Case Report

Abdominal heaviness in malaria: An unusual splenic complication

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ABSTRACT

Splenic infarction as a complication in malaria is very sparsely reported despite having millions of cases of malaria every year. It appears to be an underreported complication and its recognition now can be attributed to the availability of better diagnostic and imaging modalities. It is important to look for splenic involvement in patients with malaria, especially in patients who complain of left hypochondrial pain as it might be associated with complications. Among the different species of malaria, like the case being reported here, it has been most commonly described with *vivax* malaria and outcomes have been almost always favorable with appropriate management of the infection.

Key words: Complications, malaria, splenic abscess, splenic infarction

INTRODUCTION

According to the latest WHO estimates (December 2016), there were 212 million cases of malaria in 2015 and 429,000 deaths.[1] Moreover, according to the National Vector Borne Diseases Control Programme estimates, India reported 1 million cases of malaria, out of which more than 700,000 were *falciparum* malaria with 242 deaths in total in 2016.[2] Malaria can have a wide spectrum of complications and most deaths are attributable to them. Among the rare but nonfatal complications are splenic infarcts. And, only around forty odd cases have been reported. We present a case of splenic infarction in a patient with *vivax* malaria.

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CASE REPORT

A 48 year old male, with no known comorbidities, presented to the emergency department, with a history of sudden-onset high-grade fever associated with chills and rigor for 10 days. Fever was intermittent, associated with chills and rigors. Along with this, he had noticed yellowish discoloration of his eyes which was gradually deepening and was associated with passage of dark-colored urine over the last 5 days. For the last 2 days, he complained of a dull-aching pain in his left upper abdomen. The pain was nonradiating, constantly present, without any aggravating or relieving factors, with no significant response to analgesics. His relatives noticed increased irritability in his behavior over the last 2–3 days. There was no history of otalgia, sore throat, cough or expectoration, vomiting, loose stools, burning micturition, or any decrease in urine output.

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He was evaluated outside since day 3 of his illness where all his preliminary workups for fever were done. His previous investigations revealed mild conjugated bilirubinemia and mildly elevated liver enzymes. Leukocyte counts were within normal limits, but thrombocytopenia and anemia were present. Erythrocyte sedimentation rate was elevated and investigations for malaria, dengue, chikungunya, and hepatitis A and E were negative.

On examination, he was conscious and oriented to time, place, and person but slightly irritable. His pulse was 116/min with blood pressure of 122/60 mmHg. He was febrile (105°F). Mild pallor and icterus were present. Respiratory rate was 22/min.

On gastrointestinal system examination, right and left hypochondrial tenderness was present. Liver was palpable 5 cm below the right costal margin, edges were rounded, and surface was smooth. Splenic tip was just palpable. Other systemic examinations were normal.

Since the basic investigations [Table 1] were already done, ruling out the common causes, the patient was further evaluated. Ultrasonography (USG) of the abdomen revealed hepatomegaly and mild splenomegaly, with the latter showing a peripheral hypoechoic lesion at the mid pole with the possibilities being infarct/evolving abscess [Figure 1]. Meanwhile, blood was sent for peripheral smear, culture, leptospira, scrub typhus, HIV, hepatitis B and hepatitis C, and Widal tests. Stool for routine microscopy and occult blood was sent.

Contrast-enhanced computed tomography (CT) of the abdomen done subsequently confirmed the presence of splenic infarct at the mid pole, as well as a similar smaller infarct at the posterior margin [Figure 2a and b].

The peripheral smear came out to be positive for *Plasmodium vivax* (parasite load: 3320/ml, schizonts, and trophozoites were seen) [Figure 3a and b].

The patient was started on antimalarials and became afebrile on day 2 of treatment. He was discharged in a hemodynamically stable condition and was followed up with repeat USG at 3 weeks, which showed resolution.

Table 1: Investigations at admission

Test	Value
Hb	8.7 g/dl
Platelet count	56,000/mm^3
TLC	3700/mm^3
DLC	$N_{56}L_{29}E_{22}M_1$
ESR	60 mm/h
Urea	34 mg/dl
Cr	0.6 mg/dl
Calcium	6.7 mg/dl
Phosphate	3.1 mg/dl
Uric acid	4.5 mg/dl
Na	137 mmol/L
K	4.2 mmol/L
Bilirubin (T)	3.2 mg/dl
Total protein	5.5 g/dl
Albumin	2.5 g/dl
SGOT (<50)	49 U/L
SGPT (<50)	11 IU
ALP	212 U/L
PT/INR	13.7 s/1.24

Hb: Hemoglobin, DLC: Differential leukocyte count, TLC: Total leukocyte count, ESR: Erythrocyte sedimentation rate, SGOT: Serum glutamic oxaloacetic transaminase, SGPT: Serum glutamate pyruvate transaminase, ALP: Alkaline phosphatase, PT: Prothrombin time, INR: International normalized ratio

