International Research Journal of Medical Sciences _____ ISSN 2320 –7353
Vol. **2(10),** 26-28, October **(2014)** *Int. Res. J. Medical Sci.*

Mini Review Paper

An uncommon Presentation of severe Falciparum Malaria: Acute Pancreatitis

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Available online at: www.isca.in, www.isca.me
Received 30th August 2014, revised 22nd September 2014, accepted 16th October 2014

Abstract

In tropical countries malaria is a well known cause of fever. Plasmodium falciparum infection can manifest a variety of complications ranging from self-limiting and mild to fatal condition. Acute Pancreatitis is one of the rare complication of falciparum malaria. We hereby report a case of Plasmodium falciparum malaria who developed acute pancreatitis. Patient responded well to anti malarial drug with supportive management.

Keywords: *Falciparum*, malaria, acute abdomen, acute pancreatitis.

Introduction

Plasmodium falciparum malaria is a protozoan disease which is transmitted by the bite of infected *Anopheles* mosquito. Malaria is distributed throughout the tropical countries of the world. *Plasmodium falciparum* predominates in Africa, New Guinea, and Hispaniola, while in the Indian subcontinent the prevalence of two species *Plasmodium falciparum* and *Plasmodium vivax* is almost equal. In sever *Plasmodium falciparum* malaria the risk of mortality increases sharply when there is involvement of vital-organ dysfunction or infected erythrocytes >2% (a level corresponding to $>10^{12}$ parasites in an adult)[1].

The common causes of Pancreatitis are Gallstones, Alcohol, Hypertriglyceridemia, Endoscopic retrograde cholangiopancreatography (ERCP), Trauma, Sphincter of Oddi dysfunction, Postoperative and Drugs while infections are rare causes which include mumps, *coxsackievirus, cytomegalovirus, echovirus* and parasites[2]. There are many parasites such as *Toxoplasma, Cryptosporidium, Ascaris, Clonorchis* are known to be associated with acute pancreatitis[3]. Only few cases of falciparum malaria are reported in literature that complicated to acute pancreatitis[4,5,6,7]. Therefore it is very important to make early diagnosis and treat accordingly to decrease the morbidity and mortality in malarial endemic regions.

Case Report: A 24 years old male student, resident of lucknow was presented in our emergency department with complaints of high grade fever associated with chills and rigors, vomiting and generalized abdominal pain for five days. On the day of admission he developed severe upper abdominal pain mainly located in the epigastrium and periumbilical region which was radiating to the back and chest. Pain was worsening with duration. He had no prior history of jaundice, trauma, diabetes mellitus, hypertension, drug intake, alcoholism or any similar episodes of abdominal pain in past. On general examination patient was conscious and well oriented. He had no pallor and icterus. He was febrile (103°F). His pulse rate -104/minute, hypovolumic and regular, Blood Pressure-94/60mmHg and respiratory rate-22/min were recorded. Systemic examination revealed tenderness on palpation in epigastric and right hypochondrial region with mild splenomegaly without any guarding and rigidity. Cardiovascular, Nervous and Respiratory systems were within normal limits. Investigations showed hemoglobin(Hb)-13.0 g/dl , total leucocyte count- 11600/mm^3, diffential leukocyte count: neutrophils 80%, lymphocytes 15%, monocyte-4%, eosinophils 1% and platelet count 16,0000/mm3. Biochemical investigations showed random blood sugar level-112mg/dl, serum Na+ 136 meq/L, serum K+ 4.8meq/L, blood urea 26mg/dl, serum creatinine 1.1mg/dl and serum Ca++ 9.3mg/dl. His liver function test revealed serum bilirubin-0.3mg/dl, serum aspartate aminotransferase(AST)-39U/L, alanine aminotransferase (ALT)-23U/L, serum alkaline phosphatase-40U/L, serum protein-6.8g/dl and serum albumin-3.7g/dl. The level of serum amylase and lipase were 2800U/L and 1200U/L respectively. Chest X-Ray and electrocardiography did not show any abnormality. Abdominal ultrasonography demonstrated heterogenous and bulky pancreas, mild splenomegaly with minimal fluid in peritoneal cavity suggestive of acute pancreatitis. There were no gallstones and features of cholecystitis figure-1. Patient was kept nil per oral and managed with analgesics, hydration, intravenous broad spectrum antibiotics and supportive management. Even though patient was deteriorating. Therefore we suspected malaria as a differential diagnosis because patient was native of endemic region, having high grade fever with chills and rigor associated

with splenomegaly. Thick and thin leishman-stained peripheral blood smears were examined which showed ring-form stage of *Plasmodium falciparum* with high parasitemia (60%). Immunochromatographic test (ICT) for *Plasmodium falciparum* was also found positive. The final diagnosis of acute pancreatitis due to *Plasmodium falciparum* was made and intravenous antimalarial drug artesunate was added to previous treatment. Patient was started improving day by day and got discharged on 12th day of admission.

