Case Report

Spontaneous splenic rupture in Typhomalaria: A case report with review of literature

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ABSTRACT

Plasmodium vivax, which was previously considered 'benign', has come out with unusual severe symptoms. We, here present an unusual case of Plasmodium vivax malaria presenting with spontaneous splenic rupture with hemoperitoneum, renal and pulmonary dysfunction. Following conservative management, there was an uneventful recovery, and the patient was discharged in a good general condition.

Keywords:

Typhomalaria; Splenic Rupture; Vivax Malaria.

INTRODUCTION

Malaria and typhoid fever share many social and environmental factors, which predispose to these infections however they are caused by widely different etiological agents. Both are major public health problems in tropical and subtropical regions [1]. Spontaneous rupture of typhomalarial spleen is rare and requires high degree of clinical suspicion to arrive at a diagnosis. Ultrasonography and Computed Tomography (CT) are essential to make the diagnosis of splenic injury and grade it [2]. To the best of our knowledge, there is no case report describing spontaneous splenic rupture in malaria-typhoid co-infection (typhomalaria). We hereby report a case of typhomalaria complicated by spontaneous splenic rupture and renal-pulmonary dysfunction.

CASE PRESENTATION

A nineteen-year-old male presented to the emergency department with history of intermittent high grade fever for the last three days. The fever was associated with chills and rigors. This was followed by abdominal pain, distension, dyspnea, bilateral chest pain and repeated episodes of vomiting for the last 1 day. There was no history of spontaneous bleeding neither there was any history of similar episode in the past.

At presentation he had a pulse of 130 per minute, respiratory rate of 26 per minute, and hypotension (98/70 mm). There was also bilateral diminished air entry in the chest. On examination, abdomen was distended with tenderness in left hypochondrium. Spleen was palpable 2 cm below the left costal margin and liver was palpable 3 cm below the right costal margin. On investigations his haemoglobin was 5.5 gm% with normal total leukocyte count (7,000 per cu mm). The blood urea was 63.9 mg/dl and serum creatinine was 1.6 mg% (mildly elevated) suggesting minimal renal dysfunction. A peripheral blood smear showed Schizonts of Plasmodium vivax (Figure 1). Typhoid IgG and IgM were also positive.

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Blood sample was sent for culture, which turned out to be positive.

Ultrasonography revealed a large heterogeneous layered appearance along the superior aspect of spleen with free fluid in the abdomen suggestive of splenic rupture with perisplenic hematoma and hemoperitoneum. A non-contrast computed tomography scan (contrast scan was not done due to renal dysfunction) also revealed multiple hyperdense (HU 50-60) and hypodense region along the diaphragmatic surface of spleen with a large perisplenic hematoma and hemoperitoneum (Figure 2). Bilateral pleural effusion was also seen.

Surgery reference was sought and conservative management was started with monitoring of vitals, laboratory parameters and clinical signs. An abdominal drain was inserted which drained about 500 ml of non-clotting blood. As there was no G6PD deficiency, patient was started on artemisinin combination therapy followed by 14 days course of primaquine. Broad spectrum antibiotic ceftriaxone was also started 2gm 12 hourly. He was transfused blood and blood products. Subsequently his renal parameters improved within next one week. His general condition gradually started improving and finally made an uneventful recovery. He is on regular follow up and a repeat ultrasound after two months showed almost complete resolution of the hematoma.

Considering the immunological role of spleen in preventing pneumococcal and malarial infection, non-operative management with splenic conservation is applied in hemodynamically stable patients of traumatic and spontaneous splenic rupture. As our patient was young and became hemodynamically stable with conservative management, splenectomy was not performed. Splenectomy should be reserved for patients with uncontrolled bleeding and haemodynamic instability.

