Spontaneous rupture of a malarial spleen - A case report and review of literature

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Citation

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Abstract

Malaria has long been among the most common diseases in Southeast Asia. Spontaneous rupture of a malarial spleen is rare. Involvement of the spleen in malaria that results in splenomegaly renders this organ prone to complications such as rupture. In areas where malaria is endemic, spontaneous rupture of spleen is uncommon. Ruptures of malarial spleens do heal and attempts at splenic lavage/conservative approach should be the aim in their management. Splenectomy should be reserved for those patients with severe rupture or those with continued or recurrent bleeding. We report a case of falciparum malaria that presented with hemoperitoneum due to spontaneous splenic rupture. This patient had a laparotomy and splenectomy and made an uneventful recovery.

INTRODUCTION

Rupture of the spleen is a relatively common complication of trauma in this age of high-speed road traffic accidents. Many systemic disorders affecting the reticuloendothelial system, including infections and neoplasias, rarely present with spontaneous rupture of the spleen.[1] A still rarer subtype of rupture occurring spontaneously and arising from a normal spleen was recognized as a distinct clinicopathologic entity. This has been reported after apparently trivial insults such as vomiting and even coughing.

Changes in the structure of the spleen during the course of malaria can result in asymptomatic enlargement, or complications such as hematoma formation and rupture. The spleen plays an important role in malaria, producing antibodies against the malarial parasite. The splenic involvement in malaria causing splenomegaly makes it more prone to complications such as rupture.[1],[2] Pathological or spontaneous rupture of the malarial spleen, i.e., nontraumatic rupture is a rare complication as the malarial spleen is tougher than the normal spleen. Malarial parasites have been with us throughout human history. They probably originated in Africa (along with humans) and fossils of mosquitoes show that vectors for malaria have existed for at least 30 million years. Plasmodium parasites are highly specific with humans being the only vertebrate host and Anopheles mosquitoes the vectors.[3]

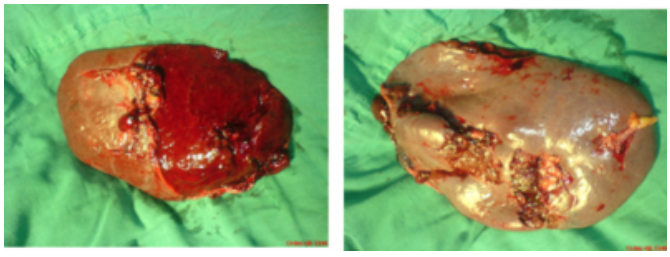
CASE REPORT

S. P., a 25-year-old male presented to our emergency department with an 8-day history of moderate to high-grade fever with chills and rigors. He also complained of a one-day history of generalized abdominal pain and abdominal distention. There was no hematemesis, melena or jaundice. He had no altered sensorium and no bleeding from any site.

On examination, the patient was febrile, 102ºF, and very pale (Hb 5.3g%). He had a tachycardia of 100/min and the blood pressure was normal. Abdominal findings revealed a generalized distension and tenderness, and guarding all over the abdomen. Per rectal examination was normal. Respiratory, cardiovascular and central nervous system examination revealed no abnormality. A peritoneal tap done in both flanks revealed frank blood that flowed very freely.

Figure 1

Fig. 1: (Diaphragmatic surface showing loss of splenic capsule, and visceral surface with multiple tears and ruptures.)



SMP was positive for falciparum malaria. BT, CT and platelet counts were normal. LFTs, RFTs, serum amylase, electrolytes and blood sugar levels were within normal limits. The chest x-ray was normal. Abdominal ultrasound showed a hepatospenomegaly. The spleen was enlarged 14cm below the costal margin with evidence of a heterogeneous, predominantly cystic area measuring 3.8cm in diameter, noted in the splenic midpole. There was a subcapsular hematoma measuring approximately 4cm in thickness with layering noted, with dense echoes noted within, and free fluid (+++) with internal echos. A CT scan confirmed these findings. Under general anaesthesia, a laparotomy was performed. We evacuated approximately 2 litres of hemoperitoneum and found a ruptured spleen. Splenectomy was performed because the spleen had multiple tears on the visceral surface, in addition to stripping of the splenic capsule on the diaphragmatic surface. The patient was started on antibiotics and antimalarials. He was also given pneumovac and Hib titre vaccines. He made an uneventful recovery and was discharged on the tenth post-operative day. The histopathology was consistent with spontaneous splenic rupture, and showed the characteristic hemozoin pigment in the Kupffer cells.

Figure 2

Fig. 2: Huge clot as big as the spleen.

