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CASE REPORT

Guillain-Barre Syndrome Following Falciparum Malaria: A Rare Association

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ABSTRACT

Malarial parasite as a cause of Guillain-Barré syndrome (GBS) is very rare and there are only a very few case reports in literature. Here we report a case of acute flaccid quadriparesis with generalised areflexia following malaria caused by P. falciparum. An adult male developed acute lower motor neuron type of quadriparesis following an episode of microscopically proven Plasmodium falciparum malaria. Clinical examination, cerebrospinal fluid analysis, and nerve conduction studies confirmed the diagnosis of GBS, depicting the rare association of GBS following falciparum malaria.

INTRODUCTION

Antecedent viral infection as a cause of Guillain-Barré Syndrome (GBS) is well-established[1]. Neurological complications such as cerebellar disturbances, bulbar paralysis, cerebral malaria[2], cranial nerve lesions, extrapyramidal tremor, transient paranoid psychosis and febrile convulsions are frequently seen following plasmodium falciparum malaria[3]. GBS as a complication of falciparum malaria is rare and only sporadic cases of acute polyneuropathy following plasmodium falciparum and vivax infections have been documented.[4,5,6]

CASE REPORT

A 25-year-old male presented to our emergency room with 6 days history of high-grade fever with chills, rigors and headache. Fever was intermittent type and used to subside with profuse sweating. On admission, his temperature was 39.7°, he was conscious and oriented, the liver was not palpable and tip of spleen was just palpable. The central nervous system was normal on examination. There was no preceding sore throat, cold or cough. He had suffered one episode of microscopically proven Plasmodium falciparum malaria 2 years back.

Laboratory investigations showed haemoglobin-8 gm/dl, TLC-5200/mm 3, DLC-$N^{65}L^{27}E^{4}M^{4}$, ESR-30 mm in 1st hour, blood sugar (Fasting)-65 mg/dl, blood urea- 25 mg/dl, serum creatinine- 0.86 mg/dl, Serum Na^{+} -136 mmol/l, Serum K^{+} -3.9 mmol/l ; Blood smear showed ring form of P. falciparum. Patient was treated with inj. artesunate & fever subsided on day 2& patient was clinically well thereafter 6 days.

On the 7th day of admission, the patient developed rapidly progressive motor weakness in both the lower limbs which ascended to involve the upper limbs within 12 hours. There was no history of unconsciousness, trauma, abnormal movements, bowel and bladder disturbance, facial weakness, difficulty in vision/speech/swallowing, root pains or recent vaccination/ surgery/ major trauma/ diarrhoea/ respiratory tract infection within last 3 months. Examination revealed generalised hypotonia of all four limbs; power was diminished in the hips, knees and ankles to Medical Research Council (MRC) grade 3. Power was also diminished in the shoulders, elbows and hands to MRC grade 3. There was no fasciculation or wasting. Sensations were intact in all 4 limbs. The ankle jerks were absent. All the other reflexes were decreased. Plantar response was flexor bilaterally. Cranial nerve examination and fundoscopy were normal. His pulse and blood pressure remained stable, resp.rate-22/min; single breath count-above 30.Clinical diagnosis of probable Guillan Burre Syndrome was made considering the coexistence of acute flaccid quadriparesis and hyporeflexia.

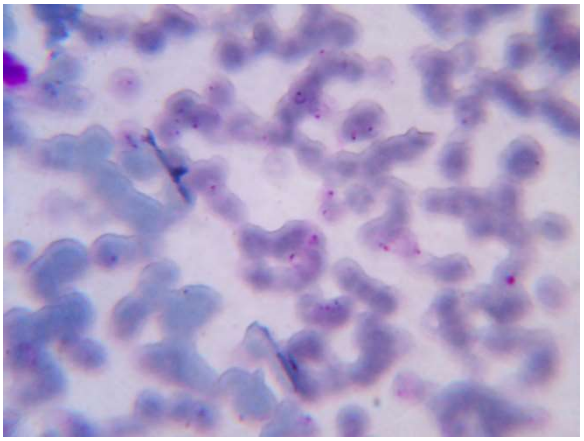

Fig 1: Blood Smear showing ring form of P. falciparum

Investigations showed fasting blood sugar 90mg/dl, sodium 138 mmol/l, potassium 4.4 mmol/l and blood urea 30mg/dl,Cr-0.7mg/dl. Cerebrospinal fluid (CSF) study was done which was clear in appearance and revealed cell count-2/mm^3; all lymphocytes; protein-86mg/dl; sugar-36mg/dl. X-ray chest was normal. Nerve conduction study (NCS) was done which showed prolongation of distal latencies in motor nerves, decrease in compound motor action potential (CMAP) in all nerves, temporal dispersion in all nerves, prolongation of F wave latencies, and conduction blocks in few nerves which was suggestive of motor-sensory primary demyelinating and secondary axonal pathology, characteristic of Guillain Barre Syndrome. Patient was immediately started on inj. Intravenous Immunogbulin (IVIG) based on the diagnosis of probable GBS and physiotherapy was commenced. Repeat CSF study was done on 20th day of admission which was clear in appearance, lymphocytes- 4/mm^3, proteins-160 mg/dl,sugar-56mg/dl.GBS was confirmed on the basis of clinical finding, supportive NCS & CSF finding. IVIG course in recommended dosages was completed. Patient was closely observed for subsequent days. No further clinical deterioration found. Patient improved clinically and was discharged after 10 days of confirmation of GBS. Patient regained almost full power within 3 months with slight residual deficit.

DISCUSSION

The neurological signs in this patient suggested lower motor neurone paralysis. Elevated proteins and the absence of cells in the cerebrospinal fluid also suggest Guillain-Barre syndrome, which was confirmed by NCS study. Guillan Barre Syndrome, manifested by areflexia/hyporeflexia with flaccid limb weakness, is an Acquired immune Polyradiculoneuropathy (AIDP). It has been previously reported following immunization,[7] surgical operations, upper respiratory tract infections, Mycoplasma pneumoniae[8] and viral infections such as Epstein-Barr, varicella, cytomegalovirus, , measles, mumps and hepatitis.[9] Viruses are usually the most common organisms responsible for causing GBS. Infections by P. Falciparum and P. vivax [4,5,6] have been documented to cause GBS in few sporadic cases although the exact pathogenesis of GBS following malaria infection is not known, but is likely to be immunogenic like that occurring after vaccination and viral or bacterial infections. Another mechanism suggested for the development of polyneuropathy following a parasitic infection is that the malaria parasite may damage peripheral nerves by vascular occlusion, thus causing anoxemia in the vasa nervosum leading to temporary demyelination and often recovery after disappearance of parasitaemia and re-establishment of normal blood flow in vasa nervosum . Release of neurotoxins, associated metabolic and nutritional disturbance, immune-mediated capillary damage, release of free radicals and tumour necrosis factor may also be the culprits in the pathogenesis of GBS after P. Falciparum infection malaria.[10,11] Usually GBS develops quickly when the patient has been previously exposed to malaria by a memory cell mediated immune response. In our patient also the features of acute polyradiculoneuropathy developed after an acute attack of P. falciparum and no history of other precipitating factors like diarrhoea/ respiratory tract infections, etc were elicited, therefore it is reasonable to assume a causal relationship. The short delay between infection and onset of neurological symptoms may be attributed to the prior suffering of malaria 2 yr back[4]. A drug-induced polyneuropathy is unlikely as artesunate which this patient received are not known to produce polyneuropathy yet either by direct neurotoxicity or idiosyncrasy. In conclusion GBS following malaria infection may occur and even earlier than GBS following viral infection.

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