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Case Report

Miller Fisher syndrome/acute motor axonal neuronopathy overlap an atypical manifestation of malaria: a case report

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ABSTRACT

Various types of neurological manifestations are described in P. falciparum/vivax malaria of which Guillian Barre syndrome and its variant like Miller Fisher Syndrome (MFS) and Acute Motor Axonal Neuronopathy (AMAN). We are reporting such an unusual case who presented with five days history of fever and weakness of three days duration. On investigations it turned out to be acute MFS/AMAN overlap with peripheral blood showing mixed infection having heavy parasitaemia of P. falciparum and P. vivax combine. All other causes of acute polyneuropathy were ruled out by history and relevant examination. Patient improved with Artemisinin based Combination Therapy (ACT) and other supportive measures.

Keywords: Malaria, Atypical manifestations, Miller Fisher syndrome, Acute motor axonal neuronopathy

INTRODUCTION

P. falciparum is causal for the most of severe and complicated malaria[1] whereas vivax malaria usually causes an uncomplicated disease, which is benign and is rarely fatal.[2] There is a paradigm shift recently where severe and fatal disease due to P. vivax infections is reported in literature.[2-8] Classical presentation is seen in only 50%-70% patients. In countries where malaria is endemic, due to development of immunity, malaria can present with unusual features. There is increasing resistance to antimalarial drugs, even the newer ones due to the indiscriminate use of antimalarial drugs[9] in such places. Many neurological manifestations are described in falciparum malaria like hemiplegia, cranial nerve palsies, myelitis-like syndrome, cerebellar dysfunction and psychosis, cerebellar ataxia, convulsions extrapyramidal disorders, etc. without unconsciousness as a presenting feature, as Post-Malaria Neurological Syndrome (PMNS) like tremors.[10] Only few case reports of peripheral neuropathy of Landry Guillian Barre type have been reported mainly from India.[11-14]

CASE REPORT

A 30 years villager was admitted with history of high grade fever of five days duration associated with diplopia, dizziness and weakness of both lower limbs with inability to walk for two days. On examination at the time of admission patient was conscious, afebrile, pulse rate was 92/min regular, BP was116/74 mmHg in supine position with a postural fall of 22 mmHg in sitting from supine position, respiratory rate 16/min, regular without any respiratory distress. On neurological examination higher mental functions and were normal. Cranial nerves palsy of 3rd, 6th and 7th was present bilaterally. Power in upper limbs was 2/5 and in lower limbs 3/5 proximally and 4/5 distally. Deep tendon reflexes were absent in upper and lower limbs with bilateral flexor plantar response. Sensory and cerebellar examination was normal. There were no signs of meningeal irritation and

no abnormal movement. The other systems were completely normal.

On investigations, Hb was 7.4 gm%, TLC 6300/cumm with neutrophils 46%, lymphocytes 52% and eosinophills 2%, ESR 13 mm in 1st hour. Peripheral blood film showed heavy parasitaemia with trophozoites of P. falciparum and Pvivax both. Quantitative buffy coat test for malaria was also positive for falciparum and vivax species. Hepatic and renal function tests were normal. Fasting blood sugar was 88 mg% with post prandial blood sugar (2 hour) of 142 mg% and urine examination was normal including urine for porphobilinogen. Nerve conduction studies performed were suggestive of Acute Motor Axonal Neuronopathy (AMAN variant of GB syndrome) and CSF cell counts were normal and protein was 82 mg%. Other causes of polyneuropathy like porphyria, organophosphorus poisoning, exposure to heavy metals were ruled out by history and relevant investigations. Patient was put on IV artesunate therapy 2.4 mg/kg stat dose, followed by 1.2 mg/kg after 12 hours thereafter repeated the later dose once daily for 5 days. He was given tab. primaquine 15 mg/kg for 14 days. Patient showed improvement on 4th day of starting specific treatment and was able to walk on tenth day with support and without support on 12th day. There was subsidence of diplopia on 13th day.

Patient was discharged on 14th day with oral haematinics. On follow up examination after six weeks patient had no neurological deficit.

DISCUSSION

Our case report represents the atypical manifestations of P. falciparum and vivax malaria in which MFS/AMAN variant of Guillain-Barre Syndrome (GBS) was present. GBS in malarial infection has been reported,[15,16] however the exact pathogenesis of Guillain-Barre syndrome in falciparum malaria is unknown, plausible explanation may be attributed to immune mediated capillary damage, toxic oxygen radicals, tumour necrosis factor, parasitic emboli obstructing the vasa nervorum resulting in anoxemic stagnation, causing transient demyelination, neurotoxin release, nutritional and metabolic disturbances. Complete recovery has been reported after clearance of parasitemia and maintenance of normal blood flow in vasa nervosum. Release of chemo/neurotoxins, metabolic and nutritional disturbance, immune-mediated capillary damage, and release of free radicals and TNF may also be causal for the pathogenesis of GBS after P. falciparum infection.[15,16,18,19]

The aim of reporting this case is to make awareness about this uncommon and atypical but treatable disease if specific treatment is initiated early and because of paucity of such cases in literature.

CONCLUSION

Malaria involves most systems and its manifestations are varied. Physicians, in tropical/endemic areas should have a high degree of suspicion for malaria so that the diagnosis and treatment are timely and morbidity and mortality reduced.

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