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Myocarditis associated with malarial fever: An unusual presentation

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ABSTRACT

Malaria is endemic in India and complicated malaria is usually caused by *Plasmodium* (P) *falciparum*, but *Plasmodium vivax* can also cause life-threatening conditions in some cases. We report one such case of 47 years male presenting with myocarditis associated with *P. vivax* malaria.

Key words: *Plasmodium vivax*; Malaria; Malarial myocarditis; ARDS

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INTRODUCTION

Malaria is a protozoan disease caused by *Plasmodium* species and transmitted by bite of infected female anopheles mosquito. It is endemic in around hundred countries over the world.[1] Usually complications occur in *Plasmodium falciparum* infection but they are uncommon with *P. vivax* infection and myocarditis is one of the rare complications.[2] This case report is about a 47-year-old male who presented with *P. vivax* malaria associated myocarditis.

CASE REPORT

A 47-year-old male presented with complains of high-grade intermittent fever, associated with chills and rigors for 5 days. He also complained of shortness of breath, headache, and abdominal discomfort for1 day. There was no history suggestive of any chronic illness, that is, diabetes mellitus, hypertension, cardiac illness, and respiratory problems. On physical examination, he was anxious, tachypnoeic, dyspnoeic with NYHA Class IV, febrile(101°F), pulse rate of 126/min, Spo_2 was 86% at room air, his blood pressure was 87/48 mmHg, bilateral crepts in chest on auscultation, tachycardia, S3 was audible, tenderness in the right hypochondrium, and no organomegaly on palpation. Rest of the systemic examination was normal. On investigation hemoglobin was 14.9g/dl, total leukocyte count 13700/ul, and platelets 80,000/ul. C-reactive protein was 211mg/l. PaO_2/FiO_2 ratio was 170. Liver function test revealed SGOT-70U/L and SGPT-103U/L. Malaria antigen test was positive for *P. vivax* antigen. Peripheral blood film examination showed schizont stage of *P. vivax* (Figure 1). Dengue NS1, Typhidot, and Scrub Typhus were negative. Blood test for leptospirosis was found negative. Kidney function test was within normal limits. Respiratory biofire panel was done

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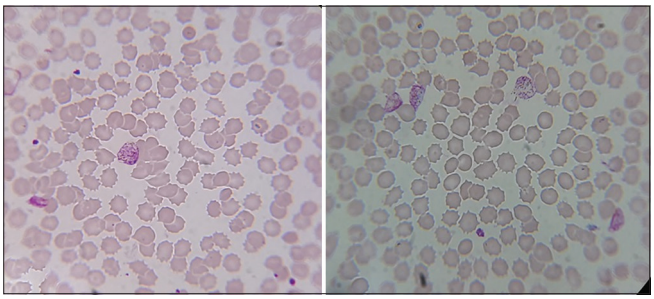

Figure 1: *Plasmodium vivax* in Giemsa-stained thin blood smear shizont stage present in peripheral blood

which came out negative, his sputum was sent for gram staining and culture sensitivity which were also found to be negative. RTPCR for COVID was negative. X-ray chest showed bilateral parahilar shadows. Echocardiography was suggestive of severe left ventricular (LV) systolic dysfunction (LVEF 25%), severe global LV hypokinesia, moderate mitral regurgitation, mild tricuspid regurgitation, and no pulmonary arterial hypertension, with dilated LV and inferior vena cava (diameter 24mm). Troponin levels were 22040ng/L. A diagnosis of complicated vivax malaria associated myocarditis with ARDS was made.

Patient was admitted in intensive care unit and managed with non-invasive ventilation (NIV) (FiO_2 0.5, PEEP-8cmH_2O, pressure support-8cmH_2O), injection artesunate 120mg BD for 1 day then once daily for 2 days, Inj. Furosemide 20mg twice daily for 5 days, and other supportive measures. Patient responded well to treatment and his fever subsided, NIV support was weaned off (FiO_2 30%, PEEP-6cmH_2O, PS-8cmH_2O) and patient was maintaining oxygen saturation on nasal pronge with O2 @ 2 L/min and within next 48 h, he was on room air. His blood investigations also showed improvement (Table 1). After 7 days, repeat ECHO was done which showed improved LVEF of 55% and patient was discharged in stable condition.

DISCUSSION

Many complications have been described in malaria such as cerebral malaria, liver dysfunction, ARDS, acute kidney injury, anemia, and metabolic acidosis, which are usually associated with *P. falciparum* infection. However, there are some case reports depicting these complications in *P. vivax* malaria patients.[2-5] However, only few cases of myocarditis due to vivax malaria have been reported.[6,7] Although an exact mechanism of cardiac involvement in malaria has not been described. Reports have suggested that capillary occlusion by malarial parasites and parasitized red blood cells could be the main factors causing myocardial damage.[8] A major contribution of tumor necrosis factor (TNF) has also been observed in the inflammatory cascade with stimulation and migration of leukocytes, macrophages, and further amplification of inflammatory response.[9] Andrade et al.,[10] reported a strong relationship between increased levels of C-reactive protein, TNF-alpha, interferon (IFN)-gamma, IFN-gamma/IL-10 ratio, and the disease severity of *P. vivax* malaria.

Table 1: Investigation profile of the patient

S no.	Investigations	Day 1	Day 4	Day 7
1	Hemoglobin (g/dl)	14.9	14.0	12.9
2	Total leucocyte count (cells/ul)	13700	12600	9600
3	Platelet count	80000	68000	1.5lacs
4	CRP (mg/dl)	211	120	36
5	SGOT (U/L) SGPT (U/L)	70 103	26 47	-
6	Malaria antigen test	Positive	-	Negative
7	Troponin (ng/L)	22040	-	230
8.	ECHO	LVEF 25%	-	LVEF 55%

Our case report signifies that *P. vivax* infection can cause life threatening complications. Hence, early detection and timely intervention can be lifesaving.

CONCLUSION

It is well known about p.falciparum infection tht it can manifest as various complications such as cerebral malaria, liver dysfunction, ARDS, acute kidney injury, anemia, myocarditis and metabolic acidosis. But, our case report is all about complication due to P.vivax, which is not common. So, we concluded from our case report that P.vivax can also manifest as life threatening complications like myocarditis and cannot be always considered as simple beningn infection.

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Authors' Contributions:
AK- First draft of case report, **PG**- Draft, proof reading, and editing of case report, **JP**- Concept and editing of case report, **GA**- Diagnosis and management of case, **KB**- Guidance for case management, and **EA**- Management of case.

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