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Case Report

Hypokalemic Quadriparesis in a case of *Plasmodium vivax* and Scrub Typhus Co-Infection - A Case Report From Eastern India

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Abstract

Both *Plasmodium vivax* malaria and Scrub Typhus has numerous reported neurological complications. In our case a middle-aged man presented with quadriparesis with history of high-grade fever with chills and rigor. On investigation, serum potassium found to be low and *Plasmodium vivax* ring form was found in blood smear. Patient started intravenous Potassium supplementation along with Anti-malarial therapy. Weakness improved within 48 hours of potassium supplementation, but fever persists after three days of antimalarials. On through examination we found an eschar in the back region and subsequently Scrub Typhus serology (IgM) came Positive, fever subsided after starting Doxycycline. Hypokalemia often seen in children with *Plasmodium falciparum* malaria infection. But co-infection of Plasmodium *vivax* malaria with Scrub typhus can also cause severe form of hypokalemia which may lead to even hypokalemic quadriparesis. This is very much atypical presentation in a rickettsia and parasitic co-infection which has not been previously reported.

Keywords: *Plasmodium vivax*; Scrub Typhus; Co-Infection; Hypokalemic Quadriparesis

Introduction

Malaria is a vector borne disease caused by mainly 5 species of *Plasmodium*, they are *P. vivax*, *P. falciparum*, *P. ovale*, *P. malariae*, *P. knowlesi*. Globally there were 229 million reported cases of malaria with 4 million deaths in the year 2019. Malaria is endemic in India; more than 1.5 million cases are reported in year 2020. On the other hand, Scrub Typhus also known as Tsutsugamushi Disease, caused by Orienta tsutsugamushi, a zoonosis caused by bite of infected Leptotombidium mite larvae(chiggers). About 1 million cases are reported per year worldwide. The disease is wide spread in rural South and Suth-Eastern Asia and Weastern pacific as well as in India & Pakistan. Scrub typhus has increasingly been reported from various regions of India especially the hilly regions of the Himalayas, Assam, West Bengal and Tamil Nadu [1]. Malaria and Scrub Typhus co-infection has been reported in many case studies [2-4]. Both Vivax malaria and Scrub has many devastating complications, so co-infection may make it fatal.

Case

A 35 years old, non-diabetic, non-hypertensive patient presented to us with history sudden onset weakness of bilateral lower limbs followed by involvement of bilateral upper limb within 6 hours periods and also associated with cramping pains in bilateral lower limbs. There is no history of any loss of consciousness, sei-

zure, trauma. No history was elicited that suggested any definite sensory loss or bowel and bladder involvement. Neither there was any history suggestive of any cranial nerve involvement. However, patient was suffering from history of high-grade intermittent fever with chills and rigor for last 3 days before admission. No history of similar type of illness in past, or in first degree relatives. On general survey, BP-118/80 mmHg, pulse 106/min high volume bounding pulse, Spo2 99% in room air, Respiratory rate - 24/min with normal abdominothoracic breathing pattern, temperature 101°F, patient was lying helplessly on bed. On neurological examination, patient was conscious, alert, co-operative and oriented to time, place & person, GCS 15/15. Higher mental function was within normal limit, no neck rigidity, cranial nerves examination within normal limit. But patient was bed bound. Motor system examination revealed hypotonia, power bilateral upper & lower limb 2/5 i.e., flaccid paralysis with loss of all deep tendon reflexes with bilateral non responsive plantar. Sensory and autonomic functions were within normal limits. Urgent blood for Electrolytes was sent, which revealed serum sodium - 131 meq/l, serum potassium - 1.09 meq/l, ABG shows respiratory alkalosis with hypokalemia, ECG shows ST depression in V1 - V5 with presence of U wave, intravenous and oral potassium correction was initiated. Routine blood revealed Hemoglobin- 12.5 gm/dl, Total leucocyte count - 2990/cmm , Neutrophil- 52%, Lymphocyte- 41%, Monocyte- 4%, Eosinophil- 3%,Basophil - 0%, platelets - 1.24 lakhs/cmm, urea- 29 mg/dl, Creatinine- 1.0 mg/dl, bilirubin- 1.0 mg/dl (direct- 0.5 mg/dl, Indirect- 0.5 mg/dl), SGPT- 39 U/L, SGOT-45 U/L, Alkaline phosphatase- 77 IU/L, serum Magnesium 1.9 mg/dl, TSH -1.907 mIU/l, peripheral blood smear shows ring form of *Plasmodium vivax* (Picture 1), Malaria parasite dual antigen test- positive for *Plasmodium vivax,* serology for Typhoid fever (S. *typhi* IgM) was negative, Dengue NS1 Ag - Negative. Immediately after getting malaria parasite in blood, we started oral Artemether (80 mg) & Lumefantrine (480 mg) based regimen started considering this atypical serious presentation of malaria from a malaria endemic zone. Chest X-Ray was within normal limits. Cerebrospinal fluid was planned to rule out Guillain-Barré Syndrome which showed cell count- 2/cmm, all lymphocyte, glucose 70 mg/dl and protein 28 mg/dl. Serology for hepatitis B virus, Hepatitis C virus and HIV I and II were non-reactive. Urine analysis no proteinuria, 00-02 pus cells per HPF, 5-6 epithelial cells per HPF. In the background of severe hypokalemia, we investigated also urine osmolality- 330 mOsm/kg and urinary potassium- 8.02 mmol/lit. After 3 days starting antimalarials patient was still febrile and repeat peripheral blood smear revealed no malaria parasites. On through general examination we found an eschar in the back region (Picture 2) and subsequently Scrub Typhus serology (IgM) came Positive (O.D-0.5118, C.O.V- 0.500). Immediately He was started on doxycycline. Weakness started improving with Potassium supplementation and power completely recovery within 48 hours with normalization of neurological findings, ECG changes were also simultaneously normalized (Table 1). Alongside oral Doxycycline 100 mg twice a day continued for 7 days. Fever subsided on day 3 of starting doxycycline and patient discharged in a hemodynamically stable and afebrile condition.

