

A Rare Presentation of a Common Disease: A Diagnostic Dilemma

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Malaria affects millions of people across the globe. The classical clinical features may be absent, but the rapid diagnosis helps in early treatment and thus avoids complications. We present a case of co-infection of Plasmodium vivax and Plasmodium falciparum malaria in a female patient presenting with fever and pain abdomen and incidental detection of the splenic infarct. The co-infection is uncommon and treatment should target both to avoid complications. Also, the exact pathogenesis is un known and though splenic infarct is uncommon and missed due to lack of symptoms, it should be followed up.

Keywords: Co-infection, Plasmodium vivax, Plasmodium falciparum, Splenic infarct

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Introduction

Malaria is one of the commonest parasitic infections causing a large number of deaths throughout the World. Humans are infected by 5 species, but infection with more than one species is uncommon in a single patient. The case discussed here is a female patient presenting with fever and pain abdomen due to malaria. Diagnosis revealed co-infection with vivax and falciparum malaria with incidental detection of splenic infarct which usually has a good prognosis.

Case Report

A 32 years old female, a housewife by occupation, residing in South 24 Parganas district presents with fever for the last 3 days with chill and rigour with pain abdomen for the last one day in the right hypochondrium with a history of nausea and vomiting for the last 2 days without any history of loose motions. The pain increased in severity particularly with the rise of temperature without any radiation or relation to food. There was no history of myalgia, arthralgia, skin rash or any bladder bowel complaints. LMP was 7 days ago and there was no history of bleeding from any site. There was no significant past history treatment history or travel history. On examination, the patient was alert, conscious and cooperative without any signs of meningeal irritation. Mild pallor was present without any icterus or lymphadenopathy. Temperature was 101^oF with tachycardia and normal blood pressure without any pedal edema.

Systemic examination revealed tender hepatomegaly and splenomegaly (non-tender) with right hypochondrial pain and tenderness without any ascites or dilated abdominal veins. Respiratory system examination revealed tachypnea with bi basal crepitations. Examination of other systems was unremarkable. A provisional diagnosis of enteric fever was done with differentials of malaria and scrub typhus as the patient resided in a Scrub typhus endemic area with a recent visit to a nearby jungle 7 days before the onset of fever.

The patient was treated with injection Ceftriaxone, injection Doxycycline, IV fluid, injection PPI, Ondansetron along with oral Paracetamol and drotaverine injection. The malaria slide showed ring forms, schizonts and gametocytes of both Plasmodium Vivax and Plasmodium Falciparum

(Figure1).MP Dual antigen was also positive for both vivax and falciparum. Hemoglobin was 10 gm/dl with normal TLC and platelet count. Peripheral blood smear revealed microcytic hypochromic anemia. Renal function tests were normal except mild hyponatremia and mild hypokalemia with normal amylase, lipase and LFT revealed no abnormality except mild hypoalbuminemia.NS1 antigen, Typhi Dot M, IgM Scrub Typhus antibody, IgM Leptospira antibody tests were negative with normal urine reports. The ferrokinetic study revealed mild iron deficiency anemia and Hemoglobin electrophoresis was unremarkable to rule out hemoglobinopathy. Chest x-ray, Echocardiography were normal with USG Whole Abdomen showing borderline splenomegaly(12.15 cm) with enlarged liver(15.17 cm) without any evidence of acalculous cholecystitis and CECT Whole Abdomen revealed multiple wedges-shaped non-enhancing areas in the spleen suggestive of splenic infarcts(Figure 2). Upper GI Endoscopy revealed gastric erosions. Injection Artesunate 120 mg iv stat APST was given followed by similar doses after 12 hours and 24 hours of the first dose followed by 120 mg for the following five days. As there was no evidence of hypoglycemia, IV fluid 5% Dextrose which was started after diagnosis was stopped after 3 days. Fever started to subside after 24 hours of starting Artesunate and the patient started feeling better. Abdominal pain started to resolve and drotaverine was stopped after day 2 of starting anti-malarial.PPI was given orally. As G6 PD was normal primaquine tablet 15 mg od was started. The patient was discharged 9 days after admission and was referred to a Gastroenterologist for a splenic infarct.

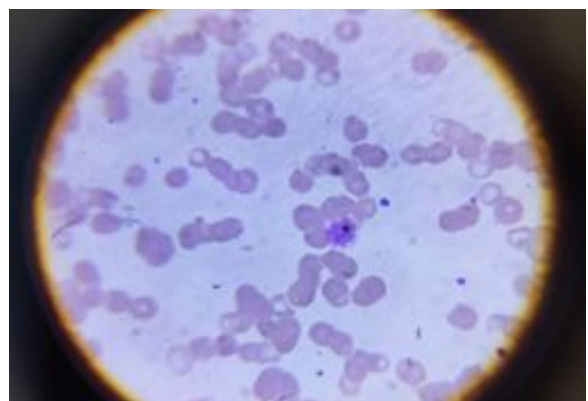


Figure 1: Ring forms, schizonts and gametocytes of Plasmodium vivax and Plasmodium falciparum.



Figure 2: CECT Whole Abdomen showing splenic infarct.

Discussion

Malaria is a parasitic infection caused by protozoa Plasmodium which is transmitted by the female Anopheles mosquito. The 5 species causing human infections are *P. vivax*, *P. falciparum*, *P. ovale*, *P. malariae* and *P. knowlesi* [1,2,3]. Among malaria patients, 5-7 % of cases are infected with more than one species and is also known in mosquito vectors [2]. After a blood meal by the mosquito, sporozoites enter liver cells within minutes and after a few weeks reach the bloodstream. The merozoites enter RBC and develop into trophozoites and schizonts over some days. RBCs rupture result in fever and release of merozoites which enter new RBCs and repeat the process and thus increase the parasite burden. Plasmodium falciparum causes cytoadherence and rosette formation and causes microvascular damage. This is the probable mechanism of splenic infarct in the case. Annually 300-500 million cases of malaria occur worldwide [4]. mostly occurring in rural tropics below 1000 m elevations. 1-3 million deaths occur per year, mostly children less than 5 years and most of the deaths are in rural areas of Sub Saharan Africa [4]. Thalassemia, sickle cell trait and G6 PD deficiency protects from death due to falciparum. Splenic complications are splenic infarction, spontaneous rupture of the spleen, hypersplenism, hyper-reactive splenomegaly syndrome, ectopic spleen, cyst and torsion [5]. Splenic infarction is rare in malaria but may be missed due to underreporting and under-diagnosis [6,7]. Spleen rupture was more frequent with *P. vivax* according to some studies [8]. As patients are asymptomatic,

Splenic infarction is usually not detected. Splenic infarction has a benign course in malaria without any complications and does not have any operative indications [9]. Splenic infarctions occur due to ischemia of the parenchyma from arterial or venous occlusion [9]. Clinical studies suggest that in vivax-falciparum co-infection, *P. vivax* may be protective with less severe disease and a lower level of parasites. Failure to detect co-infection at an early stage and treating for falciparum only, vivax may cause a persistent increase in parasite load and cause relapse.[10]

Conclusion

The importance of the case lies in the fact that dual malaria infections are uncommon and further studies are needed to determine their incidence. The pathogenesis should also be determined that whether both the infections occur together or one precedes the second infection. Lastly, splenic infarction which is rare should be followed up although the prognosis is good.

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