

alerted to check the alcohol concentration in hand sanitizers because substandard products may be marketed to the public.

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## Spleen Abscess as Malaria Complication

**To the Editor:** Changes in spleen structure, frequently encountered during malaria, may result either in a simple asymptomatic enlargement or in serious complications such as hematoma, rupture, or infarction (1–3). Hematoma or infarction of the spleen might be followed by the development of a splenic abscess, a clinical condition that has been reported in only 1 patient, to our knowledge (4).

A 21-year-old woman sought treatment at the hospital outpatient department of “Emergency,” an Italian non-governmental organization (NGO) in Freetown, Sierra Leone in May 2004, reporting malaise and persisting dull abdominal pain, accompanied by isolated episodes of spiking fever. Several recurrent malaria attacks (*Plasmodium falciparum*) had been reported by this patient in the last 2 months. At physical examination, conjunctival pallor and a tender, enormously distended abdomen were observed. A large abdominal mass, extending from the xiphoid process to the pubis, was palpable. Lymph nodes (neck, axillary, inguinal) were normal. Laboratory features showed severe anemia (hemoglobin 62 g/L; hematocrit 0.24), with low platelet count ( $90 \times 10^3/\mu\text{L}$ ) and elevated leukocyte count ( $130 \times 10^3/\mu\text{L}$ ), together with a moderate increase in liver enzymes

(both aspartate aminotransferase and alanine aminotransferase were more than twice the upper limit of normal values). No malaria parasites were observed on blood smear at admission. Results of an HIV test were negative as were results of a sickle cell test, and hemoglobin electrophoresis results were normal. Other evident septic foci (e.g., typhoid fever, urinary tract infection, osteomyelitis) were excluded. Stool and urine examination excluded schistosomiasis. Blood cultures were not available.

An abdominal radiograph showed intraperitoneal fluid without distension of the bowel, whereas results of an abdominal ultrasound, performed in a private laboratory, diagnosed a large tumor on the left ovary. After receiving a blood transfusion (2 units) and intravenous antimicrobial drug treatment (ampicillin 500 mg 4 times/day, chloramphenicol 1 g 2 times/day, and metronidazole 500 mg, 2 times/day), the patient was scheduled for an exploratory laparotomy. Abdominal paracentesis was performed the day before surgery, and 2 L of thick brownish fluid was extracted.

An explorative laparotomy found  $\approx 3$  L of infected fluid in the peritoneal cavity. Widespread fibrin membranes covered thickened ileal loops. The mass was found to consist almost entirely a very large abscess on the spleen (Figure), which contained  $\approx 5$  L of pus. Dense adhesions were observed between the spleen, greater omentum, liver, and ileal loops. The liver was normal, and portal hypertension was not found. After splenectomy, the spleen’s length was found to be 48 cm, and its weight was 6 kg. On histologic examination, splenic tissue was found to have been replaced by congested inflammatory infiltrates and fibrotic tissue. Leishmaniasis was excluded at microscopic examination. The patient completely recovered after surgery.

An enlarged spleen is found in 50% to 80% of malaria patients (1),



Figure. Dimensions of the abscess cavity are shown during the operation.

while only 25 cases of splenic rupture have been reported since 1960 (0%–2% in natural occurring infection) (5). A break of a contained hematoma is usually involved in splenic rupture, which occurs almost exclusively during acute infection and the primary attack (6). The incidence of splenic hematoma without rupture is unknown (2).

Spleen infarction is rarer than rupture and may go unnoticed. Only 9 documented cases of splenic infarction associated with malaria have been reported (3), all consequent to *P. falciparum* infection (except in 1 patient who was coinfecting with *P. vivax* and 2 cases in which the etiologic agent was unknown). Splenic rupture following infarction has not yet been described.

Recently, an abscess of the spleen caused by *Salmonella enterica* serovar Enteritidis has been reported as a complication of *P. falciparum* malaria (4) and, to our knowledge, is the only case in the literature definitely related to *Plasmodium* infection. Indeed, splenic hematoma or infarction, together with the humoral and cellular immunodepression due to malaria, might well be predisposing

factors for bacterial (e.g., salmonellae) colonization of the spleen from the gut, as likely happened in this patient, although cultures of the pus, blood, or intraabdominal fluid were not performed. Bacteremia caused by nontyphoidal salmonellae was significantly associated with malaria parasitemia (7), and splenic abscess has been recently reported as an atypical presentation of salmonellosis (8). Splenic abscesses caused by *Salmonella* infection usually occur on preexisting lesions (4) and have been increasingly reported recently (9).

