Spontaneous splenic rupture in complicated malaria is an uncommon cause of hemoperitoneum in the tropics. The exact incidence of splenic rupture is unknown, largely due to under-reporting, but has been estimated at \( \sim 2\% \). Its pathophysiology is linked to the formation of a subcapsular hematoma. Upon rupture, patients present with features of shock and peritonitis and in most cases (95%), computed tomography (CT) scan detects the splenic injury. Patients should be managed conservatively with splenectomy reserved for patients with shock and hemoperitoneum due to risk of post-splenectomy sepsis. We report the case of a 38-year-old man with severe malaria who presented with fever, chills and abdominal pains. A CT scan abdomen failed to reveal splenic parenchymal injury or any splenic extravasation of contrast. Conservative management was unsuccessful. Exploratory laparatomy confirmed the spleen as the site of bleeding necessitating a splenectomy.

**INTRODUCTION**

We present a case of splenic rupture in an adult with complicated malaria in East Africa. Despite the rarity of such presentations, this case has been published to remind clinicians about the complications of malaria that should be considered in the differential diagnosis of acute abdomen, even when radiological studies are inconclusive. The epidemiology, pathophysiology, clinical features, diagnosis and treatment of spontaneous splenic rupture in malaria are discussed.

**CASE REPORT**

A 38-year-old Caucasian male living in Tanzania complained of acute onset fever, chills and cough. He had a history of asthma and was initially treated for bronchitis, but the symptoms persisted. Six days later, he developed left upper quadrant abdominal pain that radiated to his left shoulder and severe hypotension. The initial malaria blood slide performed as an in-patient was positive and an ultrasound showed fluid in the abdomen. The patient was given an initial dose of artesunate for management of complicated malaria and referred to our facility for surgical and infectious disease specialist review because of ongoing abdominal pain and fever.

Upon arrival, the patient was sick-looking, mildly pale and jaundiced. He had a tachycardia of 122 bpm and a normal blood pressure (121/59 mmHg). On examination, he had a slightly distended abdomen, mild generalized tenderness but no acute peritonitis. The remaining physical examination was unremarkable. He had low hemoglobin (9.3 g/dl) with normal red cell indices: Mean Corpuscular Volume (81 fl) and Mean Corpuscular Hemoglobin (28.5 pg). A repeat blood slide for malaria showed parasitemia (4%, *Plasmodium falciparum*). Management for severe malaria was started with parenteral artemether and oral doxycycline.

A computed tomography (CT) scan abdomen with intravenous contrast showed free peritoneal fluid with no obvious splenic rupture or contrast extravasation. He was monitored closely in the intensive care unit with repeat hemoglobin levels. Over the next 16 h, however, the hemoglobin continued to fall from 9.3 to 7.4 mg/dl despite ongoing resuscitation with two pints whole blood. His vital signs remained normal though. His abdominal distension and pain also worsened, and therefore an exploratory laparatomy was done. This revealed \( \sim 2 \) l of frank blood without purulence or fecal contamination. The spleen was enlarged and very firm with a long circumferential upper pole laceration (involving the splenic capsule and not the true parenchyma as expected in a traumatic ruptured spleen).
laceration). The laceration was actively bleeding after mobilization of the massively engorged spleen. This rapid engorgement in acute malaria appeared to tamponade the laceration against the left lateral parietal peritoneal surface of the abdominal wall, explaining the inability to detect the laceration reliably radiologically. A splenectomy was performed. Postoperatively, the patient recovered without complications. He was discharged home after 8 days, fully recovered and advised to continue on atovaquone/proguanil for malaria prophylaxis in his asplenic state.

DISCUSSION

Malaria is endemic to much of the tropical world, resulting in over half a million [1] deaths each year. The role of the spleen in the immune response against malaria is well established. The splenic white pulp removes complement-coated blood-borne pathogens; and produces antibodies, plasma cells and memory cells in response to the specific trapped antigens. Splenic hyperfunction in severe malaria leads to complications ranging from splenomegaly (70–80% of cases) to splenic infarction and rupture. The real incidence of splenic rupture in malaria is unknown as there is believed to be substantial under-reporting of cases. However, it is estimated that perhaps because this parasite causes more pronounced splenomegaly than other malaria species [5].