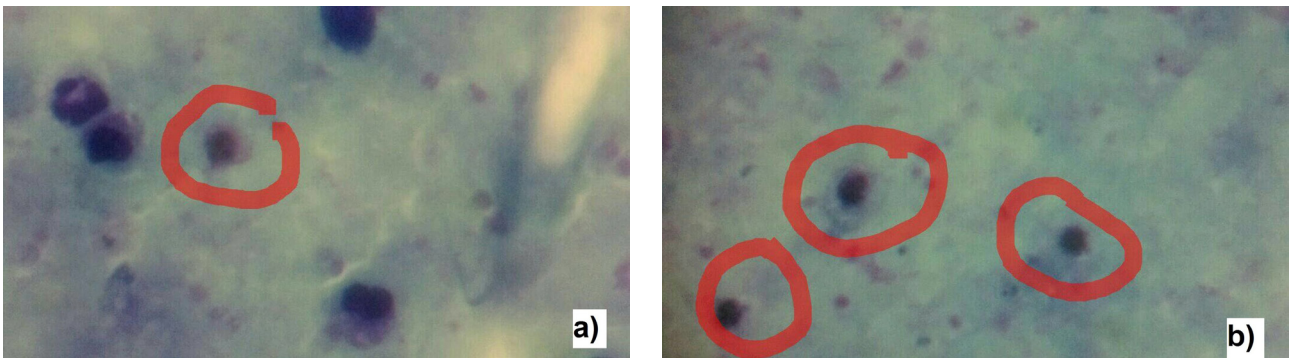
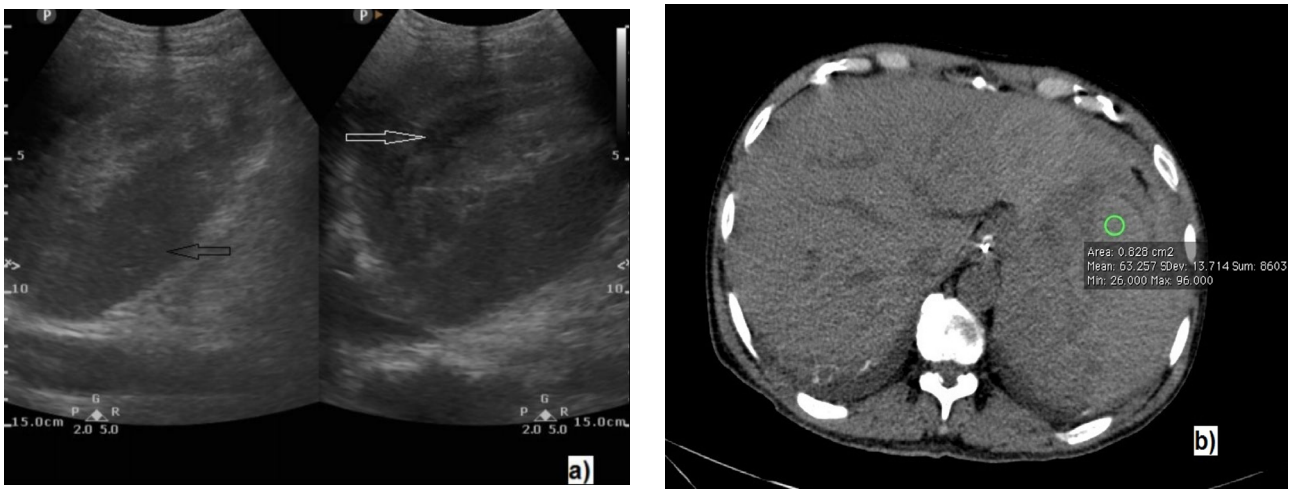
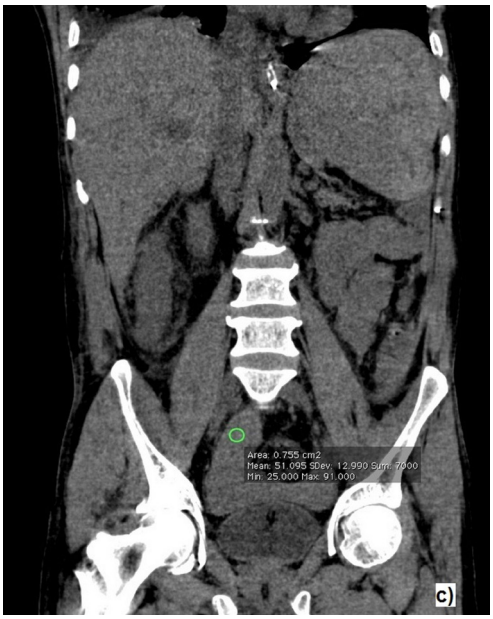


Figure 1 a & b - Peripheral blood smears showing Schizonts of Plasmodium vivax (under red circles).





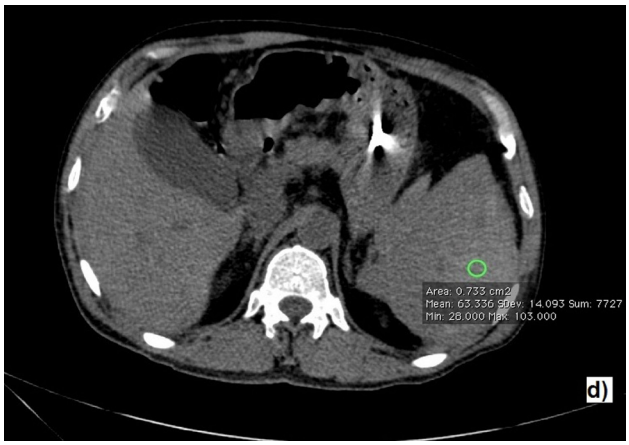


Figure 2 a - Ultrasound abdomen showing a large heterogeneous layered appearance along the superior aspect of spleen (open white arrow) suggestive of splenic rupture with hematoma formation. Part of normal spleen is also seen (open black arrow), 2b, c & d - Non-contrast computed tomography scan also revealed multiple hyperdense (HU 60-70) and hypodense region along the diagphragmatic surface of spleen suggestive of a large perisplenic hematoma and hemoperitoneum.