Because of its insidious symptoms, a spleen abscess remains a diagnostic challenge in developing countries, where ultrasounds and computed tomographic scans are not easily accessible. Moreover, as in our patient, a spleen abscess is unlikely to develop as an immediate complication of malaria.

While splenectomy was the only possible treatment in this patient, a conservative approach, whenever possible, is always desirable, especially in the tropics, where the exposure to infective agents is particularly widespread. The overall prognosis of splenic abscesses remains discourag-

ing, with 13%–16% of cases resulting in death (9), mainly consequent to late diagnosis and admission to a hospital. The growing volume of international travel will likely lead to an increase in the incidence of splenic complications in malaria patients, even in areas where the disease is not endemic. Therefore, clinicians should always keep the possibility of a superimposed abscess in mind.

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## Rickettsioses in South Korea, Materials and Methods

**To the Editor:** We read with interest the article by Choi et al. (1), which describes the molecular detection of *Rickettsia typhi* and 4 spotted fever group rickettsiae by nested polymerase chain reaction (PCR) in the serum of febrile Korean patients. The value of the study, however, is limited by imprecision, inconsistencies, and the impossibility of verifying data. First, neither epidemiologic nor clinical data are provided for studied patients, although these are essential for interpreting PCR results. Second, multiplex nested PCR is hampered by a high risk of contamination (2). Alternatively, nested PCR techniques that use a closed assay or single-use primers without positive controls limit such a risk (3). In all cases, the use of negative controls is critical (2,3). In this study, negative controls are neither described in the Materials and Methods section nor shown on the gels. In addition, the authors used as positive controls 4 of the 5

*Rickettsia* species they detected. Therefore, apart from *R. felis*, which was not used as a positive control, positive products may result from cross-contamination. Finally, technically, the data are impossible to reproduce: 1) primer sets WJ77/80 and WJ79/83/78 cited in the legends of Figures 2 and 3 are neither described nor referenced in the text, 2) sequence of the RpCS.877p primer in Table 1 differs from that in the referenced article (4), 3) described sequences have not been deposited in GenBank, and 4) all *rompB* primers described in Table 1 exhibit 1–6 nucleotide mismatches with *ompB* sequences of at least 1 of the detected species. Based on these errors, the 7 cases of dual infections with *R. conorii* and *R. typhi*, which have never been reported before, are doubtful, and these data need to be confirmed.

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## Rickettsioses in South Korea, Data Analysis

**To the Editor:** Choi et al. (1) conducted a study on sequence analysis of a partial *rompB* gene amplified from sera of humans who were seropositive for spotted fever group (SFG) and typhus group rickettsioses. They write, “These finding suggested that several kinds of rickettsial diseases, including boutonneuse fever, rickettsialpox, *R. felis* infection, and Japanese spotted fever... are occurring in Korea.”

These claims propagate some errors and may lead to an inadequate conclusion. First, *rompB* is conserved in *Rickettsia* spp. and consists of 4,968 bp with respect to the published sequence of the *R. conorii* strain Seven (2,3). Fournier et al. (4) amplified 4,682 bp of *rompB* and showed a high degree of nucleotide sequence similarity (99.2%) between *R. africae* and *R. sibirica*, *R. pakeri*, and *R. slovaca*. Choi et al. amplified ≈420 bp of *rompB* (position 3562–4077) for sequence analysis. This segment is located in a highly conserved region of the gene, which may decrease the ability to differentiate particular species from other SFG rickettsiae. This study cannot prove the existence of specific SFG rickettsioses until the results are confirmed further by, for example, isolating these SFG rickettsiae from humans, animals, or ticks in South Korea. Recently, the authors analyzed nucleotide sequences of 267-bp amplicons of *rompB* (position 4762–4496) obtained from patient sera and found that *R. conorii* could not be differentiated from *R. sibirica* (5). This finding also supports our concerns.

Second, although partial *rompB* nucleotide sequence analysis of rickettsiae obtained from 1 patient's serum showed 98.87% similarity with *R. conorii* strain Seven, the finding does not indicate boutonneuse fever is