Splenic complications in malaria: report of two cases from Turkey

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Malaria is still a major health problem in Turkey, where Plasmodium vivax malaria is endemic. Spontaneous rupture of the spleen is an important and life-threatening complication and occurs in up to an estimated 2% of cases. Hence the small number of case reports suggests underreporting or underdiagnosis. Review articles have reported only 18 malaria cases with spontaneous splenic rupture in the English language literature since 1960. Two cases of P. vivax malaria with splenic complications are reported here. One of them showed signs and symptoms of acute abdominal pain, then splenic rupture occurred.

Introduction

Malaria has long been among the most common diseases in the southeast Anatolia region of Turkey. In Turkey, Plasmodium vivax is the predominant Plasmodium species (99.9%) (Akdur, 2004). The spleen plays an important role (especially in areas where malaria is endemic) producing antibodies against the malarial parasites (Mokashi et al., 1992). Changes in splenic structure during the course of malaria can result in asymptomatic enlargement or complications such as haematoma formation, rupture, hypersplenism, ectopic spleen, torsion or cyst formation. Primary exposure to malaria, as well as infection with P. vivax, appear to be important in predisposing to spontaneous splenic rupture (Mokashi et al., 1992; Zingman & Viner, 1993). Several changes of the abdominal musculature and peritoneal folds such as stretching, relaxation and elongation cause extensive splenic mobility. Splenic cysts may occur by different mechanisms. First, splenic congestion, infarction and haemorrhage may lead to haematoma formation with or without rupture, with subsequent organization and fibrous encapsulation around a blood cyst. Absorption of the blood may then produce a serous cyst (Zingman & Viner, 1993). In this study, we report two patients with P. vivax malaria: one had a splenic haematoma and the other had splenic rupture.

Case reports

Case 1. A 21-year-old soldier with poor co-operation due to high temperature, presenting with fever, malaise, chills, nausea and vomiting for the previous 15 days, was admitted to the Department of Infectious Disease and Clinical Microbiology. He had been on duty in the southeastern region of Turkey for a long time. Malaria is endemic in that region. He did not have history of glucose-6-phosphate dehydrogenase deficiency. Physical examination revealed a pulse of 98 min⁻¹, blood pressure of 100/60 mmHg, and a temperature of 38.2 °C. Conjunctival pallor and abdominal distension were noted. The spleen was palpable 5–6 cm below the left costal margin. Laboratory studies revealed the following values: white blood cell count of 3.2 × 10⁹ l⁻¹ (77% neutrophils), a haemoglobin level of 7.3 g dl⁻¹, a haematocrit of 21%, a platelet count of 58 × 10⁹ l⁻¹ and an erythrocyte sedimentation rate (ESR) of 65 mm h⁻¹. Trophozoites and schizonts of P. vivax were seen on peripheral blood smears and malaria was diagnosed.

The patient was treated with a standard course of chloroquine (1500 mg base) over 3 days, followed by primaquine (15 mg daily) for 14 days. Malarial parasites were cleared from the peripheral blood in 72 h. He became asymptomatic in 3 days and remained well in follow-up. The abdominal ultrasonography revealed the enlarged spleen. There was a 10–15 mm anechoic crescentic collection in the lateral subcapsular area (splenic haematoma) (Fig. 1). Sequential ultrasonographies showed spontaneous regression of the splenic haematoma. The haematoma was subsequently organized, and there was no evidence of splenic laceration or blood in the abdominal space. The long axis of the spleen normally measures less than 120 mm. The axis measurements of the spleen of the patient were 155, 141, 132 and 115 mm on hospital days 7, 11, 20 and 35, respectively. The patient was discharged on day 35. At this stage, he was asymptomatic, physical examination revealed a normal spleen, and ultrasonography showed no evidence of splenic haematoma.

