J Vector Borne Dis 48, September 2011, pp. 177–179

Case Reports

Falciparum malaria complicated with acute pancreatitis: a report of case series

M.K. Mohapatra & M.P. Gupta

Post Graduate Department of Medicine, V.S.S. Medical College and Hospital, Burla, Sambalpur, Orissa

Key words Clinical manifestation; falciparum malaria; India

Clinical manifestations of malaria cover a wide range of symptoms. Although most infected individuals will only have a relatively benign febrile illness, 1–3 million deaths per year occur due to severe malaria world wide[1]. This comprises severe syndromes such as cerebral malaria, severe anaemia, jaundice, renal failure, acute respiratory disstress syndrome etc. either alone or in combination[2]. Out of 4 species of *Plasmodium* that cause human malaria, *Plasmodium falciparum* cases have the potentiality of developing all these complications and almost all deaths have been ascribed to falciparum malaria[1]. Though during the course of the disease, it affects different organ systems of the body causing various complications, little is known about pancreatitis. Hence, acute pancreatitis has been described as a rare complication of falciparum malaria[3]. In view of rarity, we describe a series of 3 cases of falciparum malaria with acute pancreatitis.

Case report 1: A 45-year old male from endemic area of malaria was admitted to male Medicine Ward with main complaints of fever for Days 4, pain in the abdomen, vomiting, and oliguria for 2 days, and disorientation for 1 day. The fever was intermittent in nature and was associated with chills and rigor. Two days after fever, he experienced pain in epigastric region of sever intensity without any relation to food, relieved slightly on bending forward without any radiation. Abdominal pain was associated with vomiting. Subsequently, patient developed oliguria and disorientation. Patient was not a known patient of diabetes mellitus, hypertension, sickle-cell disease and was not addicted to alcohol and tobacco in any form.

On examination, patient was febrile with temperature of 101° F. There was jaundice and pallor. Tenderness could not be assessed properly due to unconsciousness. Liver was enlarged 2 cm below right costal margin, soft and non tender. Spleen was not palpable.

Investigations: Hb–6 g/dl; total leukocyte count–12,000/mm^3; differential count: N–89%, L–9%, E–2%; total platelet count–1,70,000/mm^3; Urine–Normal; QBC for malaria was positive for falciparum malaria. Peripheral blood smear was stained with Giemsa stain and the parasitic count was 8000/µl. Biochemical investigations showed: fasting blood sugar–98 mg/dl; serum bilirubin-direct 1.8 mg/dl, total–3.3 mg/dl; serum glutamate oxaloacetetate transaminase (SGOT)–30.8 IU/L; serum glutamate pyruvate transaminase (SGPT)–42.5 IU/L, serum alkaline phosphatase–52.4 IU/L; serum amylase-1200.5 IU/L; serum lipase 608.6 IU/L; blood urea–75.8 mg/dl; serum creatinine–4.6 mg/dl; and serum calcium–6.8 mg/dl. HPLC was done to exclude sickle-cell disease. Ultrasonography of abdomen showed swelling of head of pancreas with peripancreatic swelling. There was no gallstone and features of cholecystitis. CT scan of abdomen with contrast showed heterogenous contrast enhancement of head of pancreas with peripancreatic edema suggestive of acute pancreatitis (Fig. 1).

He was diagnosed as a case of falciparum malaria complicated with jaundice, renal failure, and pancreatitis and was treated with injection artesunate, injection imipenem, intravenous fluids, and other supportive measures. With treatment he became afebrile after 72 h, para-

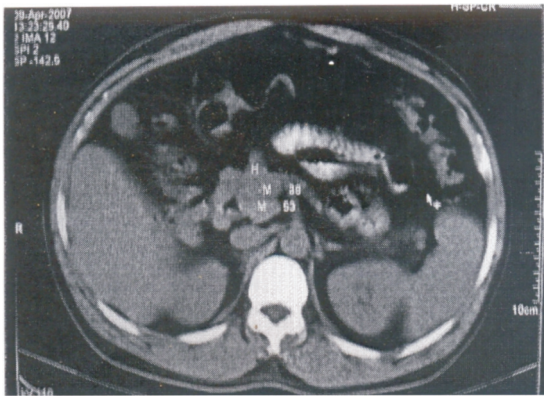

Fig. 1: CT scan of abdomen with contrast showing heterogenous enhancement of pancreatitis head

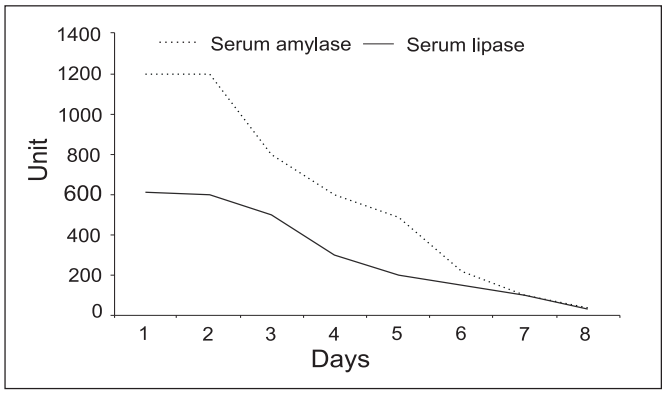

Fig. 2: Resolution of pancreatitis

sitic count became 0 after 96 h. Renal failure improved with conservative management and renal functions returned to normal on Day 6. Bilirubin was normalized by Day 5. S. lipase and amylase returned to normal by Day 8 (Fig. 2). Patient was discharged on Day 10 and followed up for another 15 days without any abnormality.