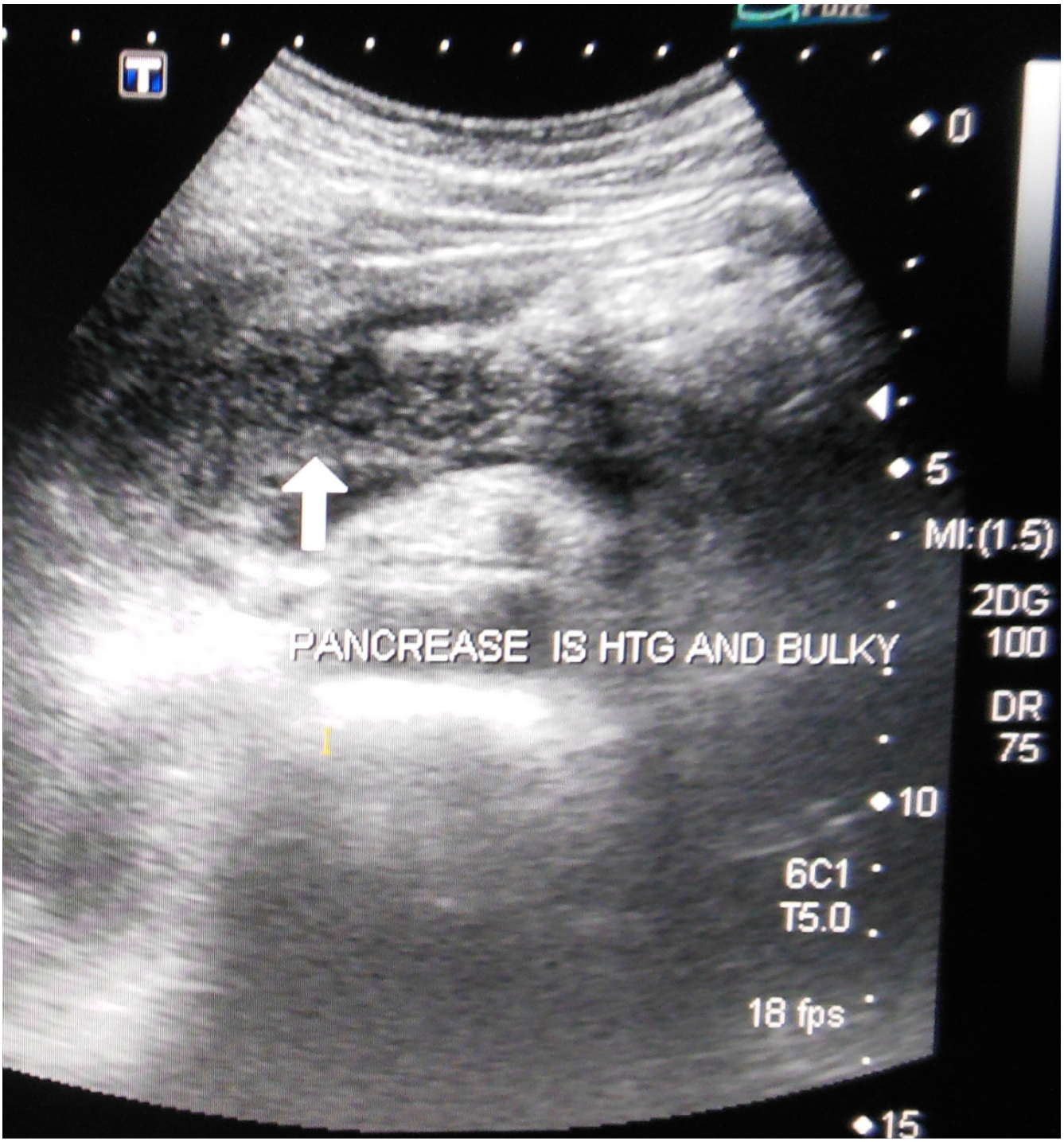

Figure-1
Abdominal ultrasonography demonstrated heterogenous and bulky pancreas

Results and Discussion

The major manifestations of severe falciparum malaria are unarousable coma/cerebral malaria, acidemia/acidosis, severe normochromic normocytic anemia, extreme weakness, bleeding, disseminated intravascular coagulation, hemoglobinuria, hypoglycemia, hypotention/shock, renal failure, pulmonary edema/adult respiratory distress syndrome, convulsions, impaired consciousness/arousable, jaundice and hyperparasitemia which indicates a poor prognosis[1]. Although abdominal pain is a clinical feature in malaria but due to worsening of abdominal pain we suspected acute pancreatitis which later on confirmed by investigations. Abdominal pain in malaria may be due to hepatomegaly, hepatitis, acute acalculous cholecystitis, splenic infarction, splenic rupture, splenic torsion and occasionally acute pancreatitis[4]. *Plasmodium falciparum* causes accumulation of parasitized erythrocytes causing thrombosis and infarcts commonly in the small vessels of spleen, liver, bone marrow and brain. Less commonly affected organs include small intestine, pancreas, heart and lungs. Parasitized erythrocytes bind to receptors on the surface of endothelial cells by the formation of knobs which lead to obstruction of capillary blood flow. This pathophysiology results to ischemia, activation of pancreatic enzymes and injury to pancreatic tissue[8-11]. We are reporting this case because the patient recovered completely after addition of antimalarial therapy which also supports our final diagnosis of malarial pancreatitis.

Conclusion

Acute pancreatitis should be kept in mind as a complication in all the patients of malaria complaining of severe, constant pain located in the epigastrium and periumbilical region radiating to the back. Our case report highlights the potential danger of Plasmodium falciparum malaria so treating physician must be aware of the development of pancreatitis in malaria because malarial pancreatitis has good prognosis. Early diagnosis and management of sever *Plasmodium falciparum* malaria can prevent life threatening complications and results in better outcome especially in endemic countries.

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