Retinal Changes in Cerebral Malaria among Sudanese Patient in Khartoum State, Sudan 2019


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Abstract- Introduction: It is thought that Malaria parasites live in red blood cells and make them stick to the inside of small blood vessels, particularly in the brain and also the eye. The light-sensitive tissue in the eye is also affected because the parasites disrupt the supply of oxygen and nutrients. These changes, known as malarial retinopathy, include white, opaque patches, whitening of the infected blood vessels, bleeding into the retina and swelling of the optic nerve.

Objectives: Our study was aimed to demonstrate malarial retinopathy in patients presented with neurological manifestations of malaria. Methodology: A cross-sectional Hospital based study included all patients with malaria seen during the period between 1-1-2019 and 25-4-2019.

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Objectives: Our study was aimed to demonstrate malarial retinopathy in patients presented with neurological manifestations of malaria.

Methodology: A cross-sectional Hospital based study included all patients with malaria seen during the period between 1-1-2019 and 25-4-2019.

Result: Almost 40 patients with neurological manifestations of malaria were included in the study, the majority of them had cerebral malaria. Out of 40 patients malaria retinopathy was detected in seven children and three adults all of them had cerebral malaria.

Conclusion: The eye can provide a very reliable way of diagnosing cerebral malaria.

I. Introduction

Malaria had been and still cause much of human’s morbidity and mortality. It is a systemic disease caused by protozoan called Plasmodium. Four distinct species infected humans: P. falciparum, P. vivax, P. ovale and P. malariae. The species differ in regards to their morphology, details of their life cycles, and their clinical manifestations. Malaria has a group of classical clinical manifestation which include fever, headache, generalized fatigability, nausea and vomiting. The severity of the attack depends on the Plasmodium species as well as other circumstances such as the state of immunity and the general health and nutritional status of the infected individual.

Non neurological clinical manifestations of Malaria: There are many non neurological manifestation of Malaria and they include dysenteric Malaria, biluoric Malaria, Malaria induced hepatitis, Malaria pneumonitis, black water fever and algid Malaria. Malaria can cause thrombocytopenia and pulmonary edema.

Several neurological manifestations of malaria have been reported. Cerebral form of malaria is the most common and potentially life threatening neurological complication. Despite adequate treatment, 10% to 18% of survivors develop neurological sequelae in the form of psychosis, ataxia, hemiplegia, cortical blindness, aphasia and extrapyramidal syndrome. Post-malaria neurological syndrome is a discrete, transient neurological syndrome seen after recovery from severe malarial infection. Cerebellar involvement can occur with malarial infection. Malaria can give rise to Guillain-Barre syndrome like presentation, mononeuritic syndromes such as facial palsy, trigeminal neuralgia, retrobulbar optic neuritis, peripheral neuropathy, proximal myopathy, cerebral infarction ,cerebral hemorrhage, sagittal sinus thrombosis and six nerve palsy. Neurological manifestations can be cause by anti malarial drugs eg ataxia ,slurring of speech and convulsion have been described after artesunate treatment for falciparum malaria.

Malaria parasites live in red blood cells and make them stick to the inside of small blood vessels. Cerebral malaria is accompanied by changes in the retina. These changes, known as malarial retinopathy, include white, opaque patches, whitening of the infected blood vessels, bleeding into the retina and swelling of the optic nerve.

II. Objectives

Our study is aimed to demonstrate malarial retinopathy in Sudanese patients presented with neurological manifestations of malaria seen in Omdurman Teaching Hospital and Ibrahim Malik Teaching Hospital in the period between 1-1-2019 and 25-4-2019.
III. Methodology

a) A cross-sectional Hospital based study

Time of Study: The period of study was from 1-1-2019 and 25-4-2019.

Study population: The study included patients with malaria and neurological complications who were admitted in the hospital and those who were presented in the Emergency room or refer clinic. The study population consisted of 100 patients with malaria and neurological complications seen during the period of the study.

Inclusion criteria: All patients diagnosed as having malaria with neurological manifestations during the study period.

Exclusion criteria
1. Those who refused to participate in the study
2. Pregnant females
3. Diabetic and Hypertensive patients.

Study Tools
A questioner was designed for collecting data from patients, it consist of full detailed history, proper clinical examination and laboratory investigations. The history includes:
1. Personal data: name, age, sex, residence, marital state, education, occupation.
2. Symptoms of classical manifestations of malaria: fever, headache, joint pain, generalize fatigability, nausea and vomiting.
3. Symptoms of non neurological manifestations of malaria: dysenteric malaria, choleric malaria, bilious malaria, pneumonic malaria, malarial hepatitis, algid malaria, black water fever.
5. Clinical examination of the patients: whether the patient is fully concuss oriented in time person and place. Assessment of both recent and remote memory. Examination of cranial nerves looking for evidence of cranial nerves palses. Fundal examination looking for evidence of malarial retinopathy. Examination of the neck for neck weakness or stiffness. Upper and lower limb examination looking for evidence of hemiplegia, quadriplegia, proximal myopathy, peripheral neuropathy and cerebellar ataxia laboratory investigations: blood film for malaria (thin and thick), complete blood counts, liver and renal function tests.

