

Acute Acalculous Cholecystitis in Plasmodium Falciparum Malaria

Authors

Meenaxi Sharda¹, Shivraj Meena², Deepti Nagar³, Anil Soni⁴

¹Professor, Department of Medicine, Govt. Medical College Kota, 4 B 14, Talwandi Kota Rajasthan India

²Assistant Professor, Department of Medicine, Govt. Medical College Kota, Type II /08 Medical College Campus Kota Rajasthan India

^{3&4}Resident, Department of Medicine, Govt. Medical College Kota

Email: *meenakshisharda@gmail.com¹, Shivraj.aiims@gmail.com², dnag87@gmail.com³, infarnaz@gmail.com⁴*

Corresponding Author

Dr. Shivraj Meena

Type II/08 Medical College Campus, Govt. Medical College Kota Rajasthan

ABSTRACT

Malaria is one of the major health problems in India. Gastrointestinal manifestations are quite common in malaria. AAC is a rare manifestation seen in P. Falciparum malaria. We report a case of AAC in P. Falciparum malaria in a 13 year old girl. Acute acalculous cholecystitis (AAC) has been described in association with various infectious agents. AAC can be due to bile stasis or gall bladder ischemia. But it remains to be determined whether AAC in P. Falciparum patients is directly related to malaria or to a secondary gallbladder bacterial infection.

Keywords: *Acalculous Cholecystitis, malaria, plasmodium falciparum*

INTRODUCTION

Malaria is one of the major health problems in India .ACC involves inflammation of the gallbladder and can be seen with cholelithiasis (calculus) or in the absence of gallstones (Acalculous). Acute acalculous cholecystitis 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. AAC can be due to bile stasis or gall bladder ischemia. Gastrointestinal manifestations are common in malaria⁴, but the ACC is extremely rare and a very few cases have been reported

previously. Here we report a case of AAC in P.F malaria in a 13 year old girl.

CASE SUMMARY

A 13 year old Hindu unmarried girl presented with history of high grade fever with chills & rigors since 10 days associated with nausea and vomiting. She also complained of pain in right hypochondrium since 4 days. There was no history of diarrhoea, headache, urinary complaints, chest pain, cough and no recent history of burns, trauma, surgery and any specific drug intake except symptomatic treatment of fever. On admission her temperature was 100F. On general examination pallor and icterus were present; there was no cyanosis, pedal edema, lymphadenopathy, clubbing and petechial hemorrhages. JVP was not raised, no signs of meningeal irritation, no evidence of vasculitis. Her pulse was 88/min with regular rhythm, blood pressure in right upper limb was 90/62mm Hg and respiratory rate was 18/min. On systemic examination abdomen was soft, non-distended, there was tenderness in right hypochondrium with Murphy's sign positive, no organomegaly and bowel sounds were present. Cardiovascular, respiratory system and central nervous system examination showed no abnormality.

Laboratory examination on admission revealed haemoglobin level 5.6g/dl, total leukocyte count of $3.69 \times 10^3/\text{ul}$ (85% polymorphs, 42% lymphocytes, 2% eosinophils and 1% monocytes), hematocrit 17.5, platelet count $22 \times 10^3 / \text{ul}$. On peripheral smear of blood showed normocytic normochromic anemia. Occult blood in stool was

negative. Thin smear examination of blood film revealed ring trophozoites typical of *Plasmodium falciparum* with low parasitemia (+). The rapid diagnostic test (antigen HRP2) for *P. falciparum* was positive. Serum bilirubin 1.8mg/dl (indirect bilirubin 1.6 mg/dl, direct bilirubin 0.20 mg/dl) SGPT 260 IU/L, SGOT 48 IU/L. Blood Urea, serum creatinine, blood sugar were within normal limits. Urine complete examination was also normal. Serology for *Salmonella typhi* and ELISA test for Dengue IgG, IgM antibodies were negative. Chest x-ray was normal.

