Case Report

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Severe Complicated Plasmodium Vivax Malaria Presenting as Shock in a Child

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Abstract

Malaria is a vector-borne disease caused by a Plasmodium parasite having five species (falciparum, vivax, ovale, malariae and knowlesi) transmitted to humans through the bite of an infected Anopheles mosquito. South East Asia is the second most affected region in the world, with India carrying the highest burden of the disease. Plasmodium vivax and Plasmodium falciparum are the most common species found in India. Severe complications of malaria have been more commonly seen in Plasmodium falciparum infections, and those caused by Plasmodium vivax have been considered benign. But in recent times, the severity caused by Plasmodium vivax has been emerging in India and is in equal ratio with Plasmodium falciparum to the malaria incidence. Here we report a case of a 5-year-old boy who presented with high-grade fever with chills, burning micturition, weak peripheral pulses, reduced oral acceptance, hypotension and pallor. The diagnosis was established by a thick and thin film of peripheral blood smear examination under oil immersion with Giemsa stain and Malarial Antigen Test. The child was started on anti-malarial therapy and fluid therapy was given to treat shock.

Keywords: Malaria, Falciparum, Vivax, Malarial Antigen test, anti-malarial, shock.

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Introduction

India, Indonesia, Ethiopia, and Pakistan are the four countries that account for more than 80% of estimated P. vivax cases out of which India alone contributes for about 80% of the SEA burden. Indeed, P. vivax predominates in countries in the pre-elimination and elimination phases.[1]

P. vivax malaria was mostly found to have a benign course with multiple relapses. Complications that are seen in P. falciparum malaria are not usually found in P. vivax mono infections. However, in the past few years, this trend in the clinical manifestations of P. vivax malaria has been changing.[2] Several studies have reported severe complicated cases of P. vivax malaria from India.[3]

Current research about the disease is insufficient probably because it is considered benign compared to more complicated P. falciparum malaria. In recent times, with the implementation of molecular diagnosis, it has become evident that P. vivax mono-infection can also lead to multiple organ dysfunction and severe life-threatening disease which was more commonly seen in P. falciparum infection.[4,5]

According to the World Health Organization (WHO), liver dysfunction is an unusual occurrence in malaria.[6] Malarial hepatitis is uncommon in P. vivax infections.[7,8] Jaundice caused by malaria infection has 0–9% frequency.[6]

Plasmodium vivax can result in severe disease and can no longer be considered a benign condition. In 2017 there were approximately 219 million cases of malaria responsible for about 435000 deaths, the majority on the African continent (WHO World Malaria Report 2018).

Parasitaemia in vivax infections is generally low and severe disease is not characterized by high parasite load.

Case Report

A 5-year-old, fully immunised, developmentally normal boy presented with high-grade fever associated with chills and sweating for 7 days, burning micturition and reduced oral acceptance since 1 day. The child also had a history of headache, retro-orbital pain, myalgia, and weakness, for which the child was given some symptomatic treatment by a local doctor. When the child was brought to our hospital,

he was sick-looking and pale. On examination tachycardia and tachypnea were present with HR- 140/min and RR-42/min, maintaining saturation 100% on room air, peripheral pulses were averagely felt, Capillary refill time was just 3 seconds, blood pressure 88/40 mmhg with no significant lymphadenopathy and icterus.

On abdominal examination, hepatosplenomegaly with the liver size of 4.5 cm and a span of 8 cm, soft in consistency, spleen 7.5 cm below the costal margin. Rest systems were normal. Blood investigations were sent in which CBC showed pancytopenia with Hb 6.7mg/dl, TLC 2.5cells/mm3 with lymphocytic predominance (60%), platelets 80,000, cells/mm3, CRP was raised 134, Malarial antigen test positive for plasmodium vivax, malarial index 5/1000 RBC's. G6PD was not deficient, urine routine, renal function test, liver function test, serum electrolytes and coagulation profile were normal.

The child was given two boluses of 0.9% NS @ 10cc/kg, Blood pressure and peripheral pulses were reassessed and started on Inj Falcigo and Ceftriaxone(7days), IV fluids 0.9%DNS as oral acceptance was poor and symptomatic treatment was given. Urine output, Blood pressure, GCS, other vitals (HR and RR) and saturation were closely monitored. On repeating CBC child's Hb and platelet dropped further with Hb 6.1mg/dl and platelet counts 74cells/mm3, persistent tachycardia decision to give one unit of PRBC transfusion was made.

Inj Falcigo 120 mg (artesunate) was given for 3 days and later was switched on to Tab Lumerax (Artemether and Lumefantrine) for another 2 days. His vitals improved over the next 48 hrs, spleen size reduced to 4 cms and liver size reduced to 2.5 cms below the left costal margin and span of 6cms, clinically and hemodynamically stable, accepting orally well. Hence was discharged and Tab Primaquine for 14 days was given to prevent relapse of Plasmodium Vivax.

Discussion

Malaria causes significant morbidity and mortality, the incidence rate of malaria is very high in INDIA. Plasmodium falciparum and Plasmodium vivax are the commonly found species in INDIA, though very reported cases of Plasmodium malariae.[9] Of all the species of Plasmodium causing infection in humans, P. vivax is the most widespread in the world. Central and South-East Asia, the Horn of Africa and Latin America are the most commonly reported places with P. vivax infection.[10] It causes significant morbidity and mortality among the people except in African populations who are mostly Duffy negative, which makes them less susceptible to malarial infection. But recent data suggest that for erythrocyte invasion, the parasite may use receptors other than Duffy (Duffy antigen/chemokine receptor).[11]

Research and analysis about the disease are insufficient probably because it is considered benign compared to the more severe presentation of P. falciparum malaria. The severe manifestations of P. vivax malaria are renal dysfunction, severe anemia, ARDS, multiple organ involvement, cerebral malaria and liver dysfunction.[12] The exact pathogenesis, parasite-host interactions, and reasons for multi-organ dysfunction due to P. vivax are unclear.[13]

Here we have reported the case of a vivax malaria in a 5-year-old male child without any prior confirmed malarial illness but had severe manifestations similar to falciparum such as extreme weakness, pancytopenia and shock.

There was no associated comorbidity or any evidence of mixed infection.

Only P. vivax species were identified by peripheral smear and were no subjective error.

Patients had no features of cerebral malaria or severe metabolic acidosis that required correction. With the low levels of hemoglobin and tachycardia, one unit of blood transfusion was needed. This case report points out that severe complications of malaria are currently often seen even in P. vivax infection as obtained in falciparum infection. The incidence of complicated vivax infection is rising.

Conclusion

Complicated vivax malarial infection may also be very easily encountered, but often under-diagnosed. Early recognition and prompt treatment can markedly reduce morbidity and mortality.

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