



Acute Pancreatitis - Rare Complication of Falciparum Malaria

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Abstract

A 26-yr old army man of commandant rank posted in forest belt of district Mirzapur Uttar Pradesh, presented to the medicine emergency department with a 4-day history of high-grade fever which was associated with chills and rigor, vomiting and generalized abdominal pain. Peripheral blood smear stained with Leishman demonstrated ring-form trophozoites of *P. falciparum* with high parasitemia (62%) with rapid antigen test for *P. falciparum* was also positive. Chest X-ray was normal. Non contrast Computed Tomography (CT) scan of the abdomen showing a bulky pancreas with peripancreatic fat stranding suggestive of acute pancreatitis. A diagnosis of severe falciparum malaria with acute pancreatitis was made on basis of investigation. Treatment in form of intravenous quinine 1000 mg as loading dose in 10% dextrose solution followed by 500 mg in same solution and was monitored for hypoglycemia. Oral quinine tab was started 8th day up to 15th day. The patient was discharged on 18th day and his conditions were good on further follow-up.

Keywords: Acute pancreatitis; Falciparum malaria; Pain abdomen; Fever; Anopheles; *Plasmodium falciparum* (*P. falciparum*)

Introduction

Falciparum malaria is a very common disorder that is prevalent in the tropics and associated with many complications. Falciparum malaria a protozoan disease is caused by *Plasmodium falciparum*, transmitted by the bite of infected Anopheles mosquitoes. Acute pancreatitis a rare complication of falciparum malaria which is well documented. It is rare complication because despite having high parasitemia there is no any other system involvement. A case report of a 26-yr old army man infected with falciparum malaria with high parasitemia and acute pancreatitis as a sole presentation was presented here. Only few case reports are available.

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Case Presentation

A 26-yr old army man of commandant rank posted in forest belt of Mirzapur district, Uttar Pradesh, presented to the medicine emergency department with a 4-day history of high-grade fever which was associated with chills and rigor, vomiting and generalized abdominal pain. There was no significant past medical history of lipid disorders, no history of biliary tract disease, and no steroids and painkillers intake. There was no history of alcohol intake or any other substance abuse. At the time of presentation on clinical examination, he was febrile (102.2°F) with mild jaundice. His temperature fluctuates over next 2 days, abdominal pain increases and he becomes drowsy. Four days after admission his condition improved. Blood pressure was normal. Systemic examinations reveal epigastric tenderness with mild generalized abdominal distension with normal bowel sounds. Systemic examinations of other system were within normal limits. Laboratory examination revealed hemoglobin 9.4 g/dL with total leucocyte count 6400/mm³ (with granulocytes 65.6%), platelet count 90,000, MCV 84.5 fl, MCH 30.2 pg and hematocrit 23.1%. Serum bilirubin was 4.2 mg/dL, aspartate aminotransferase 402 IU/L, alanine aminotransferase 202 IU/L, alkaline phosphatase 82 IU/L and serum albumin 2.8 g/dL with total protein of 6 mg/dL. His serum amylase was 502 IU/L and serum lipase 2248 IU/L. His kidney function tests were normal. Peripheral blood smear stained with Leishman demonstrated ring-form trophozoites of *P. falciparum* with high parasitemia (62%) with rapid antigen test for *P. falciparum* was also positive. Chest X-ray was normal. Contrast enhanced Computed Tomography (CECT) scan of the abdomen showing a bulky pancreas with peri-pancreatic fat stranding suggestive of acute pancreatitis (Figure 1). A diagnosis of severe falciparum malaria with acute pancreatitis was made on basis of investigation. Treatment in form



Figure 1: Contrast Enhanced Computed Tomography (CECT) scan of the abdomen showing a bulky pancreas with peri-pancreatic fat stranding suggestive of acute pancreatitis.

of intravenous quinine 1000 mg as loading dose in 10% dextrose solution followed by 500 mg in same solution and was monitored for hypoglycemia (Figure 1).

A Ryle's tube was inserted for bowel rest and acute pancreatitis was managed with analgesics, hydration and supportive therapy. Antibiotic ceftriaxone 1 g twice a day was started and marked improvements in his clinical symptoms was noted, parasitemia load were cleared by more than half after 4th day of therapy, his fever spike came down pain abdomen and jaundice subsided after 8th day. Oral quinine tab was started 8th day up to 15th day. The patient was discharged on 18th day and his condition was good on further follow up. Although abdominal pain is a frequent symptom in malaria, in this patient, the persistent and worsening abdominal pain was related to severe pancreatitis a rare complication of falciparum malaria. Abdominal pain and vomiting were reported to be more commonly associated with falciparum infections than with vivax [1]. The various causes of abdominal pain in malaria include Acalculous cholecystitis, acute surgical abdomen, splenic rupture, splenic infarction, splenic torsion and hepatitis/hepatomegaly, acute renal failure and algid malaria [2,3]. The presence of abdominal pain if associated with hepatomegaly has higher mortality in malaria. National Vector Borne Disease Control Programme reported about two million malaria cases every year, among them 50% is *P. falciparum*. The WHO estimates 100 million cases in the Southeast Asia Region [4]. Morbidity and mortality in rural population highest with varying degrees of presentation. Only few case reports are available regarding this complication of falciparum malaria [5-8]. Pancreatitis is an unusual cause of abdominal pain in malaria with less than 10 cases reported worldwide. The parasitemia in three of these patients were low (0.5% to 1.5%). As none of these patients had fulfilled WHO criteria for severe falciparum malaria. There is no acute respiratory distress syndrome either with severe malaria or with acute pancreatitis in this young man. The parasitized erythrocytes bind to receptors on the endothelial cells by the formation of knobs (electron-dense structures) and cause obstruction of capillary blood flow. Capillary blockage by parasitized RBCs and acute hemolysis was pathophysiological mechanisms that result in pancreatitis. Occlusion of blood vessels of the pancreas with parasitized RBCs and rosettes have been demonstrated in several autopsy studies. Jaundice is probably because of oedema of the head of the pancreas which compressed the intrapancreatic portion of the common bile duct. No correlation between the infestation index and the occurrence of pancreatitis has been established.

Encouraging results with successful outcome were seen in patients with acute pancreatitis in previous study and in present patient leads to early diagnosis of pancreatitis in *P. falciparum* positive cases and management in early stage so as to prevent the life-threatening complication of acute pancreatitis. Incidence of Acute pancreatitis is much higher than that being reported because of failure to report the patient presenting with acute abdomen in malaria patient. In tropical countries it is of utmost importance to look for signs and symptoms of acute pancreatitis in a patient presenting with pain abdomen in *P. falciparum* positive cases. So, a conclusion is drawn from this case and earlier cases that as falciparum malaria is common in tropical countries and it can present as pain abdomen with acute pancreatitis so routine serology testing is done to rule out the diagnosis to prevent dreaded complications and to decrease the mortality and mortality.

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