Treatment with intravenous quinine (30mg/kg/d divided TID), oral doxycycline (2mg/kg PO q day) was started. Patient was also transfused 2 units of packed cell blood in view of anaemia. Patient was afebrile after 48hrs but right hypochondrium pain and vomiting persisted. The vomiting was initially attributed to quinine and hence was temporarily withdrawn but it did not subside. Meanwhile abdominal ultrasonography performed as a routine revealed a thickened gallbladder wall surrounded by a thin rim of pericholecystic fluid and a positive sonographic Murphy's sign. No stones were visible and a diagnosis of acalculous cholecystitis was made³. She was again started on quinine, in addition to anti malarial treatment she was advised to stay nil per oral and was given injectable ciprofloxacin (10mg/kg IV 12hourly), intravenous fluids and symptomatic treatment. Within 48 hours her abdominal pain and vomiting subsided. Patient fully recovered by day 9th, the anti malarial treatment was completed and patient was discharged. She was advised to return after 1 week

for a repeat abdominal ultrasonography, which revealed normal gall bladder wall thickness and no pericholecystic fluid collection.

DISCUSSION

Malaria is a major public health problem in India. Complications in *P. falciparum* malaria include cerebral malaria, severe anaemia, hypoglycemia, bleeding tendencies and shock. Gastrointestinal manifestations are also common in malaria², but AAC is a rare manifestation of malaria.

Acute acalculous cholecystitis (AAC) is acute inflammation of the gallbladder which occurs in the absence of stones. The pathogenesis of AAC is believed to be due to bile stasis and gallbladder ischemia⁵. Etiology of AAC³ includes trauma, vascular diseases (such as polyarteritis nodosa) and infectious agents, such as *Salmonella*, cytomegalovirus, *Cryptosporidium* and microsporidium.

The pathophysiologic mechanism for AAC in malaria can be explained by following three mechanisms; first, sequestration of parasites in the gallbladder microvasculature, reduction in hepatic blood flow, anaemia, and fluid losses may lead to gallbladder ischemia. Second, the fasting state is known to produce biliary stasis. Third, the sequestration of infected erythrocytes initiates local production of inflammatory cytokines and mediators⁶.

Another possible reason for ACC in *P. falciparum* malaria could be *Salmonella* infection, as secondary bacterial infections are common in malaria, which is related to transient immunosuppression induced by malaria⁷.

To best of our knowledge and search in the Indian medical literature we could only find 2 cases of AAC in *P. falciparum* malaria⁸⁻⁹. In western literature very few cases have been reported. We want to report this case due to its rarity and to highlight that persistent vomiting in a case of *P. falciparum* malaria may not only be due to the quinine intolerance as is commonly thought and simply an ultrasonography of abdomen may reveal AAC as the cause for persistent vomiting and pain abdomen and thus prevent premature withdrawal of quinine.

The complication rate for acalculous cholecystitis exceeds that for calculous cholecystitis. Successful management of acute acalculous cholecystitis appears to depend primarily on early diagnosis and surgical intervention, with meticulous attention to postoperative care⁹. In young patients without underlying disease, AAC could be successfully treated, by antimalarial and antibiotic treatment without surgical intervention.

REFERENCES

1. Soden J, Karpen SJ. Cholangitis and cholecystitis. In: Feigin, Cherry, Demmler, Kaplan, editors. Textbook of pediatric infectious diseases. V edn. Philadelphia: Saunders 2004; p. 675–6.
2. Sakayannis DE, Kozakewich HP, Lillehei CW. Acalculous cholecystitis in children. *J Pediatr Surg* 1996; 31:127–31.
3. Bilhart LE; Acalculous cholecystitis, adenomatosis, cholesterosis, and polyps of the gallbladder; In: Sliesinger MH, Fordtran JS, editors; *Gastrointestinal and*

- liver disease. VI edn Philadelphia: Saunders1999; p. 993–1005.
4. Murphy GS, Oldfield EC III. Falciparum malaria Infect Dis Clin North Am 1996; 10:751–2.
 5. Barie PS, Fischer E; Acute acalculous cholecystitis; J Am Coll Surg 1995; 180:232– 44.
 6. Jean C. Yombi et al; Acalculous Cholecystitis in a Patient With Plasmodium falciparum Infection: A Case Report and Literature Review; Journal of Travel Medicine, Volume 13, Issue 3, 2006, 178–180.
 7. Saha A, Batra P, Vilhekar KY, Chaturvedi P; Acute acalculous cholecystitis in a child with Plas modium falciparum malaria . Ann Trop Paed 2005; 25: 141 – 142.
 8. Arvind Kumar, Amar M. Taksande & K.Y. Vilhekar; J Vector Borne Dis 45, March 2008, pp. 76–77.
 9. Longo, Fauci, Kasper, Hauser, Jameson, Loscalzo; Harrison’s Principles of Internal Medicine; 18th edtn, volume 2, p 2621-2622.