Spontaneous Rupture of Malarial Spleen: a Case Report

Sitmada Spontan Dalak Rüptürü: Bir olgu Sunumu

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Abstract  
Spontaneous rupture of malarial spleen is a rare condition. A physician particularly works in a non endemic area, may find difficulty to diagnose this condition in such emergency cases. A spontaneous rupture of malarial spleen in a male in non endemic state of Kashmir was described. Even though the presentation looked like intestinal obstruction, abdominal ultrasonography and computed tomography revealed splenic rupture. Because of persistent hypotension, patient underwent an exploratory laparotomy. As a result, the patient was discharged after given pneumococcal vaccine and proper advice.

Özet  
Introduction
Malaria is still a major burden in many parts of the world. There is long and adaptive relationship with humans and malaria parasite. An intercontinental travel spreads malaria to non-endemic areas. The doctors who work in nonendemic area must also aware of manifestations and complications of malaria. Spleen is the most common affected organ in malaria. Malaria is the most frequent tropical infectious cause of spontaneous splenic rupture. Plasmodium vivax is closely associated with rupture of the spleen (1). The capsule becomes thin friable and easily prone to rupture. Microscopic examination depicts haemozoin, parasitized and uninfected erythrocytes, and a massive proliferation of macrophages throughout the capillaries, venous sinuses and pulp spaces. Management varies from splenectomy to spleen conserving procedure.

Case Report
A 47 year old male traveler was referred from peripheral health centre with two days history of pain abdomen, vomiting, constipation and fever, managed there as case of intestinal obstruction. There was persistent hypotension. Past history revealed that patient had received treatment for Plasmodium Vivax malaria two months ago after.

General physical examination showed tachycardia of 116/min and blood pressure of 90/70 mm Hg. Abdominal examination showed distended abdomen and tenderness in left upper abdomen Hemoglobin of 11 gm/dl was present. No malarial parasite was seen on thick and thin film. X-ray of abdomen showed distended gut and multiple air fluid levels present. Abdominal sonography showed free fluid in pelvis, distended gut loops and a 16.5 centimeter nonhomogeneous spleen suggestive of splenic rupture, and computerized tomography scan of the abdomen depicting perihepatic and peripheral splenic tear that spared the hilum. In view of persistent hypotension, patient had exploratory laparotomy. Preoperative findings were 1.5 liter of blood in peritoneal cavity and spleen with multiple capsular rupture weighing 288 grams, measuring 16.5x8x3 cm, with a dark smooth external surface and a non-encapsulated fragmented area (Pic. 1). Microscopic examination showed infiltration of the cords and the sinusoids with lymphocytes, monocytes and numerous clusters of neutrophils, parenchymal hemorrhage with areas of hematoma from the rupture. Patient was given pneumococcal vaccine and was discharged with proper advice.

![Picture 1](image-url) The macroscopic view of ruptured spleen after splenectomy. A dark smooth external surface and a non-encapsulated fragmented area are observed.
Discussion
Malaria is endemic in many tropical and subtropical areas of the world. *Anopheles mosquitoes* acts as vectors whereas *Plasmodium* parasites are the only vertebrate host. A common organomegaly observed in the area where malaria is endemic is splenomegaly, found in 50-80% of some populations. Usually a palpable spleen is present, within 3-4 days of the onset of symptoms. There is progressive splenic enlargement when disease remains untreated. Other splenic complications due to malaria include torsion, cyst and hematoma formation, infarction, hypersplenism, ectopic spleen, and the hyper reactive malarial syndrome (2). Besides malaria diseases, infectious mononucleosis, splenic neoplasms and haematological malignancies may cause spontaneous rupture of spleen (3). Splenic rupture is a rare complication of *P falciparum* malaria, most frequently associated with *P vivax* malaria. Presence of hypotension, abdominal pain, or signs of intraabdominal bleeding should alert one to the possibility of splenic rupture, even if thick and thin blood films are negative.

Rupture is more frequent in nonimmune persons than in those living in endemic areas where multiple attacks result in gradual splenic enlargement, making rupture less likely. Disruption of the spleen capsule and single or multiple tears in the underlying parenchyma may be seen. Tears may be small or large and are present on any surface. A high index of suspicion is needed for an early diagnosis, as sometimes rupture simulate other acute surgical abdomen (4) as in our case. The exact mechanism of spleen rupture is not known. The mechanism for splenic enlargement implicated are (a) cellular hyperplasia and engorgement resulting in increase in intrasplenic tension, (b) splenic compression by abdominal musculature during physiological activities and (c) vascular occlusion due to reticulo -endothelial hyperplasia, resulting in thrombosis and infarction. There is interstitial and subcapsular hemorrhage with stripping of the capsule, this being vicious cycle of more and more subcapsular hemorrhage. The result is dehiscence of distended capsule. Gross examination shows congestion, hyperemia and deposition of haemozoin, imparting dark red color in acute malarial spleen. In chronic malarial spleen tends to be dark grey, heavy, often massively enlarged organ with a firm or hard capsule. Abdominal ultrasonography, computed tomography scan, and arteriography can provide confirmation of the diagnosis in a hemodynamically stable patient (5). Arterial phase CT scan shows lack of parenchymal enhancement of the enlarged spleen and portal phase shows mild enhancement of the spleen (6). Similar findings could be observed in diffuse infiltrative diseases of the spleen such as amyloidosis, lymphoma and leukemia.

Splenectomy is accepted as the treatment of choice in cases of spontaneous rupture of the spleen. In areas in which malaria is endemic, there is growing evidence to suggest that management of spontaneous rupture of malarial spleen without splenectomy. This is reserved for those patients with severe rupture or those with continued or recurrent bleeding, in patients who travel frequently to malarious areas and in patients with a high likelihood of future exposure to malaria (1, 7). Nonoperative management consists of observation for 7-14 days in the hospital, strict bed rest, and administration of fluid and blood as needed (8).
References


