

CASE REPORT

Acute Pancreatitis in a Patient with Vivax Malaria

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ABSTRACT

Context Acute pancreatitis is most commonly linked to gallstone disease or alcohol consumption. Occasionally it can follow infectious disease. Malaria, especially *Plasmodium falciparum* infection, has been associated with acute pancreatitis. **Case report** We present the case of a 17-year-old male who presented with a history of fever, abdominal pain and hypotension and revealing acute pancreatitis associated with infection by *Plasmodium vivax*. **Conclusion** Acute pancreatitis can accompany malaria, including *Plasmodium vivax*.

INTRODUCTION

Many infections including parasites like Toxoplasma, Cryptosporidium, Ascaris, Clonorchis have been implicated in acute pancreatitis. Falciparum malaria associated pancreatitis have been reported but, to date, no case of *Plasmodium vivax* related pancreatitis has been reported [1]. We report a case of fatal pancreatitis in a 17-year-old male who had evidence of *Plasmodium vivax* infection.

CASE REPORT

A 17-year-old male presented with history of severe diffuse abdominal pain lasting for two day. The patient had an episode of fever two days back, associated with chills and rigors but no rash. The patient also had multiple episodes of vomiting. The patient had no history of similar episodes of abdominal pain in the past. There was no significant medical or surgical past and no history of any addictions. The general condition of the patient had worsened over hours and he presented in a state of shock. On examination the pulse was feeble with a rate of 124/min, the blood pressure was 86/42 mmHg, the respiratory rate was 32/min and the patient was afebrile. The patient was conscious and oriented. Clinical examination revealed tenderness to palpation in upper abdomen, no guarding, rigidity or organomegaly. The investigations revealed hemoglobin 13.4 g/dL (reference range: 12-16 g/dL), total leukocyte count 12,200/mm³ (reference range: 4,000-

11,000/mm³), detailed leukocyte count: neutrophils 82% (reference range: 40-70%), lymphocytes 17% (reference range: 20-50%) and eosinophils 1% (reference range: 0-5%), and platelet count 34,000/mm³ (reference range: 150,000-450,000/mm³). Blood urea was 86 mg/dL (reference range: 14-40 mg/dL), serum creatinine 3.1 mg/dL (reference range: 0.5-1.2 mg/dL), serum Na⁺ 143 mEq/L (reference range: 135-145 mEq/L), K⁺ 4.1 mEq/L (reference range: 3.5-5.0 mEq/L), serum Ca²⁺ 7.6 mg/dL (reference range: 8.5-10.2 mg/dL) and blood glucose was 72 mg/dL (reference range: 70-110 mg/dL). Serum bilirubin was 4.2 mg/dL (reference range: 0.3-1.3 mg/dL) with direct 3.1 mg/dL (reference range: 0.1-0.4 mg/dL), transaminases were SGPT 56 U/L and SGOT 68 U/L (reference range: 8-40 U/L and 10-38 U/L, respectively), alkaline phosphatase was 148 U/L (reference range: 13-100 U/L) and serum albumin was 2.7 g/dL (reference range: 3.5-5.5 g/dL). The serum amylase was 1,234 U/L (reference range: 10-200 U/L). The diagnosis of acute pancreatitis was made. Chest X-ray and electrocardiography were normal. Urgent ultrasonography of abdomen revealed a bulky pancreas, no gallstones, ascites or splenomegaly was seen. The report of peripheral smear which became available later revealed presence of *Plasmodium vivax*. The antigen testing including parasite lactate dehydrogenase (LDH) for vivax and falciparum were done. Parasite LDH for vivax was positive. The hypotension was managed with intravenous fluids and ionotropes (dopamine and norepinephrine); however, the patient died due to refractory shock around 24 hours after admission.

DISCUSSION

Abdominal pain in malaria can result acute surgical abdominal complications like splenic rupture, splenic

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infarction, splenic torsion, hepatomegaly, hepatitis, acute acalculous cholecystitis and occasionally acute pancreatitis [1]. The reports of pancreatitis as a complication of malaria are sparse, with less than 20 cases to be associated with *Plasmodium falciparum* [1, 2, 3, 4, 5]. The pathophysiology of pancreatitis in falciparum infections is believed to be the linked to multiorgan failure or to capillary thrombosis by parasited red blood cells [5, 6]. Another hypothesis is hemolysis secondary to falciparum infection. It has been noted that pancreatitis in falciparum malaria does not depend on parasitemia and is generally mild with good prognosis [2, 5].

Till recently falciparum malaria has been considered as a malignant form of malaria while vivax has been thought to be a benign form of malaria. It was believed that the phenomenon of sequestration and cytoadherence are not a feature of vivax malaria. However, recent reports have found evidence of these phenomenon in *Plasmodium vivax* infections [7, 8]. Few reports have implicated *Plasmodium vivax* infection as a cause of severe malaria [9].

The present report is significant on two accounts. This is the first report of acute pancreatitis in a patient with vivax malaria. Second, although vivax is considered as a less severe form of malaria, the patient died. However, our patient had hypotension which itself can result in ischemic pancreatitis. To conclude plasmodium vivax can cause severe manifestations including acute pancreatitis. The clinicians working in

endemic areas should consider pancreatitis as a possible complication of vivax malaria.

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