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Case Report

Study of Computed Tomography in Patients of Cerebral Malaria

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Abstract

Cerebral malaria is a life-threatening complication of malaria seen in around 2% of all malaria cases, particularly in Plasmodium falciparum (P. falciparum) infection. There is an estimated mortality rate of between 15% and 25% in this state even with appropriate treatment and intensive care. However, patients who survive from this lethal complication often recover fully with no long-term consequences. Early diagnosis and treatment is hence crucial to obtain the best outcome. With the availability of brain imaging facilities in developing countries, where malaria is still an endemic health issue, a few case reports and series have already been published on the role of brain imaging in cerebral malaria but there is overall scarcity of literature on this. This study was done to look for the changes appearing in computed tomography scan in patients of cerebral malaria.

Keywords: Cerebral Malaria; CT Scan

Introduction

Malaria is one of the most common and dreaded diseases in the tropical countries including India. It is transmitted by the bite of female Anopheles mosquito and caused by Plasmodium species [1]. The infection caused by *Plasmodium falciparum* is the most lethal and is responsible for a number of deaths worldwide [2]. Cerebral malaria manifests as a varying clinical symptom ranging from mild drowsiness to different focal neurological deficits and deep coma. Once the diagnosis of cerebral malaria is set up, imaging techniques in the form of CT scan and MRI offer great help in determining the areas of brain involved in this pathology. Thus, a prospective study was conducted on all the patients being diagnosed as cerebral malaria and CT scan was performed in all such patients. The results obtained were analyzed to look for the extent and severity of brain involvement in these cases.

Aim of study

To study the role of computed tomography in patients of cerebral malaria.

Material and Methods

Eight males and two female patients, whose age ranged from 15-80 years and satisfying WHO criteria for diagnosis for cerebral malaria were studied over a period of two years. Other diagnosis such as viral or bacterial meningoencephalitis, tubercular meningitis, hepatic encephalopathy, uraemia, drug intoxication, past history of stroke, diabetes and hypertension were ruled out. Clinical assessment including detailed neurological and funds examination and lumbar puncture examination were performed before CT scan. The CT scanner used was spiral GE systems.

Results

Out of the ten patients studied, eight (80%) had normal CT scans and none of them died. None of these had papilloedema or retinal haemorrhages. Also, all these had normal CSF opening pressures. The rest two patients (20%) had abnormal CT scans. Patient one clinically had left sided hemiparesis with left supranuclear facial palsy, left 6th nerve palsy, simple partial seizures and aphasia. Fundus and CSF examination was normal. CT scan showed acute infarct in right parieto occipital area with surrounding oedema. Repeat CT scan done one week later showed

increase in infarct size and oedema. Patient eventually died. Patient two has Acute Disseminated Encephalo Myelitis (ADEM) like presentation. Patient had paraparesis. Fundus showed retinal haemorrhage and CSF pressure was raised. CT scan suggested bilateral periventricular cerebral oedema. This patient also died later.

Discussion

Cerebral malaria is a serious life-threatening complication of *P. falciparum* infection. This infection is acquired after being bitten by an infected female Anopheles mosquito and is a multisystem disease, with hepatic dysfunction, thrombocytopenia, and coagulopathies [3]. Neurological manifestations occur in 2% of all cases of malaria. Cerebral involvement in malaria is due to vascular sequestration of parasitized erythrocytes. A functional blockade of the microcirculation is the main pathogenetic mechanism in this pathology and therefore diffuse bihemispheric dysfunction is the most common manifestation of cerebral involvement [4]. Patients become drowsy and disoriented; hallucinations are also seen. As the disease process advances, the level of consciousness deteriorates and the patient becomes comatose [2].

Diagnosis of cerebral malaria requires demonstration of asexual form of *P. falciparum* in peripheral blood smear, in thick and thin blood smear films which are stained by Giemsa stain. Absence of parasites in some patients may be due to sequestration of the parasitized RBCs in cerebral circulation or earlier treatment with antimalarial drugs. In such situations, at least 3 smears 6 h apart should be examined. At least 3 smears then should be negative before excluding cerebral malaria. The rapid diagnostic test (antigen detection test) and PCR may be helpful in the diagnosis. CSF examination is necessary to exclude other causes of febrile encephalopathy. CSF is generally normal in cerebral malaria; however, mild pleocytosis (10-50 cells/mm^3) and protein rise up to 200 mg/dL may be seen. EEG shows nonspecific abnormalities, such as diffuse slowing, spike wave discharges, and burst suppression pattern. Cerebral malaria may occur rarely with negative blood smear; therefore, a high index of suspension is required [5].

CT and MRI are usually normal or show edema and cortical or sub cortical infarcts in watershed zone in 15%- 20% patients. Cordoliani, et al. [3] reported imaging findings in three cases of cerebral malaria. A cortical infarct was seen in one case, with hyper intense areas in the white matter on T2W and FLAIR images in the other two cases. The hyper intense white matter areas were either diffuse (which was attributed to white matter edema) or focal (probably due to gliosis) [6]. In a prospective study of 20 patients with cerebral malaria, brain scan showed that the brain volume during acute cerebral malaria was slightly more than during the convalescent phase of the disease [7]. This difference was attributed to an increase in the volume of intracerebral blood caused by sequestration of parasitized erythrocytes and compensatory vasodilatation, rather than by edema. Various reports of MRI in cerebral malaria have revealed focal or diffuse signal changes in centrum semiovale [3,8,9], corpus-callosum [3,10], thalamus and insular cortex [11]. Central pontine myelinolysis [12], myelinolysis in the upper medulla [13], cerebella syndrome with demyelization, and micro infarcts of the cerebella hemispheres have also been reported. In these, hyper intensities on T2 weighted or Fluid-Attenuation Inversion Recovery (FLAIR) images were considered to be due to oedema, ischaemia, toxic injury or gliosis. Hemorrhages based on gradient-echo imaging in single cases were described by Gupta, et al. [11] and Nickerson et al [6]. The lesions were mainly in the front parietal lobe, corpus callosum and internal capsule. Sakai et al [12] described per ventricular and sub cortical white matter restricted diffusion on the DWI, but did not reveal hemorrhagic components. Koch, et al. [14] reported a case of acute symptomatic psychosis occurring 2 weeks after successful therapy of a *P. falciparum* infection. MRI revealed multiple hyper intense lesions on the T2W images. They attributed these to an immune-mediated complication (acute disseminated encephalomyelitis, ADEM) rather than a direct result of the infection.

The ADEM like presentation seen in the second case are most likely due to the edema of acute encephalitis. Although reported brain imaging features of cerebral malaria are variable, the diagnosis of cerebral malaria should be strongly considered when acute hemorrhagic infarctions, particularly in the thalami, are encountered in patients with constitutional symptoms with or without history of travel to an endemic area.

Conclusion

In patients presenting with non-localizing signs and/or generalized tonic clonic convulsions, CT scan is usually normal and hence not indicated.

CT findings in patients presenting with focal neurological deficit and/or partial seizures may reveal areas of altered density (infarct) and/or cerebral oedema, which usually develops in the terminal stage in most fatal cases.

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