Plasmodium Vivax Cerebral Malaria - A Rare Cause of Multi Organ Dysfunction

Abstract

Cerebral malaria is the most severe complication of plasmodium falciparum infection. It is a clinical syndrome characterized by unarousable coma at least one hour after termination of seizures and asexual forms of the parasite in peripheral blood smear with no other explainable causes of coma. Usually cerebral malaria is caused by P. falciparum, but rarely it is seen as a complication of plasmodium vivax infection. We report a case of cerebral malaria caused by Plasmodium vivax complicated by seizures and multiorgan dysfunction. It was successfully treated with parenteral artesunate, mechanical ventilation and other supportive measures without any sequelae.

Keywords: Cerebral malaria; Plasmodium vivax; Multiorgan dysfunction

Case Report

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Abbreviations: P.vivax: Plasmodium Vivax; PRBC: Packed Red Cell Concentrate; ALP: Alkaline Phosphatise; SGOT: Serum Glutamateoxaloacetate Transferase; SGPT: Serum Glutamate Pyruvate Transferase; EF: Ejection Fraction; ICP: Intracranial Pressure; LDH: Lactate Dehydrogenase

Introduction

Plasmodium falciparum infection is most commonly associated with severe malaria although in the past few years many cases of severe malaria are being reported due to infection with plasmodium vivax [1]. Classically malaria caused by P. vivax is considered as "benign tertian malariae" and usually follows a benign course with few complications. However recent studies and reports have revealed the dangerous potential of P.vivax to cause severe malaria including cerebral malaria, renal dysfunction, respiratory distress and bleeding abnormalities [2]. We report a case of severe malaria due to P. vivax infection that presented to us with features of cerebral malaria with multi organ dysfunction.

Case Report

A 57 year old male patient businessman by profession presented to our hospital with complaints of fever since five days. Fever was intermittent in nature and was associated with chills and rigors. Fever was associated with abdominal pain, generalized weakness and yellowish discoloration of sclera since three days. On the day of presentation he had an episode of vomiting and was found in a state of altered sensorium since morning. He gave no history of cough, breathlessness, chest pain, burning micturition, seizures or loss of consciousness. He gave a past history of two episodes of fever which he developed while he was visiting Tanzania, Africa where he was diagnosed with malaria and the fever subsided on treatment. Drug details were not known. The last episode of fever was six months back. He was apparently normal following that episode until one week ago. He was recently detected with diabetes mellitus and was on oral hypoglycemic therapy. He had no other significant past medical or surgical history and gave no history of drug or other allergies or any previous blood transfusions.

On presentation to our hospital, he was found to be conscious but disoriented. He was febrile with a temperature of 39^0C rising to 40^0C in between and was associated with chills and rigors. There was no evening rise of temperature. His pulse rate was 107 per minute, regular and all peripheral pulses were equally felt. His blood pressure recorded was 120/70 mmHg. Patient was icteric though rest of his general examination was unremarkable. His chest was clear to auscultation, heart sounds were normal with no audible murmurs. His abdomen was soft with no palpable organomegaly or ascites. He was disoriented though there were no signs of focal neurological deficit or signs of meningeal irritation and pupils were bilaterally equal and reactive to light.

His initial lab investigations showed a hemoglobin of 9.1 g/dl, total count 8,200cells/cumm with predominant neutrophilia. Thrombocytopenia was present with a platelet count of 60,000 cells/cumm. Renal function tests were deranged with a blood urea of 190 mg/dl, serum creatinine 6.2 mg/dl. Liver function tests showed unconjugated hyperbilirubinemia (total bilirubin 7.4 mg/dl, direct bilirubin 3.6 mg/dl, albumin 3.2 g/dl, ALP 262 IU/L, SGOT 31 U/L, SGPT 47 U/L). Serum LDH was elevated with values of 644 U/L, serum amylase was 30 U/L and serum lipase was 11 U/L. His initial blood sugars, serum electrolytes and coagulation profile were within normal limits. Tests for Dengue and Leptospira were found to be negative. Peripheral smear showed normocytic, normochromic anemia, thrombocytopenia and positive for trophozoites of plasmodium vivax malarial parasite. 2DEcho showed moderate LV systolic dysfunction with global hypokinesia and an EF 40% suggestive of myocarditis. Ultrasound abdomen and CT Brain were within normal limits.

He was admitted to the intensive care unit for the problems of cerebral dysfunction, acute kidney injury, un conjugated hyperbilirubinemia, thrombocytopenia and peripheral smear positive for plasmodium vivax. He was started on iv artesunate 120 mg once daily dosing, oral chloroquine 600mg on the first 2 days and 300 mg on the third day and other supportive

Future Prospects

A poor understanding of the pathogenesis is a hindrance to research in cerebral malaria. Functional MRI to describe neural activity in coma and proton MR spectroscopy to measure levels of substrate and metabolites could be seen as future prospects.

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