

Case Report

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Acalculous cholecystitis by *P. falciparum* in a 3-year old child

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Acute cholecystitis involves inflammation of the gallbladder and can be seen with cholelithiasis (calculous) or in the absence of gallstones (acalculous). Acute acalculous cholecystitis (ACC) is rare, but important cause of cholecystitis in pediatrics¹. It is estimated that in children, 30–50% of cholecystitis cases are acalculous, compared with 2 to 17% of cases in adult patients². In children ACC mainly result from antecedent bacteremia and localization of bacteria in the gallbladder. So the majority cases have been managed by medical treatment alone. Gastrointestinal manifestations are common in malaria³, but the ACC is extremely a rare manifestation of malaria and a very few cases have been reported previously. Here we report a case of ACC attributed to severe *Plasmodium falciparum* malaria infection in a 3-year old child.

A 3-year old female child presented with history of intermittent fever since five days associated with rigors. History of abdominal pain and vomiting was present. Vomiting was non-projectile and non-bilious. On physical examination she was irritable, temperature 39°C, pulse rate 120/min, respiratory rate 28/min and blood pressure 96/60 mm Hg. She was severely pale. Icterus, cyanosis or lymphadenopathy was absent. On per abdomen examination revealed tenderness in right upper quadrant. Hepatosplenomegaly was present. Murphy's sign was positive. Other systemic examinations were normal.

On investigation, peripheral smear showed a ring stage of *P. falciparum* and the parasite density was 50,000/ μ l. The rapid diagnostic test (antigen HRP2) for *P. falciparum* was positive. Complete blood cell count showed a hemoglobin 5.4 g%, WBC 14100 cu/ μ l (85% polymorphs, 42% lymphocytes, 2% eosinophils and 1% monocytes), hematocrit 17.5, platelet count 56,000/mm³. Liver function and renal function tests were within normal limit. Widal test was non-reactive. Blood and urine culture were sterile. Hemoglobin electrophoresis was AA type. USG abdomen revealed thickened gallbladder (wall thickness was 6 mm) and hepatosplenomegaly. No abnormality detected on X-ray abdomen and chest. Child was put on IV antibiotics and antimalarial (artesunate) drug. Packed-cell blood transfusion was given. On the basis of clinical and USG findings the diagnosis of acute cholecystitis was made. After five days of treatment, child condition was improved.

ACC has been reported with various infectious agents like *Salmonella typhi*, *Escherichia coli*, *Mycoplasma pneumoniae*, leptospirosis, brucellosis, rocky mountain spotted fever, group A streptococci, and *Staphylococcus aureus* sepsis. In immunocompromised hosts, acalculous cholecystitis has been reported in fungal infection with *Candida* or *Aspergillus*, *Giardia* and *Cryptosporidium*¹. The diagnosis of ACC was made according to clinical features and sonographic findings. The clinical manifestations were fever, right

upper quadrant tenderness, and a positive Murphy's sign. Sonographic findings were a thickened gallbladder wall (defined as wall thickness > 4 mm) in absence of ascitis and hypoalbuminemia, a positive sonographic Murphy's sign (defined as maximum tenderness of the sonographically localized gallbladder), pericholecystic fluid collection, and no stone(s) in the gallbladder⁴. Patients had no recent history of burns, trauma, vasculitis, or recent surgery.

The exact pathogenesis of ACC is not clearly understood, but cholestasis and increased bile viscosity from prolonged fasting, spasm of the ampulla of Vater, endotoxemia, microangiopathy and ischemic-reperfusion injury, among the other causes have been suggested⁵⁻⁷. Intense sequestration of parasite in vascular endothelium, visceral ischemia and increased vascular permeability⁸, are well-known pathogenesises of falciparum malaria responsible for major gastrointestinal manifestations. Similarly, it appears to be responsible for the pathogenesis of acute cholecystitis in our case. The other possible reason for ACC in falciparum malaria could be *Salmonella* infection, as it is related to a transient immunosuppression induced by malaria⁹, but in our case blood culture was negative. So in this case the former mechanism appear to be involved for the pathogenesis of ACC. Although the pathogenesis of ACC remains controversial, malaria should be included in the differential diagnosis of ACC, especially in endemic zones of malaria.

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