Acute Inflammatory Demyelinating Polyneuropathy with *P. falciparum* Malaria

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**Abstract**

Various types of neuropsychiatric manifestations are described in *P. falciparum* malaria of which peripheral neuropathy has been described mainly from India. We are reporting such a case who presented with seven days history of fever and weakness of two days duration. On investigations it turned out to be acute inflammatory demyelinating polyneuropathy (AIDP) with peripheral blood showing heavy parasitaemia of *P. falciparum*. All other causes of acute polyneuropathy were ruled out by history and relevant examination. Patient improved with quinine and other supportive therapy.

**INTRODUCTION**

Various forms of neuropsychiatric manifestations are described in *falciparum* malaria which are classified mainly into three groups1 : 1) Sequelae of cerebral malaria e.g. hemiplegia, cranial nerve palsies, myelitis-like syndrome, cerebellar dysfunction and psychosis, 2) Predominant neuropsychiatric signs and symptoms like psychosis, cerebellar ataxia, convulsions, extrapyramidal disorders, etc. without loss of consciousness during acute stage as a presenting illness, 3) As post-malaria neurological syndrome (PMNS), like cerebellar ataxia, psychosis and tremors. Of all these complications very few case reports of peripheral neuropathy of *Landry Guillian Barre* type have been reported mainly from India.2-5

**CASE REPORT**

A 60 years farmer was admitted with history of low grade fever of seven days duration followed by 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 86/min regular, BP 110/70 mm Hg in supine position with a postural fall of 30 mm Hg in sitting from supine position, respiratory rate 16/min, regular without any respiratory embarrassment. There was minimal pedal oedema. On neurological examination higher mental functions and cranial nerves were normal. Power in upper limbs was 4/5 and in lower limbs 3/5 proximally and 4/5 distally. Deep tendon reflexes were diminished in upper limbs and absent in lower limbs with bilateral flexor plantar response. Sensory and cerebellar examination was normal except subjective complaint of calf pain and acral paraesthesias. There were no signs of meningeal irritation.

On investigations, Hb was 6.6 gm%, TLC 6000/cumm with neutrophils 56%, lymphocytes 42% and eosinophills 2%, ESR 10 mm in 1st hour. Peripheral blood film showed heavy parasitaemia with trophozoites of *P. falciparum*. Hepatic and renal function tests were normal. Fasting blood sugar was 74 mg% with post prandial blood sugar (2 hour) of 136 mg% and urine examination was normal including urine for porphobilinogen. Nerve conduction studies performed were suggestive of demyelinating polyneuropathy and CSF cell counts were normal and protein was 76 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 quinine di-HCl 400 mg stat in 25% GDW 100 ml over 1/2 hour followed by 600 mg in 10% GDW 500 ml over 3-4 hours every eight hourly with 25% GDW 200 ml eight hourly to prevent hypoglycaemia. Patient was able to tolerate oral quinine on fourth day so was put on oral quinine 400 mg tds upto a total of seven days. He was given tab. primaquine 45 mg on fourth day. Patient showed improvement on third day of starting specific treatment and was able to walk on seventh day. Patient was discharged on 14th day with haematinics. On follow up examination after four weeks patient had no neurological deficit.

**DISCUSSION**

Our case report represents the second category of neuropsychiatric manifestations of *P. falciparum* malaria in which predominant neuropsychiatric signs and symptoms occur without loss of consciousness during acute stage as a...
The exact pathogenesis of acute polyneuropathy in falciparum malaria is not known but has been attributed to immune mediated capillary damage, toxic oxygen radicals, tumour necrosis factor, parasitic emboli obstructing the vasa nervorum, neurotoxin release, nutritional and metabolic disturbances. The aim of reporting this case is to make awareness about this potentially fatal but treatable disease if specific treatment is initiated early and because of paucity of such cases in literature.

REFERENCES