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Case Report

Malaria and channelopathy- recurrent hypokalaemia - A case report

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ABSTRACT

Malaria is associated with various forms of neurological complications. Weakness like paraparesis and quadriparesis is being described in malaria, the pathologies being polyneuropathy, hypokalaemia, hyperkalaemia, myelitis etc. Recurrent hypokalaemic paralysis has not been described to the best of my knowledge in malaria. I hereby describe a young patient with recurrent episodes of weakness due to hypokalaemia who on investigation was found to be because of malaria. The case report is being described because of it is being novel or not described earlier.

1. Introduction

Various electrolyte abnormalities are being described in malaria.[1] Like many other neurological complications, hypokalaemia is found more often in Plasmodium falciparum than Plasmodium vivax.[2] The hypokalaemia or hyperkalaemia is specially being described from Indian subcontinent and around like Shri Lanka.[1–5] We encountered such a patient with recurrent episodes of hypokalaemic paralysis, which is being described probably first time to the best of our knowledge.

2. Case Report

A 20 years male patient presented with history of intermittent (alternate day) fever with chills and rigors, occasional vomiting, no loose motions and myalgia of four days duration. There was no history of alcohol use or abuse, diabetes mellitus, renal failure. On 5th day/the day of admission the patient suddenly developed weakness of all four limbs, without bowel, bladder and sensory involvement. There was no history of such illness or weakness in the family.

On examination, the patient was well nourished and well built, blood pressure was 122/76mm of Hg in right arm in sitting position, with no significant postural fall, pulse rate of 96 per minute, regular; respiration was also normal with respiratory rate of 20/minute, abdomino-thoracic. On neurological examination higher mental functions were normal and so was the speech and cranial nerves examination. In motor system examination the muscle bulk was normal in all four limbs and tone was diminished, power was 4+/5(Medical Research Council) in upper limbs proximally and 4/5 distally. Power was 4/5 proximally in lower limbs and 4-/5 distally. The deep tendon reflexes were absent in all four limbs. Sensory and cerebellar system examination was unremarkable.

Blood investigations showed hemoglobin 10.4gm%, total leucocyte counts 3690/cubic mm, serum calcium 8.3mg/dl, sodium-139meq/l, and potassium-2.0meq/l, fasting blood sugar was 62mg/dl, blood urea-29mg/dl, serum creatinine-0.99mg/dl, serum bilirubin1.20mg/dl, S.G.O.T.-20 IU/L and S.G.P.T.29 IU/l. Thyroid profile was absolutely normal. The

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thick peripheral blood film was positive for P. vivax malaria. Other causes of hypokalaemia were ruled out by relevant history, examination and investigations. Nerve conduction study performed on 7th day of weakness was normal in all four limbs. The cerebrospinal fluid examination showed normal cell counts and cell type along with normal protein, sugar and matching blood sugar levels.

The patient was given intravenous artisunate, intravenous potassium chloride and oral calcium supplementation. The patient showed dramatic improvement and resumed his toilet duties as usual on very same day. The patient was discharged after adequate course of antimalarials and supportive treatment in fit and fine condition.

The patient again came after approximately four months with history of fever with chills and rigor, with treatment from general practitioner in form of antibiotics and symptomatic treatment with no improvement and on examination had almost similar clinical findings as it was during first admission. On investigation it was found to have low potassium (3.2 meqL), sodium 140meq/l, calcium 9.1mg%, thrombocytopenia. Serum thyroid profile was normal. Peripheral blood film showed moderate parasitemia for Plasmodium vivax with schizonts and rings of it. The other causes of hypokalaemia were again ruled out by relevant history, examination and investigations. Again patient improved dramatically with antimalarial, potassium supplementation and supportive treatment.

3. Discussion

Various neurological complications are being described in malaria which include whole neuraxis.[6] Electrolyte abnormalities causing hypokalaemia or hyperkalaemia with weakness is far less described or reported. Most of such case reports are from Indian subcontinent and around like Sri Lanka. Recurrent episodes of malaria presenting as hypokalaemia is not yet described or reported till to the best of my knowledge. All 3 patients of Senanayke and Wimalwasana[4] had no history of periodic paralysis and the weakness precipitated during episodes of rigors and the patients started improving within 4-10 hours of starting antimalarial treatment. The authors didn't get done serum potassium levels during whole of the course of illness and concluded that the rarity of the occurrence may suggest the possibility of some genetic predisposition in the diseased patients. The authors further postulated that "the combination of transient hyperkalaemia and rigors occurring during febrile episodes of malaria is suggested as an underlying cause which precipitated the paralysis". Transient rise of plasma potassium during febrile episode of malaria is well known and is ascribed to lysis of red blood cells and intense muscular contractions during rigors.

Periodic paralysis due to hyperkalaemia or hypokalaemia in such setting is to be differentiated from, polyneuropathy of Landrey Guillain Barre (LGB) type by relevant history and appropriate investigations, and be treated accordingly. There is also necessity to require utmost care for precipitating factors like hemolysis, renal failure, acidosis, hypo glycemia, rhabdomyolysis etc. This entity may also be under recognized and may be confused with neuropathy or unrecognized in deep coma. Further such patients with hyperkalaemic or hypokalaeimic periodic paralysis require the monitoring of serum potassium levels, degree of parasitaemia responsible for hemolysis and hyperkalaemia.

Hyperkalaemic periodic paralysis, L.G.B. like polyneuropathy and delayed cerebellar ataxia are particularly reported from India and Sri Lanka, which may reflect the prevalence of particular strain of plasmodium in this subcontinent or genetic predisposition of the residing population i.e. ion channel mutation, as pointed out earlier by Senanayake & Wimalavasna.[4] Cause of hypokalaemic paralysis as in our patient again requires further observation in forthcoming cases and a larger case series or randomized trial including genetic studies.

Our patient is different from previous reports that in one of earlier case report there was hyperkalaemia[4] or hypokalaemia[1–3] and in the others from Sri Lanka potassium levels were not done.[5] This is probably to the best of our knowledge is first documentation of such a rare entity in a common parasitic infection. The awareness about this aspect will be of immense importance in malaria endemic countries and also in those with history of travelling to a malaria endemic region or country, as it is a common differential of LGB, and malaria is quite endemic in Indian subcontinent with time to time upsurge in incidence specially in post rainy season.

The drawback of our case report is that we could not evaluate the patient for ion channel disorder genetic study because of non-availability of testing at our centre and patient being poor could not afford to get it done from elsewhere.

4. Conflict of Interest

The authors declare that there is no conflict of interest.

5. Source of Funding

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