JOURNAL OF CASE REPORTS 2012;2(2):117-119

An Unusual Presentation of Plasmodium Vivax Infection with Acute Acalculous Cholecystitis

Nikhil Gupta, Arun Garg, Saroj Sahoo[1]

From the Department of Medicine, University College of Medical Sciences & GTB Hospital, Dilshad Garden & Maulana Azad Medical College[1], Bahadur Shah Zafar Marg, University of Delhi, Delhi, India.

Abstract:

Malaria due to *Plasmodium vivax* is often regarded as a cause of benign infection. There is increasing evidence that the overall burden and severity of *Plasmodium vivax* has increased in the past decade. Now, *P. vivax* is associated with various complications such as acute renal failure, ARDS, hepatitis and is no longer considered a benign disease. *Plasmodium vivax* causing gastrointestinal complications are relatively common, but *P. vivax* causing acute acalculous cholecystitis is extremely rare. We report a rare case of acute acalculous cholecystitis due to *Plasmodium vivax* malaria.

Key words: Acalculous Cholecystitis, Malaria, Plasmodium vivax.

Introduction

Malaria is one of the major public health problems in India. Around 1.5 million confirmed cases are reported annually by the National Vector Borne Disease Control Programme (NVBDCP), of which 40–50% are due to *Plasmodium falciparum*. Complications in malaria are frequently associated with *P. falciparum* such as cerebral malaria, severe anemia, hypoglycemia, bleeding tendencies and shock. There are only few reports associating acute acalculous cholecystitis (AAC) with malaria, especially in patients infected with *Plasmodium vivax* [1]. We describe a case of *Plasmodium vivax* malarial infection complicated by AAC.

Case Report

A 55 year old female, presented to emergency department with 7 days history of high grade fever & chills, right sided abdominal pain and vomiting for 4 days, yellowish discolouration of eyes & urine for 3 days. On examination patient was conscious, oriented with blood pressure of 108/76 mm Hg, pulse rate of 110/min and temperature 39.7^0 C. Her general physical examination was unremarkable except for icterus. Abdominal examination revealed hepatomegaly 3 cm below the costal margin and splenomegaly of 2 cm below the costal margin.

Her investigations revealed haemoglobin of 12.3 g/L, leucocyte count 8.4 × 10^9/L, platelets 0.26 ×10^9/L. Liver function tests showed total bilirubin 224 µmol/L with direct component being 46.17 µmol/L, liver enzymes being normal. Peripheral smear revealed ring and gametocytes of *Plasmodium vivax*. Serum LDH was 784

Corresponding Author: Dr. Nikhil Gupta
Email: nik.gupta4u@gmail.com

U/L. Ultrasound abdomen revealed liver 15 cm, spleen 15.2cm, thickened (12 cm) and edematous gall bladder with pericholecystic edema, No calculi was seen in gall bladder. A diagnosis of *Plasmodium vivax* malaria with acalculous cholecystitis was kept and patient was started on fluids, injection ceftriaxone, pantoprazole, perinorm, tablet quinine and tablet doxycycline. On the fourth day patients fever, abdominal pain, and tenderness resolved. A repeat ultrasound at 1 month showed a normal gallbladder without wall thickening or pericholecystic fluid collection.

Discussion

Malaria is one of the major public health problems in India. Around 1.5 million confirmed cases are reported annually by the National Vector Borne Disease Control Programme (NVBDCP), of which 40–50% is due to *Plasmodium falciparum*. *Plasmodium vivax* causing gastrointestinal complications are relatively common, but *P. vivax* causing acute acalculous cholecystitis is extremely rare.

Acute acalculous cholecystitis (AAC) is acute inflammation of the gallbladder which occurs in the absence of stones. It is most commonly observed in the setting of ill patients (eg, on mechanical ventilation, with sepsis or severe burn injuries [1], after severe trauma). ACC is an extremely rare complication of *Plasmodium vivax* infection.

The main mechanism of this illness is bile stasis as a result of increased bile viscosity and impaired gallbladder contraction. Dehydration, fever and absence of oral feeding leads to decrease in the cholecystokinin-induced gallbladder contraction [2]. Also, gallbladder is predisposed to ischemia due to dehydration, hypotension, and sequestration of parasites in the gallbladder microvasculature. These factors alone or in combination play a major role in development of malaria induced AAC.

Patients with malarial AAC may present with symptoms of fever, right upper quadrant pain, nausea, and vomiting. There may be tenderness on the right hypochondrium. The time of the onset of AAC in malarial infection is not exactly known. Abdominal ultrasonography is the initial imaging study in the evaluation of suspected AAC. The following criteria are used for the diagnosis of AAC: gallbladder wall thickening > 4 mm (in the absence of ascites and hypoalbuminea), ultrasonographic Murphy's sign, and the presence of pericholecystic fluid in the absence of stones [3]. Our patient had gall bladder wall thickening of 12 mm with pericholecystic fluid without any evidence of stone. CT scan abdomen can be performed with 95% sensitivity and specificity rates [3].

Plasmodium vivax induced acalculous cholecystitis generally responds to medications. However, if the patient's clinical status deteriorates with increase in liver enzyme, surgical intervention may be indicated, and cholecystostomy should be done [4].

Conclusion

Plasmodium vivax was once considered as a benign infection. *Plasmodium vivax* can manifest with rare and dreaded manifestation such as acalculous cholecystitis. Thus, treating physician should keep in mind about such a treatable complication of *Plasmodium vivax* especially in patients presenting with right sided hypochondric pain.

References

1. Theodorou P, Maurer CA, Spanholtz TA, Phan TQ, Amini P, Perbix W, *et al*. Acalculous cholecystitis in severely burned patients: incidence and predisposing factors. Burns. 2009;35(3):405-411.
2. Pongponratn E, Riganti M, Punpoowong B, Aikawa M. Microvascular sequestration of parasitized erythrocytes in human falciparum malaria: a pathological study. Am J Trop Med Hyg. 1991;44:168–175.
3. Feldman M, Freidman LS, Brandt LJ. Sleisenger and Fordtran' Gastrointestinal and Liver Disease. 9th ed. Philadelphia, Pa: Saunders Elsevier; 2010:1139-1152.
4. Khan FY, El-Hiday AH. Acute acalculous cholecystitis complicating an imported case of mixed malaria caused by Plasmodium falciparum and Plasmodium vivax. Int J Inf Dis. 2010;14S:e217–219.