Case 2. A 20-year-old soldier from the same region as the first case was admitted to hospital with a 5-day history of intermittent and high-grade fevers with chills and with sudden onset of right upper abdominal pain radiating to the left shoulder. On admission, he was conscious, though 

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Introduction

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Case 2. A 20-year-old soldier from the same region as the first case was admitted to hospital with a 5-day history of intermittent and high-grade fevers with chills and with sudden onset of right upper abdominal pain radiating to the left shoulder. On admission, he was conscious, though
unco-operative and exhausted. He did not have history of glucose-6-phosphate dehydrogenase deficiency. Physical examination was remarkable for a pulse rate of 118 min⁻¹, blood pressure of 90/60 mmHg, and temperature of 38.9 °C. Diffuse abdominal tenderness was present. The liver was palpable 3 cm and the spleen 4 cm below the costal margins. Laboratory studies revealed a white blood cell count of 18.2 × 10⁹ l⁻¹ (84 % neutrophils), a haemoglobin level of 10.4 g l⁻¹, a haematocrit of 32 %, a platelet count of 82 × 10⁹ l⁻¹ and an ESR of 80 mm h⁻¹. An abdominal ultrasound scan showed an enlarged, ruptured spleen and free blood in the Pouch of Douglas and the paracolic space. Trophozoites (ring forms) and schizonts of P. vivax were observed in peripheral blood smears. The patient was transferred to the surgical ward and a splenectomy was performed. Pathological examination revealed that the spleen weighed 900 g and measured 170 × 30 × 80 mm. Gross examination showed a greyish-brown discoloured spleen with capsular tears. Microscopy revealed congestion and dilatation of sinusoids and mononuclear infiltration with focal necrosis in capillaries and splenic pulp. A few red blood cells with P. vivax were seen on Giemsa staining. A standard course of chloroquine (1500 mg base) given over 3 days, followed by primaquine (15 mg daily) for 14 days, was prescribed. Two months later, the patient was readmitted to hospital with a 2-day history of fever. A peripheral blood smear again showed schizonts and trophozoites of P. vivax. The patient admitted to non-compliance. This clinical situation was consistent with a relapse of P. vivax malaria and the patient was again treated with a standard course of chloroquine and primaquine as previously prescribed. The patient became asymptomatic within 24 h and no parasites were seen on peripheral blood examination at 48 h. The patient remained well following treatment, and at review 1 month later had remained asymptomatic, physical examination was unremarkable and no parasites were seen on a peripheral smear.

Discussion

Malaria is a particularly important problem in the Anatolia region of Turkey. The incidence of malaria in Turkey, while showing a peak in 1994, has shown a gradual decrease, particularly over the past 5 years (Akdur, 2004). Enhanced environmental hygiene and fighting against the vector, increasing socio-economic status of people living in the southeast region of Turkey, the sharp decrease in local immigration seen among people in the region, the increasing knowledge of healthcare workers and others, and prophylactic primaquine use are the main factors causing a dramatic decrease in the incidence of malaria in recent years.

Severe complicated malaria is most frequently caused by Plasmodium falciparum, and much less commonly by P. vivax or other Plasmodium species. Non-falciparum malaria infections are generally regarded as benign and as having few severe complications. This may lead to delayed or missed diagnosis of splenic complications, some of which may be life-threatening (Table 1). As such, physicians may be familiar with the complications of falciparum malaria but less so with those of non-falciparum infection (Hamel et al., 2002).

In Turkey, the predominant Plasmodium species is P. vivax (99.9 %) (Fig. 2). Since complications are rare, they may be easily misdiagnosed. Thus ultrasonography in patients with

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<th>Non-splenic😎</th>
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<td>Cerebral malaria</td>
<td>Splenic haematoma</td>
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<td>Acute renal failure</td>
<td>Splenic rupture</td>
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<td>Severe anaemia</td>
<td>Hypersplenism</td>
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<td>Pulmonary oedema</td>
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<td>Immune complex glomerulonephritis</td>
<td>Splenic torsion</td>
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<td>Splenic cysts</td>
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P. vivax malaria presenting with abdominal pain can be important in the early diagnosis of splenic complications. Until now there have been only two reported cases of splenic rupture in Turkey, and no cases of splenic haematoma in the context of P. vivax malaria in the literature (Yagmur et al., 2000).

