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Falciparum malaria presenting as acute pancreatitis

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Falciparum malaria is a protozoan disease caused by *Plasmodium falciparum* transmitted by the bite of infected *Anopheles* mosquitoes. This is a very common disorder in the tropics associated with myriad complications. We are presenting here acute pancreatitis as a sole presentation in a 35-yr old tribal man infected with falciparum malaria with high parasitemia. Acute pancreatitis is a well-known complication of falciparum malaria but it was rare because it was only presentation even after high parasitemia without any other system involvement. Only few reports are available regarding this.

Case report: A 35-yr old tribal male, labourer by occupation, presented to the medicine emergency department with a 2-day history of fever which was associated with chills, vomiting and generalized abdominal pain. There was no significant past medical history of biliary tract disease, lipid disorders, use of steroids and pain killers, nor was there a history of substance abuse.

On clinical examination at the time of presentation, he was alert, febrile (102°F) with mild jaundice. He became drowsy but arousable two days after admission. His blood pressure was normal. Systemic examinations were within normal limits except epigastric tenderness and mild generalised abdominal distension, with normal bowel sounds. Laboratory examination revealed haemoglobin 7.6 g/dL, total leucocyte count 5800/cmm (with granulocytes 68.6%), platelet count 70,000 and haematocrit 22.2%. His kidney function tests were normal. In liver function tests, serum bilirubin was 4.7 mg/dL, aspartate amino transferase 364 IU/L, alanine amino transferase 150 IU/L, alkaline phosphatase 71 IU/L

and serum albumin 2.6 mg/dL with total protein of 6 mg/dL. His serum amylase was 472 and serum lipase 2460 IU/L. A leishman-stained peripheral blood smear demonstrated ring-form trophozoites of *P. falciparum* with high parasitemia (60%). His rapid antigen test for *P. falciparum* was also positive. His chest X-ray was normal while abdominal X-ray showed mild generalised ileus without air-fluid levels. Ultrasound abdomen demonstrated an oedematous, enlarged pancreas with minimal fluid in peritoneal cavity (Fig. 1).

A diagnosis of severe falciparum malaria with acute pancreatitis was made and treatment was initiated with intravenous quinine 1000 mg as loading dose in 10% dextrose solution followed by 500 mg in same solution. He was regularly monitored for hypoglycemia. In view of high parasitemia and severe anaemia



Fig. 1: Ultrasound showing enlarged pancreas with minimal fluid in peritoneal cavity

at presentation, two units of packed red cells were administered to the patient. There were marked improvements in his clinical symptoms. Acute pancreatitis was managed with analgesics, hydration and supportive therapy. A ryle's tube was inserted for bowel rest and antibiotic as ceftriaxone 1 g twice a day was started. His parasitemia were cleared by more than half after 3rd day of intravenous quinine, his fever spike came down pain abdomen and jaundice subsided after 8th day. He was then switched over to oral quinine tab for total duration of one week. He was doing well on follow-up.

About two million malaria cases are being reported every year by the National Vector Borne Disease Control Programme, among them 50% is *P. falciparum*, the killer parasite. The WHO estimates 100 million cases in the Southeast Asia Region, 70% of these occur in India¹. It is responsible for major morbidity and mortality in rural population with varying degrees of presentation. It is said that malaria can present like anything except pregnancy. It is important for the clinicians in tropical countries to be alert for the symptoms and signs which may progress to life-threatening disease of falciparum malaria. Although abdominal pain is a frequent symptom in malaria, in our patient, abdominal pain and vomiting was related to severe pancreatitis. There are many causes of abdominal pain in malaria like acalculous cholecystitis, acute surgical abdomen, splenic rupture, splenic infarction, and hepatitis/hepatomegaly².

Acute pancreatitis as the sole presentation of falciparum malaria is very rare. At least some of the current major medical text books like Harrison's text book of medicine, Davidson, Cecil, and Oxford text book of medicine does not include *P. falciparum* as an aetiological agent of pancreatitis. Only few case reports are available regarding this complication of falciparum malaria²⁻⁵. Probable mechanism of pan-

creatitis in this malaria is capillary blockage by parasitized RBCs and acute haemolysis. Autopsy studies have demonstrated that the small blood vessels of the pancreas were packed with parasitized red cells and rosettes. 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. Jaundice in this patient is probably due to edema of the head of the pancreas which may have compressed the intrapancreatic portion of the common bile duct. The most encouraging part is successful outcome of our patient.

In conclusion, falciparum malaria is very common in tropical countries and presents like anything, and acute pancreatitis must be ruled out by doing routine serology like amylase or lipase because of its dreadful course, hence, mortality. Our case report suggested that falciparum malaria should be included in differential diagnosis of acute pancreatitis presenting with fever especially in endemic countries.

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