spells at home a couple of times, slightly over a week prior to presentation at the hospital. These episodes lasted 5-10 minutes; he did not think to mention them at the time. He never had malaria in the past, no history of sickle cell trait, and did not recall any bug bites or mosquito bites. The first sign of his shivering spells were two days after he returned from his four-day trip to Toronto, Canada.

Given the patient's African origins and history of travel to Africa a few years back, a parasite blood smear was ordered. It came back as positivefor Plasmodium Falciparum. The percentage of infected cells was 0.37 %.CBC that morning also showed Hemoglobin/Hematocrit 11.0g/dl/33.9%, platelets of 79x 1000 microliter. Infectious disease was consulted and per their recommendations, the patient was started on IV doxycycline and Quinine, Dextrose 5% with Normal Saline and KCL. His clinical status began to improve.

At time of discharge, his percentage of infected cells was less than 0.07 %. Platelet count was 198 x 1000 microliter and potassium had stabilized to 3.7mEq/L. He was discharged with the instructions to follow up with his primary care provider.

Discussion

According to the CDC, the annual incidence of malaria in the United States is 1500 cases; in Canada, the annual incidence of malaria is 500 cases [1,2]. Malaria is rare in the United States and Canada; however, it is an important differential to consider in patients with electrolyte disturbances that do not resolve after replacement. The most common electrolyte disturbance in malarial patients are as follows: hypoglycemia, hyponatremia, and hypokalemia. [3,4]. Patients with origin or travel history to regions where malaria is endemic, namely, West Africa, South Asia and the Caribbean, should raise suspicion for this parasitic infection.

P. falciparum, has an incubation period from anywhere from 7 to 30 days; most commonly, the incubation period is between 12-14 days [7]. It is believed that the patient contracted the disease while visiting friends in Canada, despite the lack of bug bites and witnessed mosquitos. He started to experience symptoms approximately six days after he arrived to Canada, (or two days after he returned home).

Electrolyte disturbances in malaria have been well documented and can sometimes vary in presentation. Sodium is the extracellular cation, whereas potassium is the intracellular cation; both sodium and potassium play a significant role in the maintenance of pH, cardiac function as well as other biochemical reactions [6]. A possible mechanism of hypokalemia in the case of severe malaria, relates to the red blood cells (RBC) being infected with the malaria parasite. This results in increased concentration of sodium with decreased concentration of potassium in the cytosol [6]. Although malaria can cause

hyperkalemia as well, hypokalemia is quite common [7]. A study by Thanachartwet demonstrated that 4.4% of patients with malaria had severe hypokalemia, while 0.6% had hyponatremia. A study by Rathod demonstrated that hyponatremia and hypokalemia were common in both P. Falciparum and P. Vivax malaria, but the severity of both electrolyte disturbances was much greater in P. falciparum than in P. vivax [4].

Hypokalemia is quite dangerous in that it can lead to arrhythmias, hypotonia, and even death [7]. Hyponatremia is also a common complication of severe malaria; several mechanisms have been proposed for this phenomenon, namely, administration of hypotonic fluid, SIADH, salt wasting, among others [7]. It is extremely important to appropriately manage the electrolytes in malaria patients, as biochemical processes are dependent on them [7]. In the case of this patient, he experienced severe hypokalemia that improved once he was treated appropriately for his malaria.

The treatment of malaria involves antimalarial medications, close monitoring of blood glucose to prevent hypoglycemia, daily parasite blood smear monitoring, as well as monitoring and replacement of electrolytes. It is prudent to maintain a high index of suspicion of malaria in patients who present with a history of travelcyclic shaking spells, along with electrolyte abnormalities that fail to correct despite aggressive replacement. Early diagnosis leads to timely treatment and avoidance of the complications associated with malaria.

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