SPLENIC COMPLICATIONS IN MALARIA: A CASE SERIES

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Abstract. Splenic complications in malaria may be either simple asymptomatic enlargement or serious conditions, such as splenic infarction, rupture, hematoma or abscess, which can be fatal. Only a few cases have been reported in the literature since 1960. The true incidence of splenic complications is not known because of underdiagnosis and underreporting. We report here four cases which were successfully treated conservatively.

INTRODUCTION

Malaria is a major cause of illness and death occurring in more than one million cases annually in tropical countries, including India (WHO, 2006). Hence clinicians should be aware of the clinical presentations and its common and rarer complications. The spectrum of structural changes of the spleen during malaria infection vary from asymptomatic enlargement to complications, such as splenic infarct, rupture, hemoperitoneum, ectopic spleen, hypersplenism, torsion, cyst or abscess formation (Contini and Lewis, 2006). These may lead to serious conditions which can be life-threatening.

Since 1960, only 35 cases have been reported in the literature . Among these, 25 cases had splenic rupture, 9 cases had splenic infarction and one case had splenic abscess associated with malaria (Ozoy *et al*, 2004; Contini *et al*, 2006). Its true incidence is not known because of underdiagnosis and underreporting.

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This article focuses on 4 case reports of splenic complications in malaria: two with splenic infarct and two with splenic rupture.

CASE SERIES

Case 1

A 41 year man was admitted to our hospital with fever, jaundice and progressive pain in the left hypochondrial area of 7 days duration. On examination he had a fever of 39.4ºC, pallor and jaundice, hepatosplenomegaly and a tender spleen. His complete blood count and renal function tests were within normal limits (serum creatinine was 0.9 mg/dl and blood urea nitrogen was 30 mg/dl). Urine analysis revealed no abnormalities (no RBC or casts). The malarial parasite fluorescent test was positive for schizonts and trophozoites of *P. vivax*. The serum total bilirubin was 17 mg/dl and the indirect bilirubin was 1.2 mg/dl. An ultrasonogram of the abdomen revealed an enlarged spleen of 17 cm in length with ill-defined subtle hypoechoic areas, the largest measuring 2.5 x 2.04 cm, along with hepatomegaly but no intrahepatobiliary dilatation. A CT scan of the abdomen confirmed the sonogram findings by revealing multiple ill defined peripheral wedge shaped hypodense parenchymal lesions in the spleen suggestive of infarcts (Fig 1).

The patient was treated with 600 mg of chloroquine base on Days 1 and 2, then 300 mg on Day 3 (total 1,500 mg), followed by primaquine 15.3 mg base daily for 14 days along with other supportive measures. Malaria parasites cleared from the peripheral blood, the patient improved symptomatically and was discharged from the hospital. A CT scan of the abdomen taken after one month showed a significant reduction in size of the spleen (from 17 cm to 13 cm) and resolution of the infarct (Fig 2).

Case 2

A 38 year male diagnosed with vivax malaria and partially treated with chloroquine was admitted to the hospital with a history of syncope, diffuse abdominal pain, and distention. The patient had no history of abdominal trauma. On examination he had hypotension (BP was 90/70 mmHg), pallor and jaundice. A tender distended abdomen with hepatosplenomgaly was also seen. His laboratory investigations revealed a hemoglobin of 10.3 g/dl (which decreased to 9.3 g/dl on the next day). The hematocrit and blood coagulation parameters were within normal limits. A thin blood film showed schizonts and rings forms of *P. vivax*. A serum amylase, renal function tests and electrolytes were within normal limits. The liver function tests showed unconjugated hyperbilirubinemia (total bilirubin 2.0 mg/dl and direct bilirubin 0.7mg/dl). A CT scan of the abdomen showed a bulky spleen with perisplenic fluid collection (thickness of 3.3 cm) around the upper pole of the spleen. A few pockets of interloop fluid were noted between the small bowel loops and in the pelvis suggestive of hemoperitoneum secondary to splenic rupture.

The patient was treated with same antimalarial regimen as in case 1, along with close monitoring of abdominal girth, blood pressure and hemoglobin level. No parasites were seen on the peripheral blood examination

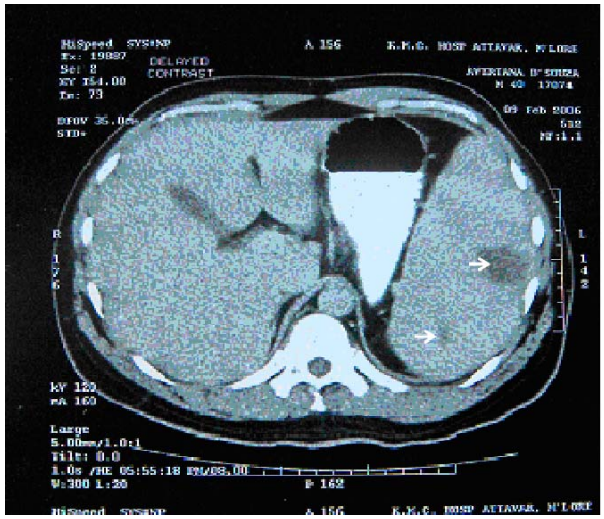

Fig 1–CT scan abdomen showing two ill defined peripheral wedge shaped hypodense parenchymal lesions in the spleen suggestive of infarcts.