Picture 1: Malaria parasite in peripheral Blood.

Picture 2: Eschar on the back.

Time	0 hours	6 hours	24 hours	48 hours
Potassium level	1.09 mEq/l	2.69 mEq/l	3.8 mEq/l	4.7 mEq/l
Potassium Supplementation	I.V. 20 mEq/hr for 6 hours + oral supplementation	I.V. 10 mEqs/hr for 6 hours - oral supplementation	Oral supplementation	Stopped
ECG	ST depression in V1-V5, prominent U waves	-	ST flattened, no U wave	Normal
Upper limb Tone	Hypotonia	Normal	Normal	Normal
Upper limb power	2/5	4/5	5/5	5/5
Lower limb Tone	Hypotonia	Hypotonia	Normal	Normal
Lower limb power	2/5	3/5	4/5	5/5
Deep tendon reflexes	Absent (0)	1+	1+	2+
Plantar	Non-responsive	Non-responsive	Flexor	Flexor

Table 1: Potassium Timeline.

Discussion and Conclusion

Electrolyte imbalance is very common in Malaria. Ashima rani et al reported that serum levels of sodium, potassium, calcium can be altered in *plasmodium* sp. [5]. Hypokalemia in children can be in cases of Falciparum malaria and the proposed cause is Metabolic Acidosis [6]. An Indian study shows that hyponatremia and hypokalemia are more common in falciparum malaria [7]. Causes of hypokalemia in vivax malaria are multifactorial including transcellular shift and increase potassium loss via urine [8]. Scrub typhus can also lead to Electrolyte imbalance by causing AKI having various dreaded outcome [9,10]. Malaria can be associated with different neurological complications [11], hypokalemic paraplegia in Vivax malaria is also reported [12]. Case report of GB Syndrome in malaria is also present. But this patient responded well and fast after intravenous potassium therapy so Nerve Conduction Study (NCS) was not needed. There was also no obvious fluid loss by means of diarrhea or vomiting. The urinary potassium was normal, so there is no increased potassium loss via urine. There was no history of similar type episode previously, no history of any strenuous exercise preceding the event which rules out Glycogen Storage Disorders. However, patient remained febrile even after weakness recovered (after potassium correction) and full course (3 days) of oral ACT regimen. After meticulous examination we found an Eschar and ordered for Scrub Typhus IgM was found to be positive and fever subsided within 3 days of initiation of Doxycycline. The authors believe that in this case Hypokalemia developed due to intracellular distributional shift of potassium which was potentiated by both Malaria and Scrub Typhus Co-infection. This severe form of hypokalemia precipitated quadriparesis which shapely improved on potassium supplementation. So, Hypokalemia can be a serious and potentially easily correctable cause of quadriparesis in a rickettsia and parasitic co-infection.

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