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Vivax malaria: a rare cause of thalamic bleed

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ABSTRACT

Most common cause of thalamic bleed is hypertension; other causes are arteriovenous malformation, aneurysm, bleeding diathesis, drugs, amyloid angiopathy, tumor etc. We present a case of *Plasmodium vivax (P. vivax)* malaria with unusual site of bleeding *i.e.* left thalamus of brain. To the best of our knowledge, this is the first reported case of thalamic bleed caused by vivax malaria in absence of severe thrombocytopenia/disseminated intravascular coagulation (DIC).

1. Introduction

The complications caused by *Plasmodium falciparum (P. falciparum)* malaria have also been recently reported to be associated with *Plasmodium vivax (P. vivax)*. Of patients with severe malaria, less than 5% have significant bleeding with evidence of disseminated intravascular coagulation. The neurological complications are cerebral malaria, intracranial hemorrhage, cerebral arterial occlusion, transient extrapyramidal and neuropsychiatric manifestations. These complications occur more frequently in children when compared to adults. Thalamic bleed is an unusual complication of severe vivax malaria, as in the present case.

2. Case report

A young adolescent male presented to us with complaints of fever, headache and bodyache of five days duration, vomiting and loose motion for one day and altered sensorium for 2 hours. Fever was intermittent, documented (104 0F), associated with chills and rigor. The patient had 2 episodes of vomiting and 4 episodes of watery loose motion. There was no history of rash, joint pain, cough, breathlessness, burning micturition, bleeding from any site. Past history, family history and personal history were not significant. On examination GCS = E2 V1 M3. He was febrile. Blood pressure was 80/60 mm Hg, PR 100/min, RR 30/min, SpO_2 = 98%. Rest general and systemic examinations were unremarkable. In view of low GCS and suspected aspiration of vomitus patient was intubated and put on mechanical ventilator, fluids replaced and inotropic support was also given.

Investigations showed Hb 11.7 g%, TLC 1500 cells/dL, neutrophil 60%, lymphocyte 40%, platelet count 80 000/dL. Peripheral smear for malarial parasite was negative. RBS 95 mg/dL, urea 66 mg/dL, creatinine 0.9 mg/dL, Na 136 meq/L, K 4.4 meq/L, bilirubin(mg/dL) (T/D/I) 2.7/1.7/1, AST,ALT,ALP(U/L) 74/264/542, ABG: (PH 7.414, PCO_2 25.8, PO_2 104.2, SO_2 97.2%, HCO_3 16.7, BE-8.1), urine routine examination showed no abnormality. Coagulation profile were within normal limit. Dengue serology (IgM) was negative. Chest X ray and ultrasonography of abdomen showed no abnormality. NCCT head showed hemorrhage in left thalamus (Figure 1). Rapid diagnostic test (Card test) was positive for *Plasmodium vivax* (Figure 2). Injectable artesunate and antibiotics were started along with other supportive measures. Patient regained consciousness after 24 hours, blood pressure also improved. So ventilator was weaned off and inotropes tapered and stopped next day. Subsequent laboratory investigations also showed improvement along with the improvement of patient's

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clinical condition. On sixth day of admission patient was stable and therefore discharged.

