

CASE REPORT

Plasmodium Vivax Malaria: An Unusual Cause of Acute Pancreatitis

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ABSTRACT

Plasmodium vivax malaria traditionally thought to be benign, nowadays is often diagnosed in patients with a myriad of systemic complications like cerebral malaria, acute respiratory distress syndrome, acute kidney injury, acute pancreatitis, hepatic dysfunction, disseminated intravascular coagulation, etc which were earlier attributed only to plasmodium falciparum. Hence timely intervention and expert care should be undertaken for vivax patients so that the mortality and morbidity associated with this form of malaria can be reduced.

INTRODUCTION

Malaria being one of the most important parasitic diseases of humans, is a very common cause of febrile illness in tropical countries. Till now complications were more commonly attributed to infection with *Plasmodium falciparum* and less commonly with *Plasmodium vivax*. Association of acute pancreatitis with parasites like Toxoplasma, Cryptosporidium, Ascaris lumbricoides and Clonorchis sinensis has been previously reported [1, 2] but rarely with *P. vivax*. We report a case of acute pancreatitis in a 23-year-old male who was diagnosed with *Plasmodium vivax* infection, was treated conservatively and discharged after 10 days of hospitalization in a stable condition.

CASE REPORT

A previously healthy 23 year old male, presented to the emergency with chief complaints of high grade fever with chills and rigors but no rash for the last 3 days. There was no history of cough, breathlessness, pain abdomen, vomiting, altered sensorium or burning micturition. There was no history of any addiction or previous hospitalization. On examination, he was conscious, oriented, febrile with temperature of 102.4°F, blood pressure of 112/78 mm Hg, pulse rate of 102/min low in volume, regular, respiratory rate 18/min with no pallor, icterus, cyanosis or clubbing. Respiratory, cardiovascular and neurological system examination were normal. Blood gas analysis was also normal. The investigations revealed hemoglobin 11.4 g/dL (reference range: 12-16 g/dL), total leukocyte count 12,600/mm³ (reference range: 4,000-11,000/mm³), detailed leukocyte count: neutrophils 77%

(reference range: 40-56%), lymphocytes 17% (reference range: 20-40%) and eosinophils 1% (reference range: 0-5%), and platelet count 88,000/mm³ (reference range: 150,000-450,000/mm³). Blood urea was 52mg/dL (reference range: 14-50 mg/dL), serum creatinine 1.5 mg/dL (reference range: 0.5-1.4 mg/dL), serum Na⁺ 141 mEq/L (reference range: 135-145 mEq/L), K⁺ 4.4 mEq/L (reference range: 3.5-5.0 mEq/L), serum Ca²⁺ 7.9 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 1.2 mg/dL (reference range: 0.3-1.3 mg/dL) with direct 0.7 mg/dL (reference range: 0.1-0.4 mg/dL), transaminases were SGPT 48 U/L and SGOT 54 U/L (reference range: 8-40 U/L and 10-38 U/L, respectively), alkaline phosphatase was 132U/L (reference range: 13-100 U/L), serum triglyceride 142 mg/dL (reference range: 70-140 mg/dL) and serum albumin was 2.9 g/dL (reference range: 3.5-5.5 g/dL). G6PD assay was normal. Chest X-ray and electrocardiography were normal.

Malarial parasite quantitative buffy coat (MPQBC) test was performed in the emergency which was positive for *P. vivax*. Subsequently, peripheral blood smear thin film examination also revealed schizonts of *P. vivax*. Typhidot was negative for salmonella typhi. Patient was started on artemesin based combination therapy (ACT), antipyretics and intravenous fluids. On 3rd day of hospital stay, he complained acute onset pain in abdomen localized to epigastrium and umbilical region. On examination, abdomen was soft, but tenderness was present in the epigastrium. bowels sounds were 4-5/min, no free fluid or organomegaly was noted. An erect x-ray abdomen was done which was normal. The serum amylase was 955 U/L (reference range: 10-200 U/L) and serum lipase was 288 U/L (reference range: 10-80 U/L). Ultrasonography of abdomen revealed a bulky pancreas, no gallstones, ascites or splenomegaly was seen. A contrast-enhanced computed tomography (CT) scan of the abdomen was done which was suggestive of diffuse pancreatitis with modified CT severity index of 6 as shown in Figure 1. A diagnosis of *P. vivax* associated acute pancreatitis was made. Patient was

