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RESEARCH ARTICLE

FEVER WITH QUADRIPARESIS: UNRAVELLING THE MYSTERY

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ABSTRACT

Here, we describe the case of a young male who presented with intertwining high-grade fever and acute-onset quadriparesis. Temporally, the weakness manifested after a seemingly innocuous treatment with intravenous 5% dextrose. A diligent fever work-up uncovered an infection with Plasmodium vivax, and biochemical analysis revealed low serum potassium. Yet, this revelation added to the riddles that entangled his diagnosis. Was this mysterious hypokalemic periodic paralysis a consequence of the innocent intravenous glucose, a silent accomplice in his plight? Or could it be an elusive manifestation of malaria, a rare expression of the disease that ensnared him?

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INTRODUCTION

Hypokalemia is one of the most common electrolyte disturbances observed in clinical practise.(1) The severity of hypokalemia is classified as mild when serum potassium levels are between 3 and 3.4 mmol/L, as moderate when serum potassium levels are between 2.5 and 3 mmol/L, and as severe when serum potassium levels are less than 2.5 mmol.(1)Significant muscle weakness occurs at serum potassium levels below2.5 mmol/L but can occur at higher levels if the onset is acute. The pattern of weakness is ascending in nature affecting the lower extremities, progressing to involve the trunk and upper extremities and potentially advancing to paralysis.(1)Periodic paralysis is a rare neuromuscular disorder. Musgrave first described hypokalemic periodic paralysis (HypoKPP) in 1727.(2) Most cases are hereditary or familial. The familial form is a rare channelopathy caused by a calcium or sodium ion channel mutation, primarily affecting the skeletal muscle cells.(3) A mutation in the CACNA1S gene, which codes for a dihydropyridine-sensitive calcium channel in skeletal muscle, is responsible for the most frequent familial form of HypoKPP, type 1. The other type of familial HypoKPP, known as type 2 HypoKPP, is caused by mutations in a gene called SCN4A that encodes a voltage-sensitive sodium channel in skeletal muscle. There have also been discoveries of disease-causing mutations in the genes KCNJ2 and KCNJ18, which code for the inward rectifier potassium (Kir) channel.(3)Acquired cases of HypoKPP are associated with hyperthyroidism, renal tubular acidosis, gastroenteritis, or endocrine causes.(3) The name periodic can be deceiving, as attacks do not occur at regular intervals but are episodic and come suddenly.(3) Rest following heavy activity and consumption of high-carbohydrate meals are the most persistent precipitating variables.(4,5) Excitement, tension, fear, cold, salt intake, glucocorticoid use, alcohol use, and anaesthetic procedures are also triggers.(4,6)It is hypothesised that these precipitating events increase plasma epinephrine or insulin levels, resulting in an intracellular shift of potassium and a decrease in serum potassium levels, precipitating an episode of weakness.(7) A young male with high-grade fever and acute-onset quadriparesis is described here. Intravenous administration of 5% dextrose precipitated acute paralysis. During a comprehensive evaluation of a fever, a Plasmodium vivax infection and low serum potassium levels were detected.

CASE LETTER

In a rather perplexing case, a young male was brought to the hospital in an ambulance with complaint of high-grade intermittent fever with chills that plagued him for seven days.

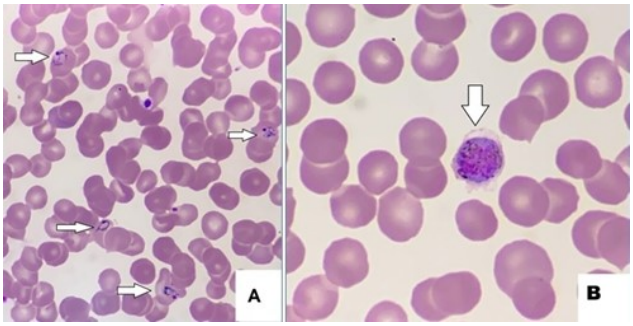

Figure 1. The blood film reveals various forms of Plasmodium vivax malaria. A. Multiple ring forms (right-pointing arrows) in the RBC have a thick ring with a large chromatin dot. B. The gametocyte seen (downward- pointing arrow) has a spherical and compact structure that almost fills the RBC (Leishman and Giemsa stain, x1000)

The fever was punctuated by a solitary bout of vomiting that was non-bilious and contained ingested food material. Previously after a few days of the fever, he sought medical assistance from a local doctor, who administered two bottles of intravenous 5% dextrose. Little did he know that his seemingly benign intervention would usher in a new chapter of enigma and perplexity? He then developed sudden onset of progressive weakness in all four limbs that evolved over 48 hours to a stage where he could barely move. There was no history of diarrhoea or any other systemic complaints. The bladder or bowel remained unaffected. There was no history of difficulty in respiration, blurring of vision, double vision, facial asymmetry, nasal regurgitation or difficulty swallowing. As we delve deeper into his medical history we found no history of recent vaccinations, the use of diuretics, or any trauma or seizures. There was no history of a similar weakness in his past or significant family history. The patient denied any history of alcohol abuse or recreational drug use. He was febrile with a temperature of 102.5° F, pulse rate of 92/min and blood pressure of 109/65 mm Hg. There was no clubbing, icterus, cyanosis or any palpable lymph node. Per abdominal examination revealed, mild hepatosplenomegaly. His cardiac and respiratory system examination was within normal limits. On neurological examination, the patient had a bilaterally symmetrical pure motor quadriparesis of MRC Grade 2-3/5, with more proximal weakness than distal. No cranial nerve involvement or cerebellar signs were present.