A palpable spleen may be present within 3–4 days of the onset of symptoms and may be noted in 50–90 % of patients with malaria. The spleen may subsequently become more hyperaemic, swollen and tender with each febrile paroxysm, though partial resolution can occur between paroxysms. Following treatment, the spleen usually diminishes in size within days to weeks (Zingman & Viner, 1993).

Worldwide, malaria is estimated to be the primary cause of spontaneous splenic rupture. In most cases, the initial event appears to be the formation of a subcapsular haematoma. Minor or unnoticed local pressure (due to vomiting, bending, coughing, defecation, etc.), in addition to splenic haemorrhage, infarction, congestion and focal necrosis, lead to distention of the splenic capsule by the haematoma and subsequent rupture. Defective haemostasis, due to thrombocytopenia or treatment of fever with aspirin, may contribute to intrasplenic haemorrhage.

Though believed to be frequent, the incidence of subcapsular haematoma formation without rupture is unknown (Hamel et al., 2002). In our case with the splenic haematoma (case 1), no rupture was detected and the haematoma resolved spontaneously. In the second case, it is not known whether a splenic haematoma preceded the spleen rupture or not.

The incidence of rupture of the spleen in malaria is poorly defined. Spontaneous rupture of the spleen is an important and life-threatening complication and occurs in up to an estimated 2 % of cases. Most of the cases of spontaneous splenic rupture in malaria occur during acute infection and are associated with P. vivax, although there have been rare cases associated with other Plasmodium species. Fever, tachycardia, vomiting, prostration, abdominal pain, tender splenomegaly, hypovolaemia and rapidly worsening anaemia are common presenting features of spontaneous splenic rupture. Abdominal pain may be mild to severe, generalized or localized. When localized, pain may be in the splenic region alone, across the upper abdomen, or in the lower abdominal quadrants because of pooling of blood (Howard et al., 1973).

Confirmatory tests and procedures for the diagnosis of splenic rupture include diagnostic peritoneal lavage, arteriography, laparoscopy and abdominal CT scan or ultrasonography (Zingman & Viner, 1993). In our cases, the diagnosis of splenic haematoma and rupture was confirmed by ultrasonography (Fig. 1).

Review articles have reported only 18 malaria cases with spontaneous splenic rupture in the English language literature since 1960. The predominant Plasmodium species in these cases were P. vivax (11 patients), followed by P. falciparum (5 patients) and Plasmodium malariae (2 patients) (Yagmur et al., 2000; Hamel et al., 2002). In the study reported by Schmidt (1978), 1461 monkeys were inoculated with P. falciparum and 272 with P. vivax. Two deaths due to splenic rupture occurred in monkeys inoculated with P. vivax, and none in those with P. falciparum. Further, splenic enlargement was observed to be dramatically more pronounced in fatal cases of P. vivax infection than in those of P. falciparum infection. Some authorities claim that the same is true in humans, with splenomegaly being more pronounced during acute infection with P. vivax than with other malarial species (Zingman & Viner, 1993).

Splenic complications should be sought in patients with malaria who are suffering from symptoms of nausea, vomiting and abdominal pain and distention. If a splenic haematoma is diagnosed, it should be monitored by repeat ultrasonography to assess its progress or resolution. Additionally, physicians must be aware of the possibility of a diagnosis of malaria in any patient who has developed a splenic haematoma or splenic rupture if he/she has a recent history of fever.

In conclusion, primary exposure to malaria and infection with P. vivax appear to be important factors in spontaneous rupture of the spleen. The ability to properly diagnose and to
manage splenic complications of malaria is particularly important in malaria-endemic areas such as Turkey. Prophylactic precautions should be taken by tourists who travel to southeast Anatolia, especially during the summer.

References