Formation of a subcapsular hematoma is believed to be the initial event in most cases. Then, minor pressure (due to simple physiological activities like bending) is hypothesized to add to the splenic hemorrhage, infarction, congestion and focal necrosis caused by malaria ultimately resulting in splenic rupture [6]. Clinically, patients usually present acutely with overt hypotensive shock and peritonitis. They may also have a positive Kehr’s sign (diaphragmatic irritation leading to referred pain to the left shoulder) and Balance’s sign (palpable tender mass in the left upper quadrant). The CT scan is the preferred tool for the confirmation of diagnosis, with a sensitivity and specificity of 95% [7].

Patients who are hemodynamically stable have been managed successfully non-operatively [8]. Repeated ultrasonography or CT scan is done to assess the healing of the ruptured spleen, which is usually complete in 2–3 weeks [9]. Splenectomy should be reserved for patients with severe or ongoing shock that is nonresponsive to resuscitation with blood products. The risk of post-splenectomy sepsis, a well-established, potentially lethal complication of splenectomy, especially in children, has encouraged clinicians to avoid splenectomy when the clinical condition suggests that active bleeding has stopped.

The case described here involved splenic rupture due to complicated P. falciparum malaria in a non-immune patient living in a malaria endemic zone. Our patient came with a history of hypotensive shock (but a normal blood pressure), abdominal pain and a positive Kehr’s sign strongly suggesting splenic rupture though this was not visible on CT. The laceration, likely being tamponaded as described above and not extending deeply into the parenchyma, resulted in a slowed rate of bleeding after the initial episode of rupture and hence the rupture was not seen on the CT scan. The timing of the CT scan, a week after clinically when the first rupture likely occurred, may also have contributed to the lack of confirmation of the splenic injury preoperatively.

On histopathology, the spleen was enlarged but otherwise grossly and microscopically normal. Additionally, our patient had no history of trauma, no perisplenic adhesions and no other disease affecting the spleen; thus, it fulfilled the four criteria by Orloff and Peskins [10] for spontaneous splenic rupture. Attempts at conservative management had been made with regular monitoring and use of intravenous fluids and blood for our patient. This, however, was unsuccessful as the patient was deteriorating due to ongoing splenic bleeding necessitating splenectomy.

CONFLICT OF INTEREST STATEMENT

None declared.

REFERENCES

1. WHO. Malaria Fact sheet N°94 Updated March 2014. http://www.who.int/mediacentre/factsheets/fs094/en/ (June 2014, date last accessed).
2. Martelo OJ, Smoller M, Saladin TA. Malaria in American soldiers. Arch Intern Med 1969;123:383–7.
3. Khan MY, Zinneman HH, Hall WH. Vietnam malaria: clinical experience with 50 patients. Minn Med 1970;53:331–4.
4. Howard WA, Krotoski WA, Slonim MS, Contacos PG. Spontaneous splenic rupture in vivax malaria: case report. Mil Med 1973;138:32–5.
5. Imbert P, Rapp C, Buffet PA. Pathological rupture of the spleen in malaria: analysis of 55 cases (1958–2008). Travel Med Infect Dis 2009;7:147–159.
6. Tengman BS, Viner BL. Splenic complication in malaria: case report and review. Clin Infect Dis 1993;16:223–32.
7. Jeffrey RB, Laing FC, Federle MP, Goodman PC. Computed tomography of splenic trauma. Radiology 1981;141:729–32.
8. Clezy JK, Richens JE. Non-operative management of a spontaneously ruptured malarial spleen. Br J Surg 1985;72:990.
9. Ruptured spleen in the adult: an account of 205 cases with particular reference to non-operative management. Papua New Guinea Splenic Injury Study Group. Aust N Z J Surg 1989;59:549–53.
10. Orloff MJ, Peskin GW. Spontaneous rupture of the normal spleen: a surgical enigma. Surg Gynecol Obstet 1958;106:1–11.