Case report 2: A 28-year old female from an endemic area of malaria was admitted with chief complaints of fever, vomiting and pain in upper abdomen for 2 days. Fever was high grade, continuous, and not associated with chills and rigor. It was accompanied by pain in upper abdomen. The pain was dull aching, radiating to back, and associated with vomiting. It was not relieved by food or any posture. For fever she had taken 2 tablets of paracetamol. She was afebrile at the time of admission (98° F). There was no anemia and jaundice. Abdominal examination revealed tenderness in the epigastrium and there was no hepato-splenomegaly. Pulse rate was 110/min and blood pressure was 110/80 mm of Hg. Investigation showed: Hb–10.2 g/dl; total leukocyte count–18,000/mm^3; differential count: N–86%, L–10%, E–4%; total platelet count–1,75,000/mm^3; urine was normal. Peripheral blood smear was negative for malaria parasite. Hb electrophoresis and HPLC was done to exclude sickle-cell disease and were normal. Biochemical investigations showed FBS-98 mg/dl; serum bilirubin–1.8 mg/dl; SGOT–30.8 IU/L ; SGPT–42.5 IU/L, serum alkaline phosphatase–52.4 IU/L; serum amylase–2200.5 IU/L; serum lipase 860.6 IU/L; blood urea–24.8 mg/dl; serum creatinine–1.8 mg/dl and serum calcium–6 mg/dl. Ultrasonography of abdomen showed swelling of head of pancreas. There was no gallstone and features of cholecystitis. She was diagnosed as a case of acute pancreatitis. She was treated with i.v. fluids, injection piperacillin and tazobactam, and injection metoclopromide for vomiting.

With treatment she did not improve. She became febrile and became drowsy after 36 h of treatment. Repeat investigations revealed no major changes in biochemical investigations. Giemsa stained peripheral blood smear showed asexual form of *P. falciparum* with a count of 9200/µl. CT scan of abdomen with contrast showed heterogenous contrast enhancement of head of pancreas with peripancreatic edema suggestive of acute pancreatitis. She was treated with injection artesunate, intravenous fluids, and other supportive measures. The condition of the patient improved. She was discharged on Day 7.

Case report 3: A 38-year old male from endemic area of malaria was admitted to Surgery Ward for fever for 2 days, and pain in upper abdomen for 1 day. Fever was continuous and was varying from 101 to 103° F. Abdominal pain was severe and localized to epigastric region without any radiation. There was only one episode of vomiting. There was no relation to food. He was not an alcoholic. Investigations in the emergency department showed: Hb–8 g/dl, total leukocyte count–16,000/mm^3; differential count: N–82%, L–14%, E–4%; total platelet count–2,20,000/mm^3; FBS–102.8 mg/dl; serum bilirubin–1.4 mg/dl; SGOT–30 IU/L ; SGPT–41.7 IU/L, serum alkaline phosphatase–62.6 IU/L; serum amylase–2050 IU/L; serum lipase–960.8 IU/L; blood urea–25.8 mg/dl; serum creatinine–1.6 mg/dl and serum calcium–6.8 mg/dl. Ultrasonography of abdomen showed swelling of head of pancreas. He was diagnosed as a case of acute pancreatitis and was treated with injection imipenem and cilastatine, intravenous fluids, injection octreotide, and other supportive measures. In spite of the treatment, patient did not improve. He became drowsy after 48 h of treatment and unconscious on the next day. He was still febrile. A physician call had been given and he was transferred to Medicine Ward. The repeat investigations did not show any gross change. EDTA blood was sent for QBC and ICT test and peripheral blood smear was drawn for malaria parasite. Falciparum malaria was positive in all the three tests and the parasitic count was 6200/mm^3. ABG analysis revealed metabolic acidosis with pH–7, pCo_2–21, pO_2–86 mm Hg, bicarbonate–5 mEq/L, and O_2 saturation–88%. CT scan of abdomen showed peripancreatic inflammation. He was diagnosed a case of falciparum malaria with acute pancreatitis and treated with artesunate in addition to the treatment as advised in Surgery Ward. He started improving after 24 h. By Day 7 all the biochemical investigations returned to normal. He was discharged on Day 11 of admission to Medicine Ward.