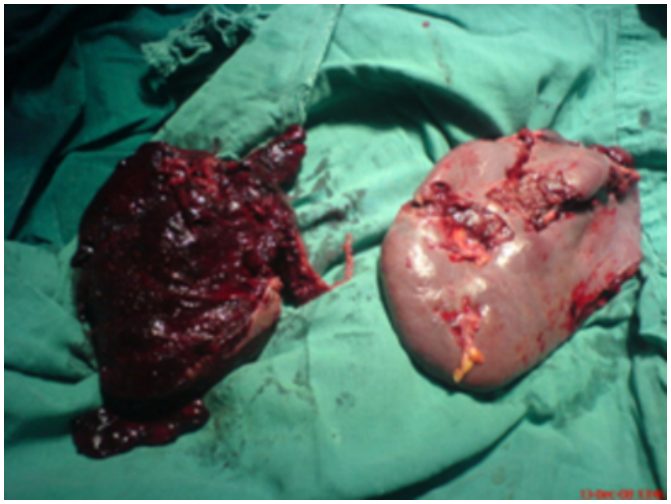
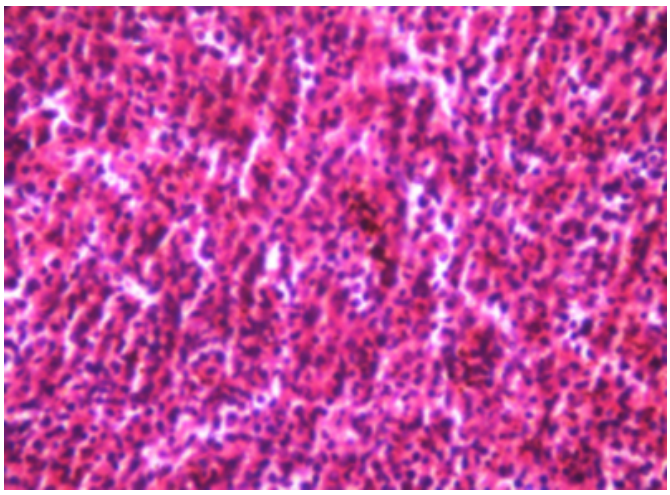


Figure 3

Fig. 3: Histopathology showing characteristic malarial pigment in Kupffer cells.



DISCUSSION

Plasmodium parasites are highly specific, with humans as the only vertebrate host and Anopheles mosquitoes as the vectors. This specificity of the parasites also indicates a long and adaptive relationship with humans.[3] The exact mechanism of spleen rupture is not known. However, three mechanisms have been implicated in the process.[2] The first of these mechanisms is increase in intrasplenic tension that is due to cellular hyperplasia and engorgement. Second, the spleen may be compressed by the abdominal musculature during physiological activities such as sneezing, coughing, defecation, and sitting up or turning in bed. Finally, vascular occlusion due to reticulo-endothelial hyperplasia, resulting in thrombosis and infarction, may be involved. This leads to interstitial and subcapsular haemorrhage and stripping of the capsule, which lead to further subcapsular haemorrhage. The distended capsule finally gives way. Despite the often massive splenic size in chronic malaria, spontaneous rupture of the spleen occurs almost always exclusively during acute infection and usually during the primary attack. This occurrence is probably due to rapid hyperplasia, stretching of splenic parenchyma and the capsule, a high frequency of small infarctions, haemorrhage, tears, a lack of extensive connective tissue and fibrosis (as found in chronic malarial spleens), an increased risk of minor stress to the spleen (e.g., vomiting, rigors) and a lack of prior immunity.[4],[5] Abdominal ultrasound will detect splenic rupture, subcapsular hematoma, perisplenic collection and free fluid (blood) in the peritoneal cavity. Computerized tomography (CT) scan of the abdomen will detect the smallest subcapsular hematoma of the spleen before the rupture

occurs. CT scan is most useful in diagnosis and monitoring a patient in whom conservative management for splenic rupture is considered. Peritoneal lavage or four-quadrant aspiration may be useful and reveal the presence of blood.

In areas in which malaria is endemic, there is growing evidence to suggest that management of spontaneous rupture of malarial spleen without splenectomy should be attempted. A conservative strategy is also reasonable in patients who travel frequently to malarious areas. Splenectomy should be reserved for those patients with severe rupture or those with continued or recurrent bleeding.[6] Increased preoperative and postoperative risk of splenectomy has led to attempted nonoperative management of splenic rupture (in cases of penetrating and blunt trauma) in many parts of the world. In addition to the significant morbidity and mortality of splenectomy itself, reasons to avoid splenectomy in areas where malaria is endemic include increased risk of fatal malaria, the possibility of remission, poor postoperative wound healing and the risk of cerebral malaria. Moreover, as fragmentation of the spleen is unusual when operative therapy is adopted, a large part of the spleen can be surgically repaired (splenorrhaphy)[4]. Nonoperative management consists of observation for 7-14 days in the hospital, strict bed rest, and administration of fluid and blood as needed[7]. A conservative strategy is also reasonable in patients who travel frequently to malarious areas. Apart from appropriate antimalarial therapy, splenectomy is accepted as the treatment of choice in cases of spontaneous rupture of the spleen. Splenectomy should be reserved for those patients with severe rupture or those with continued or recurrent bleeding [4]

Spontaneous splenic rupture is a fatal complication of malaria, which requires critical decision making in its management. Most malarial splenic ruptures do heal; hence, attempts at splenic salvage should be the aim in their management.[8]

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