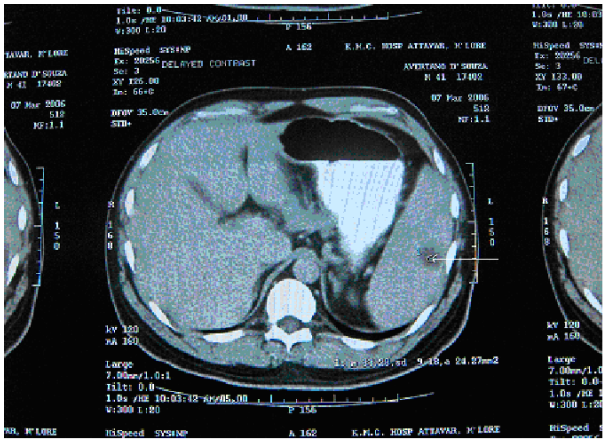

Fig 2–Repeat CT scan of the abdomen (after a month) showing a significant reduction in the size of the spleen (from 17 cm to 13 cm) and resolution of infarcts.

after 72 hours. The patient improved symptomatically and his hemoglobin level increased to 11.5 g/dl at the time of discharge.

Case 3

A 65 year male presented to our emergency department with a complaint of fever

associated with chills and rigor. A physical exam revealed pallor, hypotension BP (90/60 mmHg) and splenomegaly. The patient had no history of abdominal trauma. He had a low hemoglobin (8.4 g/dl) and platelet count (94,000/mm^3). He had hyperbilirubinemia (total bilirubin 5.0, direct bilirubin 4.0), abnormal renal function tests (serum creatinine 1.8, blood urea nitrogen 82 g/dl). A peripheral blood film revealed schizonts of *P. vivax* of 4+, trophozoites of both vivax (4+) and falciparum (1+). On the second day of hospitalization (seventh day of fever) he developed left hypochondriac pain. A tender spleen was noted on examination. An MRI of the abdomen showed features suggestive of splenomegaly with multiple sub-capsular infarcts. A blood culture did not yield any growth.

The patient was treated with IV Artesunate 120 mg followed by 60 mg daily for 5 days followed by mefloquine 500 mg once daily for 3 days and primaquine 15.3 mg once daily for 14 days.

Case 4

A 15 year male was admitted to the hospital with high fever (39.4ºC) associated with chills and rigor of 1 week duration and left hypochondrial pain of 2 days duration, which was moderate to severe in intensity. On examination, jaundice and tender splenomegaly were noted. Blood investigations showed anemia (hemoglobin 9.2 g/dl), a low platelet count (95,000/mm^3) and a high serum indirect bilirubin (3.5 mg/dl). A peripheral smear showed a mixed malarial infection (both *P. vivax* and *P. falciparum*). An initial ultrasonogram of the abdomen showed splenomegaly with a normal echo texture. After 4 days, because of worsening abdominal pain and tachycardia pulse (108 beats/min), sonography was repeated which showed an increase in spleen size with a 14 mm hypo/isoechoic band noted in the interpolar area of the spleen with a mild amount of free fluid in the pelvis, suggestive of hemoperitoneum with a ruptured spleen. Both cases 3 and 4 were treated with a full course of Artesunate followed by mefloquine 500 mg daily for 3 days, upon which the patient, improved symptomatically. In the last 3 cases imaging abnormalities resolved within 3 months of treatment.

DISCUSSION

Splenic enlargement is common in malaria. It depends on the immune status of the individual. If the disease remains untreated it can progress and result in splenic rupture. *P. vivax* malaria is the most closely associated with splenic rupture. Spontaneous rupture of the spleen occurs almost exclusively during a primary attack (Zingman and Viner, 1993). The incidence of splenic hematoma without rupture is unknown (Mokashi *et al*, 1992). Splenic infarction is rarer than rupture and may go unnoticed (Bonnard *et al*, 2005).

In all 4 cases above, *P. vivax* was the common species present. Each patient presented with left hypochondriac pain. This suggests that vivax malaria may be associated with splenic complications, which usually develops at the end of the first week of fever. Clinicians must be aware that left hypochondrial pain occurring during treatment for acute malaria may be due to splenic infarction. The clinical outcome was favorable in all four cases with conservative management. The spleen plays an integral role in the host defense against plasmodium and other intravascular parasites (Hamel *et al*, 2002). An attempt at splenic salvage should be made in order to avoid future fatal malaria infections and possibility of remission (WHO, 2006). A conservative approach, including complete bed rest, close observation of vitals and hematocrit levels, a correct dose of antimalarial medication, fluid resuscitaton, blood transfusion as needed and avoiding coughing and vomiting to prevent an increase in intraabdominal pressure are all important

measures. If the patient becomes hemodynamically unstable with conservative management, a laparotomy may be indicated. A conservative surgical approach with splenic repair or only partial excision, is preferred. Splenectomy should be performed only as a last resort (Imbert *et al*, 2006).

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