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Case Report

Case Report of P. Vivax: An Atypical Presentation in Pediatric Age

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Abstract

Worldwide Malarial infection remains a major cause for morbidity and mortality. Only 50-70% patients showed classic presentation of malaria with paroxysms of fever and rest unusual presentation might be because of impending resistance to antimalarial drugs and judicious use of antimalarial drugs and establishment of immunity.

Here, we report peadiatric age group case of Vivax malaria with unusual presentation with history of generalized weakness with severe anemia and going for renal failure. He presented to emergency ward with failure to thrive and treatment started for nephrotic syndrome apparently no improvement in the patient. During laboratory investigation, found to be having malarial parasites in peripheral blood smear, for which immediate therapy was started. After first dose of antimalarial drug patient started showing some signs of recovery. Thus we would like to highlight atypical presentation of malaria, where early recognizing with treatment can save lives. Clinicians must be aware of unusual presentations and constant changes in epidemiology; difficulties in early detecting of severe manifestations of Plasmodium vivax malaria, emphasis must be made to lower the disease burden.

Key words: P.vivax, anemia, malaria.

Introduction

Malaria is a protozoan disease caused by the bite of a female anopheline mosquito which is infected by the Plasmodium. Malaria is the world's third most infectious disease after tuberculosis and HIV/AIDS.[1] Currently, 6 species of Plasmodium (P. falciparum, P. vivax, two sympatric species of P. ovale, P. malariae, and P. knowlesi) can cause malaria.[2] Of these killer species, P. vivax was known to cause malaria for 14.8 million cases in 2016, with most spot cases (76%) occurring in the Southeast Asia Region (SEAR) where India share the same disease burden.[3]

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Previously only P. Falciparum was considered to cause complicated malaria exclusively. However, over the past few previous years, reports from various studies from different parts of world have shown that P.vivax has emerged to cause equivalent disease complication. In various cases, P.vivax malaria have been reported with severe complications, like anaemia, cerebral malaria, hepatic dysfunction, renal failure, ARDS[4-7] and retinal haemorrhage.[8] But P.vivax cases have not much presented with neurological complications as seen with P. falciparum malaria like cerebral infarction.[9-10]

This pediatric age case report highlights the unusual presentations of P. vivax malaria which should be aware of, while making clinical differential diagnosis in endemic zones as well as hypoendemic zones of malaria in countries like India.

Case report

A 5-yr-old male child, residing in the rural area of Kolar, Karnataka, India presented to the emergency department of the Sri Devaraj Urs Medical College with complaints of generalized weakness and lethargy since one week and pain abdomen from 2 days. A detailed history was elicited which suggested no significant past illnesses or any significant illnesses in the family.

On clinical examination on Day 1, the patient had a pulse rate of 60/min and blood pressure of 120/80 mmHg. The patient was conscious and well oriented. Examinations of cranial nerves, the motor system, sensory system as well as the cardiovascular, respiratory and abdominal examination were found to be normal. Routine blood investigations including a complete blood count, random blood sugar, renal function was performed. (Table 1)

On Day 3, the patient had a pulse 90/min and blood pressure 130/90mmHg. The patient had developed ascites with increased abdominal circumference and dull note on percussion. Repeat laboratory investigation was sent. Since haemoglobin was very low, peripheral smear was performed. The PBF showed RBCs with severe anisopoikilocytosis and microcytic hyphochromic anemia with 57/100 WBC nucleated RBCs. WBCs were increased in number with neutrophilic distribution and shift to left till band forms. Platelets were reduced in number and had few giant platelets. PBF revealed Trophozoites and Schizont forms of Plasmodium vivax which was later confirmed by Jaswant Singh-Bhattachariji (JSB) special stain (Figure-1, 2 & Figure –3). There was derangement in few parameters shown in Table 1.

Patient was started with antimalaral drugs from Day 3, oral Quinine 3 × 400mg/day for 7 days and Primaquine therapy 1 × 15mg/day for 14 days. On day 5, patient's symptoms started improving. On day 10, patient's attendees decided to get leave from the hospital before total recovery and to continue to finish the course at house.

Table 1: Laboratory parameters of the patient.

Laboratory investigation	Normal reference range	Day 1	Day 3	Day 5
Hemoglobin (g/dl)	**11-14g%**	**2.1**	**4**	**6.1**
Red Blood Cells (mil/mm^3)	**4-5.2million /mm^3**	**0.76**	**1.37**	**2.78**
Total leucocyte count (Thousands/mm^3)	**5-15 Thousand /mm^3**	28100	23700	13600
Platelet count (Thousands/mm^3)	**150-450 Thousand/mm^3**	36	97	128
Bilirubin—Total (mg/dl)	**0.2-1.3mg/dL**	0.4	0.51	0.43
LDHi (U/L)	**120-246U/L**	1548	1430	922
Serum Creatinine (mg/dl)	**0.6-1.3mg/dL**	0.3	0.4	0.6
Total protein (g/dl)	**6.4-8.3g/dL**	4.2	3.9	5.1
Blood urea (mg/dl)	**12-40mg/dL**	114	109	57
Urine routine	**-**	Nil	Nil	Nil
Random blood sugar (g/dl)	**140mg/dL**	86	99	95

Figure 1: Peripheral smear showing ring forms of Plasmodium vivax. Leishman stain, 40x