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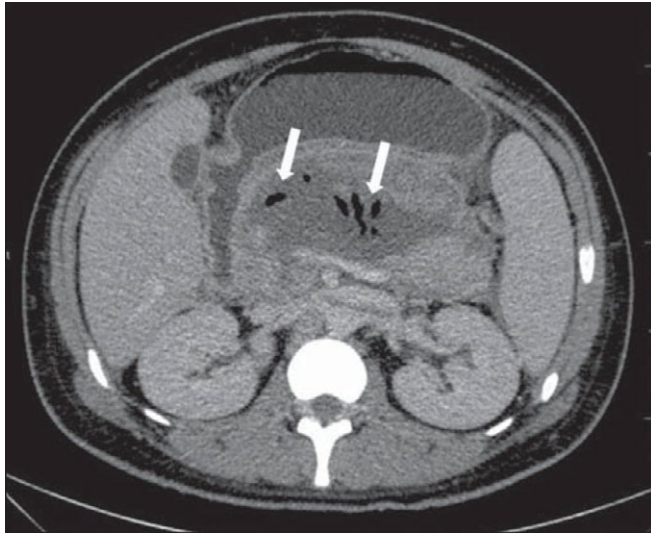


Figure 1. Infected acute necrotizing pancreatitis. Contrast enhanced axial CT image, venous phase showing liquefied area in the pancreatic body, compatible with necrosis, with gas inside (arrows) without an outlined fluid gas level, but intermingled with the fluid, indicating the presence of thick fluid/pus content suggestive of infection.

managed conservatively with intravenous fluids, analgesics and imipenem-cilastin 500mg thrice daily which was continued for 7 days along with artemesin combination therapy for 7 days. Patient recovered completely and was discharged on 10th day of admission in a stable condition with 14 days of primaquine therapy for radical cure of *P. vivax*.

DISCUSSION

Malaria is a major public health problem in India, accounting for sizeable morbidity and mortality. Benign tertian malaria, the term commonly implicated with *P. vivax* mono-infection is now in oblivion [3, 4, 5, 6]. Recent studies have shown that 11% of severe malaria are because of *P. vivax* monoinfection [7]. However pancreatitis as a complication of *P. vivax* infection is extremely rare [4]. Though the exact pathogenesis of pancreatitis is still not clear it can be hypothesised that it may be due to ongoing pancreatic injury due to systemic infestation or direct invasion of the pancreatic tissue by *P. vivax* through mechanisms of cytoadherence and sequestration [8, 9]. It has also been proposed that acute pancreatitis may be due to multi organ failure or capillary thrombosis caused by parasitized red blood corpuscles [10, 11].

In our case the patient did not have typical presenting signs and symptoms of acute pancreatitis on day 1 of admission. There was no evidence of alcoholism, gall stones, any structural anomaly of the pancreas, drugs, trauma, or any metabolic abnormality leading to pancreatitis in our patient and hence diagnosis of malaria induced pancreatitis was justified. Values of serum amylase and lipase (serum lipase is a more specific marker) three times the upper limit of normal

along with contrast enhanced CT (CECT) abdomen suggestive of pancreatic inflammation, peripancreatic fluid collection or pancreatic necrosis are diagnostic of acute pancreatitis. In our case on CECT abdomen there was no evidence of any gallstones or any ascites but there was peripancreatic fluid collection and pancreatic necrosis <30 % with a modified CT severity [12] of 6 (moderate acute pancreatitis).

Conclusion

This report highlights the current changing spectrum of *P. vivax* infection which was earlier not considered to be malignant. Patients diagnosed as a case of *P. vivax* malaria should be watched carefully, as the complications may not be apparent at the time of presentation always. As pain abdomen in such patients can rarely be either due to gastrointestinal haemorrhage [13] or acute pancreatitis, a meticulous clinical workup can be life saving.

Conflicting Interest

The authors had no conflicts of interest.

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