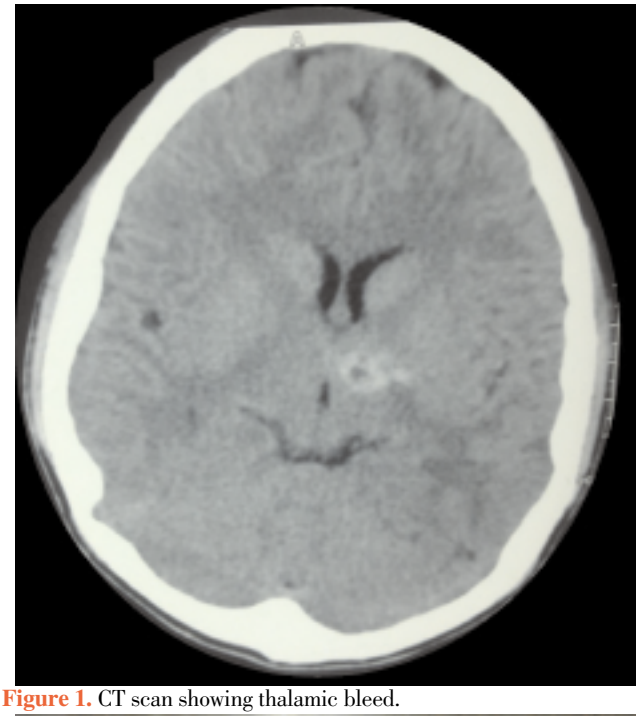

Figure 1. CT scan showing thalamic bleed.

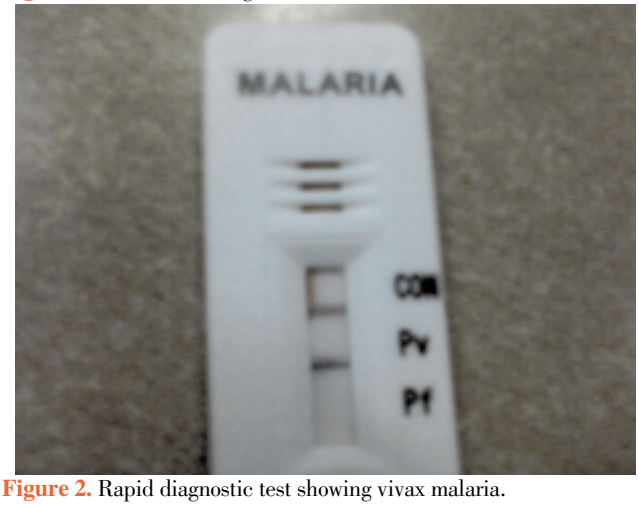

Figure 2. Rapid diagnostic test showing vivax malaria.

3. Discussion

Although there are promising new control and research initiatives, malaria remains today, as it has been for centuries, a heavy burden on tropical communities, a threat to nonendemic countries, and a danger to travelers. *P. vivax*, as was believed, is no longer a benign species and is causing presentations similar to P. falciparum.

P. vivax causing cerebral malaria, thrombocytopenia, disseminated intravascular coagulation, renal failure, circulatory collapse, severe anemia, hemoglobinuria, acute respiratory distress syndrome and jaundice has been reported in the past few years[1-6].

A unifying hypothesis for the genesis of cerebral malaria proposes that parasite antigens (released by replication in blood, surface molecules on parasitized erythrocytes, or merozoites) activate platelets that, in turn, contribute to the activation of the inflammatory response and increased levels of endothelial cell adhesion molecules (eCAMs). Increased levels of eCAMs result in further parasitized-erythrocyte sequestration and marked local inflammation that might disrupt the brain microvasculature, which can result in vascular leak and/or hemorrhage into the brain; similar processes can occur in other vascular beds, including the lung[7].

Abnormalities in platelet structure and function have been described as a consequence of malaria, and in rare instances platelets can be invaded by malarial parasites themselves[8].

The increased burden of disease, the emergence of resistance to antimalarial agents, and now the deployment of expensive artemisinin-based combination therapy into regions where malaria is highly endemic are increasing the need for rapid, accurate diagnosis of patients who may be infected with malaria[9]. Given the absence or poor execution of microscopy, especially in some areas where malaria is highly endemic, alternative diagnostic strategies are needed today. Where quality microscopy is available, then microscopy and rapid diagnostic test (RDT) can run in parallel, with RDTs providing a rapid or screening diagnosis and microscopy reserved for resolution of confusing cases, verification of negative results where the pretest probability of malaria seemed high, and overall quality control of the RDT program[10].

In addition, it is likely that RDTs will be cost-effective due to improved treatment and health outcomes for febrile disease not due to malaria along with cost savings associated with antimalarial drugs[11].

On June 2007, the U.S. Food and Drug Administration approved the first RDT for use by hospital and commercial laboratories in the United States.

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