Correspondence

Spontaneous Rupture of the Spleen during Malaria: A Conservative Treatment Approach May Be Appropriate

Sir—Spontaneous rupture of the spleen is a rare complication of malaria. We report a case of splenic rupture that was successfully treated with a conservative approach.

A formerly healthy 23-year-old man was admitted to the hospital with a 2-week history of fever, myalgia, and diarrhea, which occurred after a stay of several weeks in Kenya. The patient had not received malaria prophylaxis. The initial physical examination showed that he was severely ill, with a body temperature of 39.8°C, blood pressure of 99/41 mmHg, and a heart rate of 126 beats/min. There was no abdominal tenderness or rigidity, and the spleen was not enlarged. Laboratory tests revealed a WBC count of 5.4 × 10^9 cells/L, a hemoglobin level of 128 g/L, a platelet count of 27 × 10^9 platelets/L, and parasitemia (Plasmodium falciparum percentage, 2.2%). Quinine treatment was administered intravenously.

Two days after admission to the hospital, the patient became dyspneic, with a respiratory rate of 35 breaths/min; the patient had an arterial partial pressure of oxygen of 10 kPa while breathing 3 L of oxygen/min. A chest radiograph showed bilateral infiltrates, and the left ventricular ejection percentage was 40%. Noninvasive ventilation and diuretic therapy promptly resolved these anomalies. On day 4 of hospitalization, abdominal tenderness and enlargement appeared, associated with a drop in the hemoglobin level to 62 g/L. Abdominal CT showed splenomegaly (maximum spleen diameter, 23 cm), with 2 areas of rupture, a contained hematoma, and an abundant hemoperitoneum (figure 1). Because hemodynamic parameters were stable, a conservative treatment approach was selected. The patient was discharged from the hospital 19 days after admission, after abdominal CT demonstrated that the perisplenic hematoma and the hemoperitoneum had decreased. The patient was regularly followed up for 8 months. The last CT scan, performed 1 month after admission, showed only a

Figure 1. Abdominal CT scan showing a splenic hematoma and hemoperitoneum
small anterior splenic hypodensity. Eight months after admission, the patient had no abdominal symptoms, and the findings of ultrasonography were normal.

Although spleen enlargement is a very common feature of malaria, the incidence of spontaneous splenic rupture is not well known; it ranges from 0% to 2%. Using the Medline database, we found 19 cases reported in the literature since 1960 [1, 2]. Formation and subsequent rupture of an initially contained hematoma is believed to be the usual mechanism of splenic rupture. In the present case, polypnea and the use of positive-pressure noninvasive ventilation may have played a role in the formation of a splenic hematoma.

To our knowledge, a very limited number of cases of spontaneous splenic rupture during malaria have been treated with a nonsurgical approach [2, 3]. Splenectomy has always been considered the treatment approach whenever possible. Few data are available concerning infection-related hemorrhagic shock is absent [4]. Few data are available concerning infection-related rupture of the spleen. However, the significant morbidity and mortality and the increased risk of complicated or fatal disease from exposure to malaria that are associated with splenectomy should lead to the use of a conservative treatment approach whenever possible.

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References


Agranulocytosis and Citrobacter Infection Associated with Jamu, a Herbal Remedy Containing Phenylbutazone

Sir—A 75-year-old woman with osteoarthritis obtained jamu, a herbal tonic from Indonesia, from a local health food store. Her pain quickly disappeared. Her regular regimen of medication included zopiclone, diclofenac (which she had been receiving for many years), and chondroitin/glucosamine capsules. After ingesting 8 sachets of jamu during a 6-week period, she was admitted to the hospital with a 3-day history of fever, sore throat, and dysphagia. She had cellulitis of the anterior neck and severe pharyngitis. Flexible nasendoscopy revealed erythema and edema of the glottic and supraglottic structures. Her temperature was 38.0°C. A complete blood cell count revealed a hemoglobin level of 11.3 g/dL, a WBC count of 0.3 × 10^9 cells/L (neutrophil count, 0.0 × 10^9 neutrophils/L), and a platelet count of 71 × 10^9 cells/L, with an erythrocyte sedimentation rate of 72 mm/h. Serum vitamin B12, folate, and immunoglobulin levels were within the normal range. Bone marrow aspirations showed plentiful megakaryocytes and erythroid precursors but an absence of neutrophils and myeloid precursors, which is consistent with a diagnosis of agranulocytosis. Treatment was started with dexamethasone, metronidazole, ticarcillin-clavulanate, and gentamicin. Cultures of blood samples and throat swab specimens subsequently yielded Citrobacter koseri that was susceptible to ticarcillin-clavulanate and gentamicin. Four doses of subcutaneous filgrastim (granulocyte colony-stimulating factor) were given. The patient gradually recovered, with the WBC count reaching a peak of 78 × 10^9 cells/L before returning to normal levels, and she was discharged.

The jamu was stated to contain ginger, black pepper, sinthok bark, Massoia aromatica Becc., Abrus precatorius (i.e., wild licorice), and Orthosiphon stamineus (i.e., Java tea). A literature search of the PubMed database revealed a report of fatal toxic epidermal necrosis associated with jamu supplemented with phenylbutazone [1]. Analysis of two 14-g sachets of the jamu confirmed the presence of phenylbutazone at a concentration of 0.32% weight-to-weight (45 mg of phenylbutazone per sachet). Phenylbutazone was detected using mass spectrometry for 7 principal transitions from its molecular species.

A report was found that described microbial contamination of jamu [2]. Cultures of jamu yielded Klebsiella pneumoniae, Enterobacter sakazakii, and Clostridium species but not C. koseri. The aerobic colony count was 8.0 × 10^6 colony-forming units per gram.

Phenylbutazone is an effective anti-inflammatory drug, but it was a common cause of agranulocytosis until its use was restricted in the United Kingdom to the treatment of ankylosing spondylitis [3]. The prognosis for neutropenia due to phenylbutazone use is generally good [4]. A causal relationship between exposure to phenylbutazone and agranulocytosis cannot be established with certainty in this case, because diclofenac has occasionally been associated with agranulocytosis [5],