Falciparum malaria is known to cause one or several organ complications. But pancreatitis is a less described complication of falciparum malaria and only less than 10 cases have been reported[4-8]. All the reported cases were

associated with multiple organ dysfunctions like ARDS, haemolysis, pleural effusion etc. In 1907, in a review of 105 cases of pancreatitis the author could found one patient of pancreatitis was caused by falciparum malaria[4]. In 1977, a 26-year old service man presented with falciparum malaria, abdominal pain, pleural effusion and high amylase with upper gastrointestinal X-ray series consistent with acute pancreatitis[5]. Patients of malaria with ARDS and haemolysis complicated with pancreatitis had been reported[6]. Also, 3 cases of malaria with pancreatitis have been reported with fever, abdominal pain, and vomiting without multi organ failure[7,8]. In the present series, we have 2 cases with multiple organ dysfunctions and one case (Case report 2) without multiple complications and 2 patients had heavy parasitaemia where as one had low parasitic count. Relation between parasitic count and occurrence of pancreatitis could not be established, as parasitemia varies from 0.1 to 60% in the reported cases[6].

Whenever pancreatitis has been described as a complication of falciparum malaria, always a query arises whether it is a mere association or there is a causal relation[6-8]. This has been further aggravated when such cases have been reported from endemic areas. It is argued that a significant proportion of the population from endemic areas may be asymptomatic carriers of the parasite; hence, the detection of malaria parasite in any disease condition may be a mere association[1]. But once malaria parasite has been detected in a hospitalized patient one has to administer antimalarial irrespective of the malaria endemicity of the area and the associated disease[1]. Hence, it is difficult to determine the causal relation. However, autopsy study in severe falciparum malaria showed haemorrhage in pancreatic parenchyma similar to acute pancreatitis of other aetiologies[6]. Therefore, falciparum malaria as a cause of pancreatitis cannot be ruled out.

In this case series, we describe 3 patients of falciparum malaria from endemic areas complicated with acute pancreatitis. As there was no evidence to implicate drug or toxin induced injury or an obstructive cause, it is reasonable to think that pancreatitis was secondary to malaria. However, exclusion of limited etiological factors of acute pancreatitis does not necessarily conclude that the cause of pancreatitis was malaria. Diagnosis of acute pancreatitis is based on the presence of abdominal pain and biochemical evidence of pancreatic injury. The elevation of serum amylase more than 3 times of the normal has been considered useful for the diagnosis. However, serum lipase has a higher sensitivity and specificity and has supplanted amylase for the diagnosis of acute pancreatitis[3]. CT scan is a reliable and non-invasive imaging modality to evaluate the pancreas[3]. In addition to antimalarial drugs, correction of dehydration and electrolyte imbalance, bowel rest, and prophylactic antibiotic should apply to patients of malarial pancreatitis. The prognosis is good.

The most possible mechanism of pancreatitis in malaria is the microvascular occlusion with resultant ischaemia, activation of pancreatic enzymes, and injury to the pancreatic tissue[6-9]. Apart from the capillary blockade, acute hemolysis in falciparum malaria, may induce acute pancreatitis.

In conclusion, malaria pancreatitis, though rare, is usually associated with multiple organ involvement. In patients of malaria with abdominal pain, pancreatitis should be suspected. The prognosis is good in cases of malarial pancreatitis.

REFERENCES

1. Severe falciparum malaria. *Trans R Soc Trop Med Hyg* 2000; *94* (Suppl 1): 1–90.
2. Mohapatra MK, Das SP. The malaria severity score: a method for severity assessment and risk prediction of hospital mortality for falciparum malaria in adults. *J Assoc Phys India* 2009; *57:* 119–26.
3. Steinberg WM. Acute pancreatitis. In: Feldman M, Freidman LS, Brandt LJ, editors. Sleisenger & Fordtran's *Gastrointestinal and liver disease,* VIII edn. Elsevier, India 2007; *1:* 1241–69.
4. Egdagl A. A review of 105 reported cases of acute pancreatitis, with special reference to aetiology: with report of two cases. *Johns Hopkins Med J* 1907; *18:* 130–2.
5. Johnson RC, DeFord JW, Carlton PK. Pancreatitis complicating falciparum malaria. *Postgrad Med J* 1977; *61:* 181–3.
6. Gurman G, Schlaeffer F, Alkan M, Heilig I. Adult respiratory distress syndrome and pancreatitis as complications of falciparum malaria. *Crit Care Med* 1988; *16:* 205–6.
7. Sarma PS, Kumar RS. Abdominal pain in a patient with falciparum malaria. *Postgrad Med J* 1998: *74:* 425–7.
8. Desai DC, Gupta T, Sirsat RA, Shete M. Malarial pancreatitis: report of two cases and review of the literature. *Am J Gastro* 2001; *96:* 930–2.
9. Kumar S, Jain AP, Vikas. Falciparum malaria presenting as acute pancreatitis. *J Vector Borne Dis* 2010; *47:* 121–2.

Correspondence to: Dr M.K. Mohapatra, Professor of Medicine Qr. No. C-1, Medical Campus, V.S.S. Medical College & Hospital, Burla, Sambalpur–768 017, Orissa.
E-mail: mohapatra.manoj@rediffmail.com

Received: 25 January 2011 *Accepted in revised form:* 20 June 2011