Acalculous Cholecystitis in a Patient With Plasmodium falciparum Infection: A Case Report and Literature Review

Jean C. Yombi, MD, Christelle M. Meuris, MD, Alfons M. Van Gompel, MD, Myriam Ben Younes, MD, and Bernard C. Vandercam, MD

*Department of Internal Medicine, Unit of Infectious Diseases, St-Luc University Hospital, Brussels, Belgium; †Department of Clinical Sciences, Institute of Tropical Medicine (ITMA), Antwerpen, Belgium

DOI: 10.1111/j.1708-8305.2006.00023.x

Acute acalculous cholecystitis (AAC) can occur without gallstones in critically ill or injured patients and has also been associated with various infectious agents.¹⁻⁴ We report here a case of AAC in a patient with Plasmodium falciparum malaria.

Case Report

A 24-year-old Cameroonian female was admitted with a 4-day history of fever, headache, nausea, vomiting, and diffuse abdominal pain. The symptoms appeared 3 weeks after she had returned from Cameroon. It was her first trip back home in 9 years. On admission, her temperature was 39.5°C, her pulse 118 beats/min, and her blood pressure 95/50 mm Hg. Physical examination revealed tenderness on abdominal palpation.

Laboratory studies showed white blood cell (WBC) count of 4.1 × 10⁹/L, hemoglobin level of 107 g/L, and platelet count of 38 × 10⁹/L. The C-reactive protein (CRP) was 18 mg/dL (Normal value [N1] < 1 mg/dL). Serum aspartate aminotransferase (AST) level was 43 IU/L (N1 < 33 IU/L). Examination of blood thin smear revealed infestation with ring trophozoites, typical of Plasmodium falciparum with 6% of erythrocytes being parasitized. Treatment with intravenous quinine and oral doxycycline was started. Because of vomiting, doxycycline was replaced by intravenous clindamycin. Two days after, the percentage of erythrocytes parasitized was 1%, but the patient was still febrile (39.5°C). Physical examination showed increased pain and inspiratory arrest on subcostal palpation of the right upper quadrant. Liver function tests showed mildly elevated AST at 50 IU/L (N1 < 33 IU/L), alanine aminotransferase (ALT) at 70 IU/L (N1 < 63 IU/L) in the presence of a total bilirubin level of 1.5 mg/dL (N1 < 1.2 mg/dL), and a direct bilirubin level of 0.5 mg/dL (N1 < 0.2 mg/dL). The WBC count was normal (4.2 × 10⁹/L) in the presence of a CRP level of 21 mg/dL. An abdominal ultrasonography revealed a thickened gallbladder wall (10 mm) in the absence of calculi and in the presence of ultrasonographic Murphy sign, defined as maximum tenderness over the sonographically localized gallbladder. Intravenous cefuroxime and metronidazole were started. Within 24 hours, the fever as well as the abdominal pain disappeared. The treatment was completed with atovaquone/proguanil, and the patient was discharged after 3 days.

Discussion

This is the fourth, well-documented case of malaria-related acute acalculous cholecystitis (AAC).²⁻⁵ They were one Caucasian and two African females living in nonendemic area for many years before traveling to Africa. They were 46, 24, and 26 years of age, respectively. One case has also recently been described in a 7-year-old girl living in India.⁶ They had
Acute Acalculous Cholecystitis Associated With P falciparum Infection


In the context of malaria, the three pathophysiological mechanisms are present.

First, sequestration of parasites in the gallbladder microvasculature; removal of hepatic blood flow, anemia, and fluid losses may be involved in gallbladder ischemia.

Second, the fasting state is known to predispose to biliary stasis.

Finally, sequestration of infected erythrocytes is thought to initiate the local production of inflammatory cytokines and mediators.

Conclusion

In conclusion, AAC is a rare complication of P falciparum malaria. Clinical and biological findings are poorly specific, and the diagnosis of AAC remains a challenge. Ultrasound of the gallbladder should be considered in patients with right upper quadrant tenderness, persistent fever, leukocytosis, hyperamylasemia, or abnormal aminotransferases.

Although cholecystectomy or cholecystostomy remain the mainstay of therapy, the review of four cases suggests that in young patients without underlying disease, AAC could be successfully treated, under close monitoring, by antimalarial and antibiotic treatment without surgical intervention. However, in the absence of larger case studies and because of the high rate of complications observed in non-malaria-related AAC, surgery should always be considered especially in critically ill patients, particularly older patients with a high WBC count, in patients with complicated AAC, in patients not responding rapidly to medical treatment, and in the presence of conditions predisposing to gallbladder ischemia.

Acknowledgments

Both the first author and second author equally contributed to the work.

Declaration of interests

The authors state that they have no conflicts of interest.

References