Data analysis: All the collected data was finally were entered the computer using statistical package program for social science (SPSS) to analyze the data. Results were expressed in numbers and percentage (tables and graphs), represented statically and then discussed.

b) Result
Almost 40 patients with neurological manifestations of malaria were included in the study(29 having cerebral malaria, 3 with post malarial cerebellar ataxia, 2 with post malarial syndrome and abnormal movement, one with peripheral neuropathy, one with proximal myopathy, one had cerebral infarction, one had cerebral haemorrhage, one had sagital sinus thrombosis and one had six nerve palsy. Out of 29 patients with cerebral malaria 14 were children and 15 were adult. Malarial retinopathy changes were detected only among those with cerebral malaria (7 children and 3 Adult).

IV. Discussion

Malaria has been and still is the cause of much morbidity and mortality. Malaria is caused by members of the genus Plasmodium. Plasmodium species are apicomplexa and exhibit a heteroxenous life cycle involving a vertebrate host and an arthropod vector. Four distinct species infected humans: P. falciparum, P. vivax, P. ovale and P. malariae. The species differ in regards to their morphology, details of their life cycles, and their clinical manifestations. 3 Plasmodium infection causes an acute febrile illness which is most notable for its periodic fever paroxysms occurring at either 48 or 72 hour intervals, the fever is associated with generalize fatigability, joints pain. Non neurological manifestations of malaria include bilious malaria characterize by excessive vomiting, Dysenteric malaria which mimic dysentery and present with diarrhea and tensmus. When malaria present with profuse watery diarrhea it is called choleric malaria, looks like cholera. Sever malaria is often associated with other serious systemic complications hypoglycaemia, hyperpyrexia, severe anaemia, shock, renal failure and pulmonary oedema.

Several neurological manifestations of malaria have been reported. Cerebral form of malaria, because of severe falciparum infection, is the most common and potentially life threatening neurological complication. Cerebral malaria is characterized by un arousable coma. Loss of consciousness can develop very rapidly, convulsions are frequent, especially in children. The prognosis of cerebral malaria is poor, even under optimal conditions of care. In majority of the cases, examination of thick and thin films of the peripheral blood will reveal malarial parasites. Deaths from cerebral malaria usually occur within first 24 hours of admission to the hospital, but occasional late deaths are also encountered. Some patients make a rapid recovery but
deterioration in conscious level and recurrent episodes of convulsions and hypoglycaemia may occur after an initial period of apparent improvement. Despite adequate treatment, 10% to 18% of survivors develop neurological sequelae in the form of psychosis, ataxia, hemiplegia, cortical blindness, aphasia and extra pyramidal syndrome. These sequelae are more common in children.

Post-malaria neurological syndrome is a discrete, transient neurological syndrome seen after recovery from severe infection. Criteria for inclusion under this syndrome are: recent symptomatic malarial infection with parasites cleared from blood, full recovery of consciousness in cases of cerebral malaria and the development of new neurological or psychiatric symptoms within two months of acute illness. The syndrome was self limiting.

Cerebellar syndromes in malaria: Cerebellar involvement is the most consistent neurological manifestation of complicated as well as of uncomplicated malaria. Severe gait and truncal ataxia are striking features suggesting that the disease predominantly affects midline cerebellar structures. The majority of patients have a febrile period before the onset of cerebellar symptoms. It is always associated with Plasmodium falciparum infection. The exact mechanism of delayed cerebellar ataxia is unknown, however, there is some evidence to suggest involvement of immunological factors in the pathogenesis. Malaria can give rise to Guillain-Barre syndrome like presentation, mononeuritic syndromes such as facial palsy, trigeminal neuralgia, retrobulbar optic neuritis, cerebral infarctin, cerebral haemorrhage, sagital sinus thrombosis, proximal myopathy and cranial nerves palsies especially the six cranial nerve.

Neurological manifestations can be due to antimalarial drugs, prolonged administration of this drug may cause a vacular myopathy, the quinine and quindine may be associated with tinnitus and high-tone deafness. Increased incidence of convulsions in artemether treated group was reported. Ataxia and slurring of speech have been described after artesunate treatment for falciparum malaria. Malaria parasites live in red blood cells and make them stick to the inside of small blood vessels, particularly in the brain and also the eye. It is thought that this causes the unique whitening of eye blood vessels. The light-sensitive tissue in the eye is also affected because the parasites disrupt the supply of oxygen and nutrients.

In patients with cerebral malaria small cerebral vessels are packed with parasitized red blood cells. Electron-dense knobs are present on the surface of parasitized red blood cells, close to their point of contact with endothelial cells. Numerous patechial ring haemorrhages are seen in the white matter. These haemorrhages result from the rupture of end arterioles proximal to the occlusive plugs of parasitized red blood cells.

Cerebral malaria is accompanied by changes in the retina, the light-sensitive tissue at the back of the eye. These changes, known as malarial retinopathy, include white, opaque patches, whitening of the infected blood vessels, bleeding into the retina and swelling of the optic nerve. The first two of these signs are unique to severe malaria, and not seen in any other disease. So these changes-malaria retinopathy-can be elicited by examination of the fundus (by ophalmoscope) of patient presented with cerebral malaria. Out of 29 patients with cerebral malaria 14 were children and 15 were adult. In our studied group malarial retinopathy changes were detected only among those with cerebral malaria (7 children and 3 Adult).

V. Conclusion

The eye can provide a very reliable way of diagnosing cerebral malaria by looking at the changes in the retina (malarial retinopathy). Our research demonstrates that the detection of malarial retinopathy is a much needed diagnostic tool in cerebral malaria.

References Références Referencias