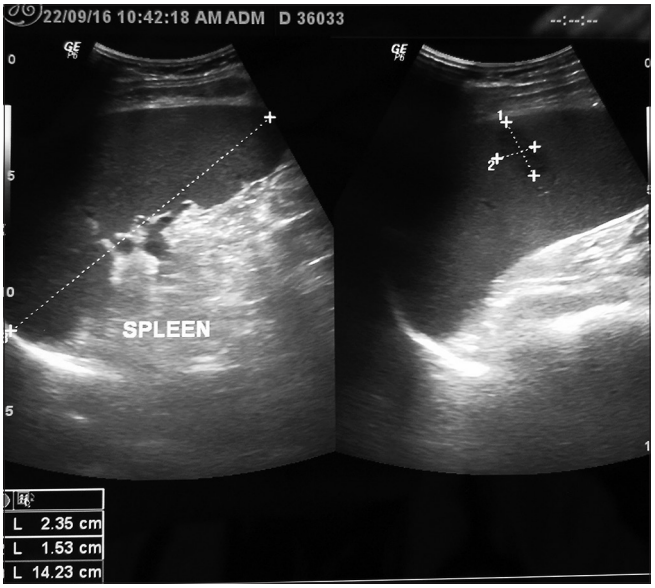

Figure 1: Ultrasonography: Hypoechoic lesion 13 mm × 14 mm peripherally at midpole

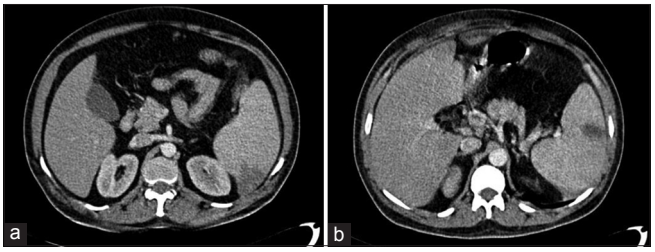

Figure 2: (a) Contrast enhanced computed tomography abdomen: Wedge shaped splenic infarct at the posterior pole. (b) Contrast enhanced computed tomography abdomen: Peripherally located splenic infarct

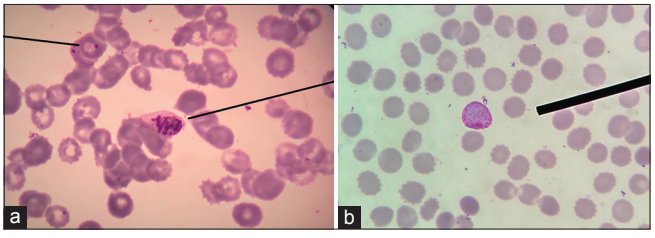

Figure 3: (a) Giemsa stain showing schizont and trophozoite of *Plasmodium vivax*. (b) Giemsa stain showing gametocyte of *Plasmodium vivax*

DISCUSSION

Malaria is known to be associated with many complications such as cerebral malaria, acute respiratory distress syndrome, acute renal failure, derangement of liver functions, hypoglycemia, leukopenia, thrombocytopenia, and severe metabolic acidosis.[3] Splenic complications as such are rare. There are case reports on splenic infarcts and abscesses in malaria. Of the 44 cases of splenic infarcts in malaria, 14 cases were due to infection with *Plasmodium falciparum*, 24 were due to *P. vivax*, 5 were mixed infections (*P. falciparum*/*P. vivax*), and 1 case was associated with *Plasmodium ovale* infection.[4] These reports are mostly recent and perhaps imply increased detection of such complications due to the advancement in imaging and diagnostics. Splenic abscess in malaria is very uncommon and is usually due to a superadded infection or a septic embolus to a prior infarct. Spontaneous splenic rupture is also reported in patients with malaria; however out of the 55 cases reported till date, only 2 were associated with splenic infarction.[5]

The pathogenesis of splenic infarction is not clear; however, the following factors can explain the hypoxic state attributable to splenic infarction: (1) hypercoagulable state; (2) intrasplenic structural change by adhesion of malaria-infected red blood cells (iRBCs) to endothelial cells, with rosetting of iRBCs and non-iRBCs and splenic cellular hyperplasia; and (3) anemic hypoxia.[6]

Splenic infarction is mostly seen in the acute stage, and therefore complaints of abdominal pain, especially in the left upper quadrant or a clinically palpable spleen in patients with malaria, should not be overlooked. An ultrasonological evaluation or a CT scan of the abdomen should be performed to look for any splenic involvement. However, routine USG/CT to actively seek for splenic complications in asymptomatic patients would be unreasonable, especially in resource-limited settings.

Identification of splenic infarction does not always warrant active intervention. It only requires meticulous observation and supportive treatment. Specific recommendations could be relative rest to minimize the chances of trauma to the already fragile spleen. The mainstay of medical therapy of splenic infarct is analgesia with either narcotics or nonsteroidal anti-inflammatory agents and prevention of secondary infection.[7]

It is important to follow up with repeat imaging to look for resolution or development of complications which include splenic abscess, sub-phrenic abscess, hemorrhage, pancreatic fistula, and gastric fistula. The above complications would usually require surgical intervention.[7] CT scan is more sensitive than ultrasound for evaluation of infarcts and other complications; however in resource-poor settings, it may not be readily available. It may be difficult to differentiate an abscess from an infarct on imaging alone; however, signs such as distribution of the lesion (random in abscess vs. oriented toward the capsule in infarct), margins (ill defined in abscess vs. sharp in infarct), and contrast enhancement (rim enhancement in abscess vs. none in infarct) may help us in making a correct diagnosis.[8] Persistent fever despite antimalarial therapy in the background of a splenic infarct should raise the suspicion of development of abscess; however, routine aspiration to rule out abscess should not be done.

Since spleen plays an important role in checking intravascular infections, all attempts should be made to conserve the spleen. Large splenic infarctions may lead to a degree of hyposplenism. Vaccination against common bacterial infections could be recommended in such patients.[4]

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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