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Case Report

Rare case of Plasmodium Vivax causing malarial splenic infarction at LUH, Jamshoro

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Abstract

Malaria is a common parasitic infection in tropical countries. It has many serious complications like anemia, acute kidney injury, cerebral malaria, acute respiratory distress syndrome. Splenic infarction due to malaria is a rare complication. Up to 40 cases has been reported from 1960 to 2012. Plasmodium Vivax remains the common species. We report a case of a patient with Plasmodium Vivax infection who developed abdominal pain after 2 days of high-grade fever. Ultrasound abdomen revealed mild splenomegaly with multiple hypoechoic areas suggestive of splenic infarcts, which were further confirmed by abdominal CT scan. Patient responded to antimalarial and analgesics and was discharged from the hospital, when stable. This rare case is reported here as a learning opportunity for all physicians and general practitioners to keep this complication in mind should a similar case arise in the future.

Keywords: Plasmodium Vivax, malaria, splenic infarction.

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Introduction

Malaria is a serious parasitic infection in tropical countries. According to the World Health Organization, Pakistan is one of the seven countries in the Eastern Mediterranean region that account for 98% of the total malaria burden in the region. Around 217 million people in Pakistan are at moderate risk and 63 million people are at high risk for malaria.[1] In Pakistan two species are most common; Plasmodium Vivax and Plasmodium Falciparum. Almost all severe forms of malaria are caused by P.falciparum, but serious complication such as severe anemia, respiratory distress, splenic complications, shock and multiple organ dysfunction can also develop with P. Vivax infection.[2]

Case Presentation

A woman, 35-year-old, with no previous comorbidities presented with high-grade fever with rigors and chills for the last 8 days. Onset of fever was sudden, and at night time, but was relieved by taking antipyretics. She also complained of abdominal pain for the last 6 days, which was severe in intensity more in the left upper quadrant, throbbing in nature, continuous, with some mild improvement after taking analgesics. At the time of admission, she was conscious and oriented with a GCS of 15/15. Vitals were recorded as: blood pressure 120/80 mmHg, pulse 110 beats/minute, temperature 102°F and respiratory rate 19 breaths/minute. The abdominal examination revealed marked tenderness in the left hypochondriac region. Rest of the systemic examination was unremarkable. Baseline investigations revealed a hemoglobin level of 6.6 g/dl with a Mean Corpuscular Volume of 74 fl. Total Leucocyte Count was 4.8×109 with a platelet count of 102,000. Immunochromatography was positive for P. Vivax malaria. Dengue NS-1 antigen was negative. Serum electrolytes included sodium levels at 138 mmol/L, potassium 3.5 mmol/L, chloride 100 mmol/L, and bicarbonate 16 mmol/L. Bilirubin was 1.1 mg/dL and serum creatinine were 0.7 mg/dl. Serum Amylase was 20 U/L. A Urine DR revealed protein 2+, hemoglobin 2+, RBC plenty and nitrite negative.

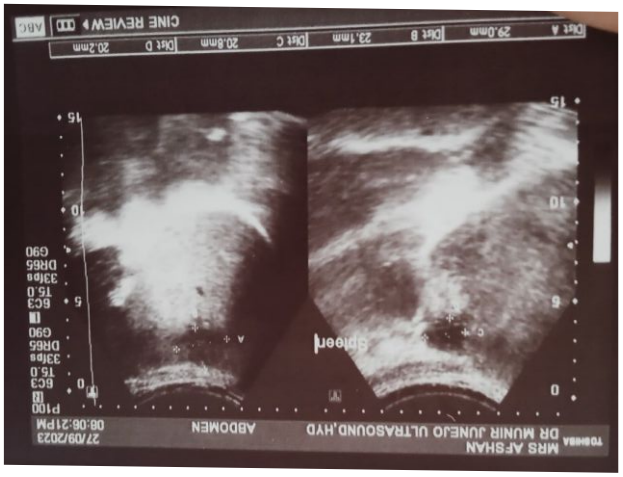

Figure 1: *Ultrasound abdomen revealed splenomegaly with multiple hypoechoic areas.*

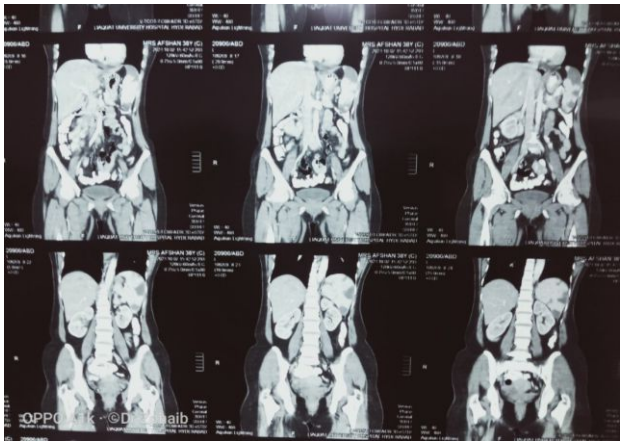

Figure 2: *CT scan abdomen with contrast revealed a splenic size of 11.3 cm with multiple wedge shaped non-enhancing hypo dense areas.*

Our differentials included pancreatitis, splenic abscess and peptic ulcer. Amylase came out to be normal. Splenic abscess was ruled out by the presence of non-enhancing lesions on CT scan abdomen. Injectable Artesunate was administered according to WHO guidelines during hospital admission along with antipyretics (paracetamol) and injectable Tramadol as an analgesic. Fever improved after 2 days along with gradual improvement in pain, hemoglobin level and platelet count. Patient was discharged with oral Artemether/lumefantrine and analgesics, followed by primaquine and advised to follow up after one week.

Discussion

Malaria is a parasitic infection caused by Plasmodium species. Splenomegaly is a common manifestation[3]. Splenic rupture, infarction, hyper-splenic, malarial hyper-reactive splenomegaly and splenic torsion are also reported[4]. Hyperemia of spleen, plasmodium infection endotoxin and allergic response are major possible causes of splenic infarction[5].

CT scan abdomen has 100 percent sensitivity 6 to diagnose splenic infarction. CT scan will show multiple wedge-shaped regions of low attenuation, which are distinctively different from those observed on CT images of splenic rupture or sub-capsular hematoma. Ultrasonography is another tool that can be used in the evaluation of splenomegaly, but this technique is less sensitive than CT in the acute stage of infarction. In our patient abdominal scan done after second day of pain was normal. Repeat ultrasound abdomen on 7th day of pain revealed splenic hypoechoic areas. Management of splenic infarction due to malaria is conservative which includes antimalarials and analgesics. We also prescribe Primaquine to decrease recurrence of P. Vivax malaria. A case of splenic rupture and abscess has been reported in a patient of splenic infarction due to other causes, but it is not reported in malaria so far[7].

Conclusion:

Splenic infarction is a rare complication of malaria. Our patient was diagnosed with a splenic infarct after P.Vivax infection. Splenic infarct was confirmed by contrast enhanced abdominal CT. Workup like ultrasound should be performed in a patient presenting with left hypochondrium pain after Plasmodium infection. Therefore, it is essential to keep in mind that such complications can be suspected in Plasmodium infections, especially with Vivax species.

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Authors' Contribution: Role and contribution of authors followed ICMJE recommendations

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