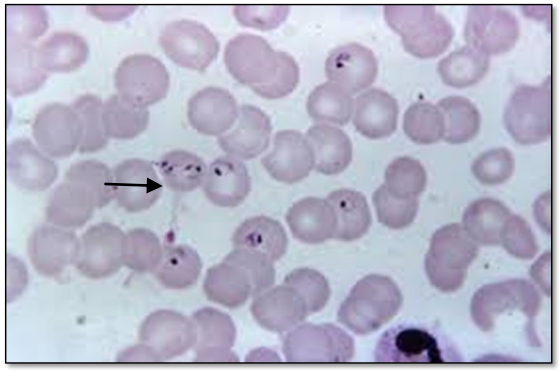


Figure 2: Peripheral smear showing trophozoites forms of Plasmodium vivax. Leishman stain, 40x

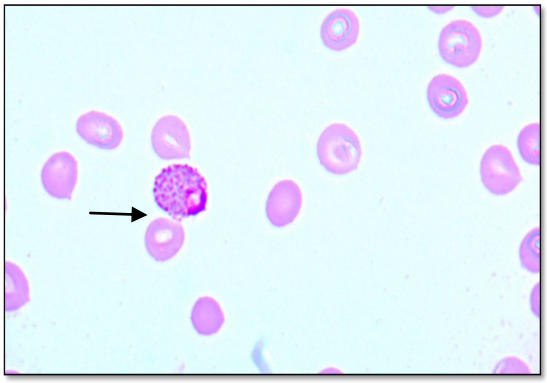
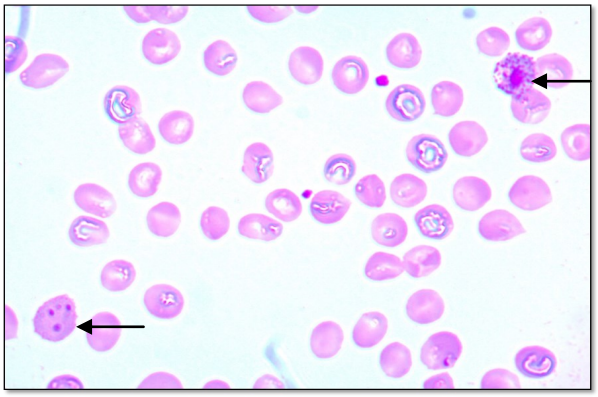


Figure 3: Peripheral smear showing both ring forms and trophozoites forms of Plasmodium vivax. Leishman stain, 40x



Discussion

Malaria remains to be mysterious leading cause for morbidity and mortality in humans, inspite of intensive efforts from the past decades to understand the disease process and control disease burden. An approximate estimate of 218–269 million people suffer from malaria each year and 12% deaths annually according to WHO 2020 survey.[1,2] The intraerythrocytic protozoa of the genus Plasmodium, which is known to cause malaria in humans being infected by one or more of the Plasmodium species like Plasmodium falciparum, Plasmodium vivax, Plasmodium ovale, Plasmodium malariae, and Plasmodium knowlesi. Malaria is mainly transmitted by the bite of an infected Anopheles female mosquito or through exposure to infected blood products and mighty be by congenital transmission.[2] in rare scenarios. Majority of severe and fatal malaria cases reported in various studies were mainly due to P. falciparum infestration.[3] P. vivax malaria usually the term as 'benign tertian malaria', since it is an uncomplicated disease with a uneventful course and occasionally fatal.[4] This clinical paradigm has been challenged recently and has been reported by few studies about its severity and even deaths due to P. vivax monoinfections.[4–10] Tatura et al showed that P. vivax infection in neonates presented with severe sepsis in endemic areas which was diagnosed by incidental findings of peripheral smear.[11]Severe anemia is the most common feature of *P. falciparum* malaria worldwide; however, in *P. vivax*-endemic areas, especially younger age group children are more prone for severe anemia with *P. vivax* infection.[2] The low parasite biomass burden of *P. vivax* indicates that due to rapid destruction of infected RBCs causing severe anemia and might be other pathophysiology plays adds on. Malariotherapy studies have disclosed that for every infected RBC destroyed during *vivax* infection even non infected RBCs are also, when removed from the circulation compared to the loss of 8 RBCs for every infected erythrocyte in *falciparum* malaria. Might be the cytokine-related dyserythropoiesis contributes to anemia in *P.vivax* infestation.[14]

P.vivax malaria has been considered to have a benign course and as established for its multiple relapses, but the typical complications seen with *falciparum* malaria is not documented with *P.vivax* monoinfection. In this present study it was shown that *Plasmodium vivax* malaria, endemic prone area can also cause impact complications such as severe anaemia, symptomatic or asymptomatic thrombocytopenia, melena, acute renal disturbance, hepatic dysfunction, nephrotic syndrome and jaundice. Destruction of RBCs was very intense leading to severe anemia, derangement in biochemistry values leading the diagnosis towards kidney diseases. For which patient symptoms did not improve. Incidental identification of parasite forms in peripheral smear led for administration stat antimalarial drug. Patient symptomatically showed signs of improvement. Thus rapid diagnostic tests can result in prompt timely diagnosis, microscopic examination is a diagnostic tool and if possible additional molecular confirmation is necessary to safely diagnose mixed or rare species of infections. Intensive management and effective supportive measures following with standard protocols of management are required to treat such rare cases.[7,8] *P.vivax* pathogenesis needs to be elicited in detail for better understanding of the disease pathophysiology and progression of the severity of the disease outcome.

Conclusion

Clinicians must be aware of emerging of unusual and prompt manifestation of *P. vivax* malarial infections. Rapid timely diagnosis and management can minimizes disease associated morbidity and mortality. Due to constant changes in epidemiology patterns, early recognition of severe manifestations of *P. vivax* malaria, and knowledge about emerging drug resistance, emphasis must be on strict preventive measures to reduce the burden of this *P.vivax* malarial disease.

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