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CEREBRAL ATROPHY WITH NEUROPATHY AND DYSPHONIA RESPONDING TO ARTESUNATES: A CASEREPORT

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ABSTRACT

Malaria imposes a great socioeconomic burden on humanity and accounts for 85% of global infectious disease burden.[1] India alone contributes 76% of total cases of Southeast Asia.[1] DIC have been reported to involve in pathogenesis of malaria and can cause multiorgan dysfunction. Neurological deficit with DIC in malaria is rare but a fatal complication and require early diagnosis and treatment. Here we report a case of malaria with mild diffuse cerebral atrophy with small leukoariotic changes.

KEYWORDS: Malaria, Complication, Cerebral atrophy, Leukoariotic Changes.

INTRODUCTION

Since ancient time, it has been known that characteristic recurrent cycle of fever and chills is a major and most obvious symptom of malaria. But now from many years, asymptomatic malarial infections have been recognized i.e with no obvious symptoms. It results from partial immunity, which limits but does not completely eliminate the infection.[2] Such chronic infections tend to be of substantially lower density than acutely symptomatic infections; indeed, they can besubmicroscopic i.e., not detected by conventional diagnostic methods like peripheral blood film (Gold standard method) or by rapid diagnostic test (RDT).[3] It has been reported that bothmicroscopy and RDT will miss 30-50% of infections because of low parasites density (<10 parasites/µL).[4] As in patients with no previous history of malarial exposure poor immunity may result in severe disease even with low parasitic index (< 2%), these diagnostic methods can neglectthe malaria and underestimate the prevalence and severity of malarial infection.[5]

Perhaps there are many reports available to support the notion that elevated levels of D-dimer are present in mild or severe malaria as coagulation abnormalities and DIC are frequent in malaria.[6]

CASE REPORT

A 43-year-old female patient presented with speech difficulty, coughing, breathlessness and left limb weakness since July, 2019 and were getting worse since then. Patient gave a history of recurrent episodes of headache since 2016. MRI brain was done in October, 2019, suggested no significant brain parenchymal abnormality. MRI brain repeated on December, 2019, revealed mild diffuse cerebral atrophy with small leukoariotic changes. Nerve conduction study of both upper and lower limbs was done in May, 2020 and showed early neuropathy in B/L lower limbs with loss of H reflex. Repeat study of the same on July, 2020 showed decremental response in B/L deltoid and trapezius muscle to repeated stimulation. EMG study and CT abdomen and chest werefound to be normal.

Patient came to us on 11.04.2021. Platelets were decreased (101.0 thous/mm^3), Fibrin degradation product (FDP) and D-dimer were raised (5-20 µg/mL, 0.62 mg/L respectively). Anti dsDNA Ab and Rheumatoid factor were negative. RDT and peripheral blood smear for malaria also came negative. Thyroid profile, lipid profile, LFT, KFT and serum glucose levels were normal.

With the evident presence of DIC and after ruling out various other causes of neuropathy patient was empirically treated with injectable Artesunates. Patient showed fast recovery. Coughing and breathlessness disappeared within a week. Speech became almost normal within 2 months. Weakness of left limb is still improving. Initially patient was unable to move without helpin home surroundings, today she is attending her office independently.

DISCUSSION

Malaria is a complex syndrome and involves processes of coagulation, inflammation and pRBC sequestration. These processes interact to cause coagulopathy, DIC and microcirculatory dysfunction. *P.falciparum* causes knob like protrusion on infected RBC membrane which mediates adhesion to vascular endothelium. Rupture of schizont stage parasite causes exposure of phosphatidylinositol anchor and induces different inflammatory mediators which in turn activatesendothelial adhesion molecules enhancing cytoadherence of parasitized cells. Tissue factor expression by the endothelium and the amplification of the

coagulation cascade by pRBCs and platelets result in coagulation–inflammation cycle, particularly at sequestration sites that contributes to organ dysfunction in malaria.[6]

DIC has been observed in 30% of non-immune patients with severe complicated falciparum malaria. Whereas, in the autopsy cases, incidence observed was 55%.[7] Though, till date it has been documented by various international studies in endemic areas that in countries where either chloroquine prophylaxis was administered at mass level to children or where malaria has been eliminated cognitive function has been improved.[8-11]

This particular case showed unexpected age-related changes appearing early at 41 year of age confirmed by MRI brain with cerebral atrophy with leukoariotic changes and patient showed recovery with artesunates (antimalarials), suggestive that much of the ischemic neurological disease bulk especially of microvessels could be attributed to malarial process in endemic area. This needs further clinical and scientific evaluation so that much of the treatable microvascular ischemia in malarial endemic areas will find a simple medical treatment. This is to be further underlined that just negativity of RDT and Peripheral smear does not simply rule out the malarial presence.

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