Investigations and Treatment: A complete blood count provided us with valuable insights, showcasing a haemoglobin of 13.2g/dl, a WBC of 4.71 x10^9/L and a platelet count of 30 x10^9/L. The blood film unveiled the presence of Plasmodium vivax malaria, with its characteristic ring forms and gametocytes(figure 1). Delving further into the realm of biochemical results, which were as follows; AST 41 (23-50) U/L, ALT 43 (22-67) U/L, total bilirubin 1.8 (0.1 to 1.2) mg/dL, serum urea 35 (29-60) mg/dL, serum creatinine 0.9 (0.4-1.2) mg/dL. Serum magnesium, folate, vitamin B12, calcium and blood glucose were within limits of normality. His urine test results were also within the normal range. The patient tested negative for dengue, HBsAg, Anti HCV and HIV. TSH, Total T4 and Total T3 were normal. Among the electrolytes, the sodium stood at 136mmol/L (135–145 mmol/L). However, it was serum potassium that provided a vital clue with a value of 2.7mmol/L (3.5–5.0 mmol/L). The ECG was normal. Guided by this revelation, a therapeutic intervention unfolded. Intravenous potassium chloride 40 mEq was given in ringer lactate solution @100ml/hr.

After 80 mEq of intravenous potassium, serum potassium rose to 4.9 mEq/L. Potassium supplementation was then stopped. Within a mere 24 hours of presenting to us, a remarkable transformation occurred. The patient's motor function recovered completely. In view of the multidimensional nature of our patient's condition, he also received intravenous artesunate 120mg, 12 hourly for five days.

Outcome and follow-up: The patient became afebrile and was discharged on day five on oral artemether and lumefantrine twice daily for three days with a serum potassium level of 4.9 mEq/L.

DISCUSSION

Malaria is caused by protozoan parasites in the genus Plasmodium.[8]Humans are infected with one (or more) of the following species of Plasmodium: *falciparum*, *vivax*, *ovale*, *malariae* and *knowlesi*.[8] The classical presentation is seen in 50%–70% of the cases, with the rest having atypical manifestations involving the nervous system, musculoskeletal system, haematological system, renal system, respiratory system, cardiac system, gastrointestinal system and metabolic derangements. Musculoskeletal manifestations reported with malaria include rhabdomyolysis, myositis, and periodic paralysis.[8]In a study by Thanachartwet *et al*., 81% of patients with uncomplicated malaria had disturbances in serum sodium and potassium.[9]They found that hypokalaemia was the most common disturbance in 43% of patients with Plasmodium vivax infection.[9]In their study, volume depletion was the predominant risk factor for hypokalaemia. Hypokalaemia was due to intracellular translocation of potassium from the extracellular fluid and urinary potassium loss.[9]

In contrast, hyponatremia occurred in 37% of the patients infected with Plasmodium falciparum.[9]Maitland *et al*. observed hypokalaemia in thirty-eight children with falciparum malaria, where the underlying reason was the correction of acidosis.[10]Within 12 hours of the parasite's entrance into the host, Dworak *et al*. reported a gradual reduction in sodium and potassium levels.[10]Motor weakness secondary to hypokalaemia has been mentioned in infectious diseases in adults, namely chikungunya, dengue fever, and leptospirosis.[10]However, motor weakness due to malaria has been rarely reported, like in a 26-year-old male with Plasmodium vivax who developed hypokalemic paraparesis.[10]His serum potassium at admission was 1.47 mEq/L, and treatment involved correcting the hypokalemia and treating malaria. The pathophysiology behind developing hypokalemic paralysis in various infections is not well understood. Hypokalemia is thought to be caused by a pathophysiological process involving potassium redistribution as a stress reaction caused by the production of catecholamines, insulin, and renin or increased potassium loss from the body.[10]

Our patient was a young male who presented with relentless high-grade fever and an abrupt and bewildering quadriparesis. Laboratory work-up brought forth a revelation-an insidious infection with Plasmodium vivax, and a deficiency in serum potassium levels. This was the first time that the patient had shown symptoms of quadriparesis leaving us yearning for answers to the mysteries that entwined his ailment. In this intricate dance between malaria and quadriparesis, the delicate balance of causality remains elusive.

While it is challenging to attribute causality to malaria infection in our patient who presented with HypoKPP, dismissing malaria as an innocent bystander with no etiopathological contribution in the face of such complexity would be naïve. In conclusion, all cases of HypoKPP must be evaluated thoroughly to exclude secondary causes, and motor weakness secondary to complicated infectious diseases must also be kept in mind, especially by physicians in endemic areas, for an early diagnosis and timely intervention and treatment.

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None to declare

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