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An Adult Case Of Plasmodium Vivax Cerebral Malaria

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Abstract

Introduction: Cerebral malaria is conventionally attributed to Plasmodium falciparum with clinical manifestations of coma and seizures. Nevertheless, Cerebral malaria has been infrequently reported in Plasmodium vivax malaria.

In this article, we report the case of a 70-year-old man with Cerebral malaria due to Plasmodium vivax infection who was admitted to our Medical Intensive Care Unit complicated by altered sensorium and seizures, suggestive of Encephalitis. He was diagnosed with the aid of microscopy and rapid diagnostic test for malarial parasites and treated with Intravenous Artesunate.

Plasmodium vivax infection is emerging as a life-threatening disease that's seldom seen in the southern districts of Maharashtra. It has been barely researched despite the high prevalence and its debilitating consequences. In endemic areas, the prospect of Cerebral malaria should be considered even with Plasmodium vivax infection.

Conclusion: This case report hence renders clarity on the fact that solitary Plasmodium vivax infection can cause multiple organ dysfunction as seen in Plasmodium falciparum infection which may be fatal and can no longer be considered benign.

Keywords: Plasmodium Vivax, Cerebral Malaria, Encephalitis, Seizures

Introduction

The World Malaria Report 2021 states that there were 241 million cases of malaria in 2020 compared to 227 million cases in 2019 with an increase of 69,000 deaths over the previous year [1].

Plasmodium vivax infection is topographically pervasive. Countries like Ethiopia, India, Indonesia, and Pakistan account for more than 80% of estimated cases of Plasmodium vivax. India alone is liable for 83% of Southeast Asia's malaria burden [1]. Plasmodium vivax infection is emanating as a public health problem in India with high prevalence in various regions of the country. Negligence and misdiagnosis of Plasmodium vivax infections with Plasmodium falciparum and other tropical infections have led to poor management and treatment resulting in an increased disease burden. A benign course with multiple relapses was the characteristic feature of Plasmodium vivax malaria. The typical complications seen in Plasmodium falciparum malaria are seldom found in Plasmodium vivax mono infections. Nonetheless, lately, the trend in the clinical manifestations of Plasmodium vivax malaria has been dynamic [2]. Severe complicated cases of Plasmodium vivax have been reported in many isolated studies conducted in India [3]. As of now, only 45 cases of Plasmodium vivax with CNS manifestation have been identified in the Scientific

literature since 1920 with predominance in the paediatric age group [4,5].

Case Report

A 70-year-old male, hailing from Kavthemahankal, Sangli presented to the Bharati Hospital ER in an altered mental state following an episode of Generalized Tonic-Clonic Seizures with Loss of Consciousness lasting an hour with a history of fever for 4 days associated with chills and rigours, evening rise in temperature, night sweats, malaise, chest discomfort, and dry cough. He had been diagnosed with HTN and IHD 5 years back on regular treatment with oral Telmisartan 40mg and Atorvastatin(20mg)-Aspirin(75mg) combination. History was elicited from his Spouse, a reliable informant.

On admission, the patient was febrile (101^{o}F) and drowsy with response to verbal commands and spontaneous eye opening and movements in all 4 limbs with 11/15 on GCS (E4V3M4). on clinical examination, the patient had Grade II Splenomegaly. He was admitted to the Medical Intensive Care Unit for further evaluation and management under the suspicion of Encephalitis. Routine haematological tests showed thrombocytopenia (35,000/cu.mm.), leucopenia (4500/cu.mm.), and anaemia (11.3g/dl). The biochemical tests showed deranged renal and hepatic parameters with Sr. Creatinine (1.8mg/dl), Urea (59mg/dl) and SGOT 68(IU/L), SGPT 75(IU/L), Total Bilirubin 3.5(mg/dl), Direct 2.3(mg/dl) and Indirect 1.20(mg/dl). ESR was 14 mm/hr. The USG Abdomen & Pelvis read Splenomegaly(13.9cm), Grade I B/L Raised renal cortical echogenicity, Cystitis, and Prostatomegaly. CT Scan of the Brain and EEG were unremarkable. CSF analysis revealed decreased Glucose level (40mg/dl) with a normal Protein level (23mg/dl) and 3 leukocytes (100% Lymphocytes).

Rapid Malarial Test for Plasmodium Vivax was Positive and was confirmed on a Peripheral Smear for Malarial parasites which revealed occasional schizonts and few ring forms of Plasmodium Vivax while that for Plasmodium Falciparum was negative. The rest of the Fever Panel consisting of Rapid Dengue Test, Widal and Tropical Fever Panel (Leptospira, Weil Felix, and Brucella) were negative.

2D ECHO was done I/V/O history of IHD, HTN, and ECG changes which revealed Global Hypokinesia of LV with EF 35-40%.

A diagnosis of Plasmodium Vivax Cerebral Malaria was made. The patient was started on Intravenous Fluids, Inj. Ceftriaxone 1g BD for 14 days and Inj. Artesunate 120mg OD for 3 days f/b T. Primaquine 15mg OD for 14 days along with Inj. Levetiracetam 1g stat f/b by 500mg BD with supportive treatment for IHD and HTN. Repeat Peripheral smear for Malarial Parasite was negative. The patient improved over the next 48 hours and was discharged on the the15th day.

Discussion

Plasmodium vivax is an emerging cause of cerebral malaria in adults in India and should be considered in the differential diagnosis of cerebral malaria for the proper management of patients.

Organ dysfunction indicative of Plasmodium falciparum malaria is seldom seen in Plasmodium vivax infections. Any patient infected with Plasmodium vivax who exhibits severe malaria is conjectured to be suffering from mixed infection [5,6]. Nonetheless, it is apparent in the present report, that Plasmodium vivax infection can also present as cerebral malaria.

Kochar, et al. demonstrated that Plasmodium vivax can cause both sequestrations related and non-sequestration-related complications of severe malaria which are usually attributed to Plasmodium falciparum infections [2]. A study by Sachdev and Mohan on the clinical-laboratory profile of six patients with Plasmodium vivax cerebral malaria revealed clinical manifestations of acute febrile encephalopathy, convulsions, and coma while focal neurological signs were observed in one patient [7]. A similar study conducted by Ozen, et al. described a case of Plasmodium vivax cerebral malaria that manifested as status epilepticus [4].

Conclusion

The reason for an increase in the virulence of Plasmodium vivax over the years is a conundrum. The probable pathophysiology could be alterations in the parasite's genomic make-up, drug resistance, and impaired host response.

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Owing to the enormous disease burden of Plasmodium Vivax in South East Asian countries, adequate research and implementation of molecular diagnosis aiding the traditional blood film microscopy and rapid antigen test for malarial parasites should be considered for prompt diagnosis and management. This case report hence renders clarity on the fact that solitary Plasmodium vivax infection can cause multiple organ dysfunction as seen in Plasmodium falciparum infection which may be fatal and can no longer be considered benign. This propels us to provide greater attention to the control of Plasmodium vivax in South East Asian countries.

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