Journal of Research in Medical and Dental Science
2023, Volume 11, Issue 1, Page No: 299-300

Available Online at: www.jrmds.in
eISSN No.2347-2367: pISSN No.2347-2545

A Typical Presentation of Malaria with Stroke

Manimekalai Periyasamy*, Pon Divya Bharathi, Vinatha, Kannan Meera Devi

Department of General Medicine, Sree Balaji Medical College and Hospital, Chrompet, Chennai, India

ABSTRACT

Malaria is one of the most common vectors borne disease in many parts of the world. It is also an endemic parasitic disease in India. Malaria caused by Plasmodium falciparum can affect the central nervous system and the patient can present with encephalitis which can ultimately lead to neurological squeal or death. But presentation of stroke with lesion is quite rare. The mechanism involved can be mechanical or humeral. This article is a case report of a malaria patient presented with stroke and had a lesion in the middle cerebral artery territory and had no signs of encephalitis, mostly probably due to the mechanical theory.

Key words: Malaria, Stroke, Cerebral malaria encephalopathy, *Plasmodium falciparum*, Parasitic disease

HOW TO CITE THIS ARTICLE: Manimekalai Periyasamy, Pon Divya Bharathi, Vinatha, Kannan Meera Devi, A Typical Presentation of Malaria with Stroke, J Res Med Dent Sci, 2023, 11 (1): 299-300.

Corresponding author: Dr. Manimekalai Periyasamy
E-mail: mekalap32@gmail.com
Received: 29-Aug-2022, Manuscript No. JRMDS-22-75747;
Editor assigned: 01-Sep-2022, PreQC No. JRMDS-22-75747 (PQ);
Reviewed: 15-Sep-2022, QC No. JRMDS-22-75747;
Revised: 03-Jan-2023, Manuscript No. JRMDS-22-75747 (R);
Published: 16-Jan-2023

INTRODUCTION

Malaria stills has a high prevalence rate across the world and also associated with increased number of mortalities. The reason for such high rates is due to the development of resistant strains to the antimalarial drugs [1]. Infection with Pla*smodium falciparum* is associated with high mortality as compared to other species of *Plasmodium*. This is because, the disease caused by falciparum is severe in most cases and also it affects the central nervous system and causes cerebral malaria encephalopathy with long term neurological sequelae. Though the host and vector factors, clinical, environmental and therapeutic factors of the disease are widely studied, the pathophysiology is still not completely understood. This article focuses on the neurological manifestation and its probable mechanism.

Malaria has a wide range of presentation. Hence, all the atypical presentation of malaria should also be known and studied in order to diagnose correctly, treat accordingly, to prevent complication and to reduce disability.

CASE PRESENTATION

A 32-years-old male, from Jharkhand, came to the casualty with the complaints of fever with chills for three days, vomiting for 1 day and no other specific complaints, no known comorbidities. He gave travel history to his home town in Jharkhand one week ago. No significant personal and family history. On examination he was very weak, vitals were stable, and all systemic examination was normal but had reduced power in the left upper limb. Suspecting malaria, peripheral smear was done. Malarial parasite was isolated in thick smear and *Plasmodium falciparum* was identified in thin smear. Malaria was diagnosed and the patient was admitted. On the next day of admission, the patient suddenly developed left sided muscle weakness and loss of strength. Neurological examination showed increased tone (spastic type) and exaggerated reflexes with babinski sign positive on the left side. Computerized axial tomography of the brain showed ischemic lesion near the internal capsule in the middle cerebral artery territory. All other blood investigations including the complete blood count, clotting tests, fasting lipid profile, erythrocyte sedimentation rate and fibrinogen was normal. Serology for syphilis was negative. Chest radiograph, echocardiogram, EKG and carotid doppler was normal. ANA, ANCA, APLA, RF and complements C3, C4 were all normal. The patient was treated with antimalarial artesunate (2.4 mg/kg IV at 0, 12 and 24 h; then once daily). Following treatment and physiotherapy, fever settled but the hemiparesis persisted. After a year of follow up, his neurological examination remains unchanged and physiotherapy is still continued (Figure 1).

