Plasmodium Falciparum Malaria Presenting With Lenticulostriate Infarct In A Young Male

Vikram Amale

Abstract

Malaria is a parasitic disease with high prevalence in several regions of the world. Cerebral malaria is a complication of plasmodium falciparum malaria and can result in various neurological manifestations. The neurological manifestations of cerebral malaria secondary to vascular occlusion and infarct are rare.

We report a rare case of lenticulostriate infarct in a young male with plasmodium falciparum infection. Our case is unique in that we have a young 16 yrs old male with a documented plasmodium falciparum infection and developing hemiparesis while he was still febrile but not showing any signs of encephalitis or cerebral malaria.

Keywords: Cerebral malaria; Lenticulostriate infarct; Plasmodium falciparum.

Introduction

Almost three out of every four people in the world who suffer a fatal stroke live in developing countries. Tropical infectious diseases account for up to 10% of causes of stroke in adults [1]. The important infections include malaria, Chagas disease, neurocystisercosis gnathostomiasis, viral hemorrhagic fevers due to arenavirus and flavivirus.

Cerebral malaria is the most severe neurological complication of plasmodium falciparum malaria [2]. Cerebral malaria can cause cerebral edema, diffuse or focal compromise of the subcortical white matter and cortical, cerebellar and pontine infarctions [1]. Cerebral malaria generally presents as encephalitis and does not usually cause focal neurological dysfunction. We present a rare case of falciparum malaria manifesting as left lenticulostriate infarct in a young male without any contributing factors. To the best of our knowledge only four such cases are reported in literature secondary to P. falciparum infection and ours is the fifth case. Knowledge about this rare entity may help in having a degree of suspicion in such cases.

Case report

A 16 year-old male patient with no known co morbidities and no significant past history was admitted in a private hospital with complaints of fever, headache and generalized weakness for past 2 days. While he was being investigated in the hospital he complained of severe headache which was followed by brief period of unconsciousness. Immediately CT scan of head was done which was apparently normal. Within few minutes the patient regained consciousness but he was drowsy, restless and did not follow any verbal command, opening eyes only to deep painful stimuli, aphasic with decreased movement on right side of the body. There was no history of hypertension, diabetes mellitus or previous stroke. The patient was a nonsmoker. A provisional diagnosis of cerebral malaria was made and then he was shifted to our hospital. He was further investigated and blood tests revealed that he was positive for plasmodium falciparum antigen peripheral smear also showed few rings of plasmodium falciparum. Platelet count of 132×103 mm3, WBC count of 2.8 ×103/mm3.Vitamin B12 level 119.2 pg/ml & serum homocysteine 48.52 µmol/l. Tests for plasmodium vivax, dengue, HIV were negative. Electrocardiogram, chest x-ray, echocardiography and ultrasound examination of the abdomen were normal. Magnetic resonance imaging of the brain showed evidence of acute infarction in the left lentiform nucleus, caudate nucleus, internal capsule, insular cortex with no evidence of hemorrhage or encephalitis (Fig. 1).

The rest of the brain parenchyma and both lateral ventricles appeared normal. CT-angiography of cerebral vessels were within normal limits. Fundoscopic examination was normal. A final diagnosis of acute p. Falciparum malaria with ischemic stroke was made.

The patient was treated with inj Artesunate; cap Doxycycline, Aspirin, Paracetamol, Mannitol, Primquine and other supportive therapy. The patient showed improvement and became afebrile within two days. The patient improved marginally and was finally discharged on the 12th day of hospitalization with residual weakness on right side, facial weakness and non fluent aphasia.

Discussion

Cerebral Malaria is the
most important course of *P. falciparum* infection with mortality rates of up to 50% [3]. The World Health Organization’s clinical definition of cerebral malaria includes the following: a Blantyre coma score ≤ 2, *P. falciparum* parasitaemia by blood film, and no other evident cause of coma (e.g. meningitis, post-ictal state, hypoglycaemia) [4]. The patho physiological mechanisms of the cerebral lesion are not totally clear. In cases with cerebral malaria with neurological involvement there may be role of nonspecific vasoactive substances that lead to deranged tissue permeability causing cerebral edema and other manifestations [5, 6, 7, 8, 9]. The other theory is of simple mechanical block caused by the deformed erythrocytes that may give rise to localized infarcts leading to focal neurological deficits [10, 11].

In our case, since there was no other risk factors like hypertension, diabetes mellitus, smoking, dyslipidemia and any history of previous stroke, the possibility of this stroke being a chance occurrence with *P. falciparum* malaria in a young patient is highly unlikely. The development of neurological dysfunction well after the onset of fever and the rapid improvement in the level of consciousness, followed by steady improvement in neurological dysfunction, after starting the antimalarial treatment strongly suggest that the ischemic stroke was mainly due to cytoadherence and sequestration of erythrocytes containing the parasites in the lenticulostrate arteries which are end arteries. Moreover young age of the patient and normal fundoscopic findings argue against the selective development of atherosclerosis in the lenticulostrate territory predisposing the patient to ischemic stroke. Therefore in all probability, heavy parasitemia appears to be the crucial factor in the development of stroke, not the process of atherosclerosis. Homocysteine is believed to cause atherogenesis and thrombogenesis via endothelial damage, focal vascular smooth muscle proliferation probably causing irregular vascular contraction, and coagulation abnormalities [12]. Although our patient had high homocysteine level but whether that contributed in the development of stroke in the given clinical situation remains elusive. Thus the effect of hyperhomocysteinemia in patient with falciparum malaria and stroke needs further evaluation. As the patient was out of window period when he reached our hospital we did not thrombolyse him but the role of thrombosity in such patient needs further studies. During our literature review we could find four such cases secondary to plasmodium falciparum infection. Kampf et al. reported a case compatible with secondary pontine myelinolysis which was expressed by vertical nystagmus. The diagnosis was made by magnetic resonance and became a landmark, so rare are reports of focal forms in the literature [13]. Another case report is from Brazil where a 28 year old man developed hemiparesis during febrile illness and was diagnosed as malaria with stroke but they could not confirm whether the febrile illness was malaria [14]. Another case report is from India where a 47 year old male with plasmodium falciparum malaria developed vertebrobasilar stroke [15].

Fourth case is also reported from India where 26 year old male developed bilateral mid brain infarcts secondary to falciparum malaria [16]. Similar cases are also reported secondary to *Plasmodium vivax* infection but may be rare compared to *P falciparum* [17, 18].

Our case is unique in that we have a young 16 yrs old male with a documented plasmodium falciparum infection and developing hemiparesis while he was still febrile but not showing any signs of encephalitis or cerebral malaria. To conclude that in endemic areas of malaria or given a history of a recent visit to such an area, in a case of fever presenting with features suggestive of stroke, a malarial etiology should also be considered.


Conflict of Interest: Nil
Source of Support: None