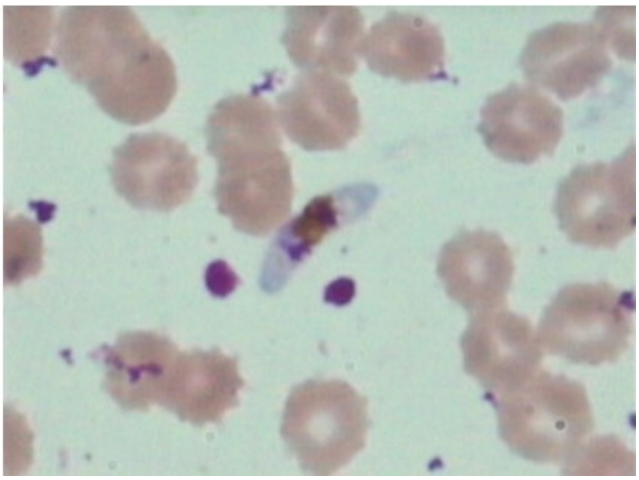

Figure 1: Peripheral blood film showing the gametocyte of *Plasmodium falciparum.*

DISCUSSION

People in endemic states, children, pregnant women and immune compromised people are more prone to develop severe malaria. Central nervous system involvement in a case of malaria can be due to mechanical or humoral effects.

Mechanical effects include the obstruction of the cerebral capillaries and venules by the parasite infested red blood cells leads to change in the morphology, elasticity and plasticity of the erythrocytes thus, changing its physical properties. Reduced capillary blood flow and endothelial dysfunction leads to increased risk of thrombosis, tissue necrosis and stroke. Endothelial damage can also increase the permeability leading to increased chances of haemorrhages [2].

Humoral effects include the effects of the vasoactive inflammatory mediator which are non-specific such the tumour necrosis factor and interleukin 2, increase the capillary permeability; decrease the blood flow to the tissues thus causing cellular hypoxia. Due to exaggerated inflammatory reaction towards the central nervous system leads to cerebral malaria which was proved by many clinical trials including both animal and human models. Repeated exposure to the parasite can produce immunity towards the infection thus causing a mild form of disease or no disease.

Neurological manifestation in case of cerebral malaria encephalitis presenting as stupor or coma, decerebration, decortication postures, convulsions and retinal haemorrhages. Neuroimaging may show signs of cerebral oedema and diffuse petechial haemorrhage mostly ring shaped around the arterioles [3]. Less common features such as benign intracranial hypertension, intracranial haemorrhages, psychiatric and extrapyramidal symptoms might occur [4]. Multisystem involvement often present, such as pulmonary oedema, renal failure, hepatic dysfunction, severe anaemia, metabolic acidosis, sepsis and shock. Though the manifestations might be severe, the patients who survive have a very good prognosis and most of the cases fully recover within a year [5,6].

CONCLUSION

This case report about a young male with no known comorbidities and normal coagulation profile but from an endemic region of malaria developed stroke lesion in the brain after the onset of febrile phase of malaria without any features of encephalitis is highly suggestive of involvement of malarial parasite infested erythrocyte and its mechanical effect on the cerebral small vessels. Though the surviving patients in a case of cerebral malaria have a good prognosis, in this case the resulting brain damage was focal and permanent.

REFERENCES

1. Singh UK, Kumar R, Sharma VK. Increased urinary frequency as a presentation of *Plasmodium falciparum* malaria. Pediatr Infect Dis J 1994; 13:1024.
2. Patnaik JK, Das BS, Mishra SK, et al. Vascular logging, mononuclear cell margination and enhanced vascular permeability in the pathogenesis of human cerebral malaria. Am J Trop Med Hyg 1994; 51:642-647.
3. Miller LH, Good MF, Milon G. Malaria pathogenesis. Science 1994; 264:1878-1883.
4. Warrell DA. Cerebral malaria. Schweiz Med Wochenschr 1992; 122:879-886.
5. Leopoldino JF, Fukujima M, Gabbai AA. Malaria and stroke: Case report. Arquivos de neuro-psiquiatria 1999; 57:1024-1026.
6. Yacoub S, Lang HJ, Shebbe M, et al. Cardiac function and hemodynamics in Kenyan children with severe malaria. Critical care med 2010; 38:940-945.