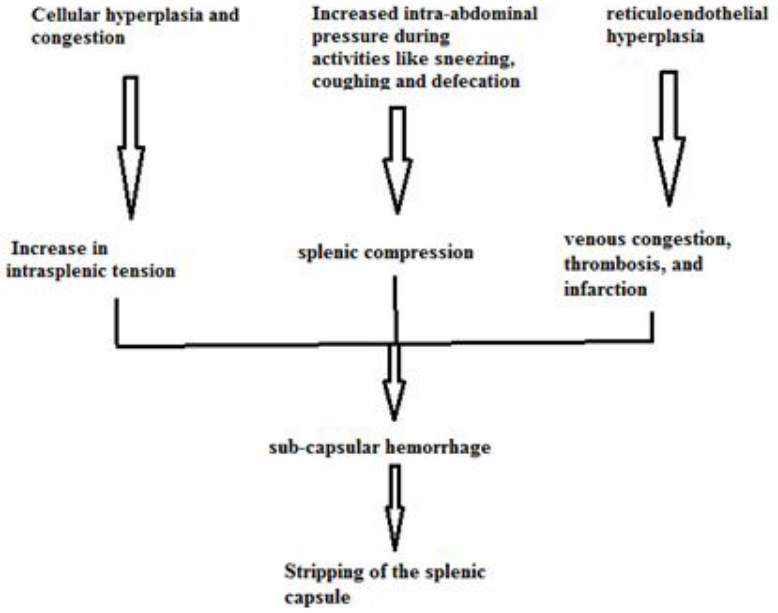


Figure 3 - Flow chart showing mechanism of spontaneous splenic rupture in malaria.

DISCUSSION

An army doctor, Woodward first described Typhomalaria in 1862 among young soldiers during the American Civil War who were suffering from febrile illness that seemed to have features of both typhoid and malaria [3].

Malaria and typhoid fever although caused by widely different etiological agents, share many social and environmental factors, which predispose to these infections. Both are major public health problems in tropical and subtropical regions. There is similarity in the clinical features of both infections, which can lead to misdiagnosis of linking signs and symptoms to single pathology instead of co-infection. Therefore a high index of suspicion is required to prevent complications and misuse of drugs [1].

There are few postulated mechanisms, which predispose to typhomalaria. One of them is reduced antibody response to Salmonella typhi during acute episodes of malaria [4]. Hemolysis, which occurs in malaria, may also predispose to infection with gram-negative organism such as Salmonella typhi [5].

Plasmodium (P) falciparum is notorious for causing severe and fatal malaria. But recently P. vivax, which was previously considered 'benign', has come out with unusual symptoms and even deaths. In endemic regions, development of immunity and resistance to anti-malarial drugs leads to atypical manifestations with P vivax monoinfection. Due to lack of awareness of these atypical manifestations as well as co-infection with typhoid can lead to severe complications like splenic rupture, renal and pulmonary dysfunction as in our case [6].

Splenic complications reported in malaria include hypersplenism, abscess or hematoma formation, hyper-reactive malarial syndrome and very rarely (reported in up to 2% of cases) splenic rupture [7]. Most cases of splenic rupture occur during acute primary attack. Chronically enlarged spleens are less prone to rupture. Even appropriate anti-malarial prophylaxis and treatment may not prevent this complication. Spontaneous splenic rupture is more common with P. vivax than P. falciparum malaria [8].

Although the exact mechanism of splenic rupture in malaria is not clear, the mechanisms have been suggested in the flow chart (Figure 3) [9].

Orloff and Peskin recommended few diagnostic criteria for labeling a case as spontaneous splenic rupture: (i) absence of any history of trauma; (ii) absence of any pre-existing splenic disease; (iii) absence of adhesions or scarring in the spleen; and (iv) presence of grossly normal spleen, macroscopically and microscopically [10].

Cross sectional imaging, mostly Computed Tomography (CT) is essential to make the diagnosis of splenic injury and grade it. The most common features on CT are splenomegaly with splenic lacerations, peri-splenic hematoma and hemoperitoneum [2].

Eun Mi Kim et al performed a retrospective study to identify abdominal CT findings in malaria. Malaria group included 34 of 405 patients who had a positive peripheral blood smear for *Plasmodium vivax* and had underwent abdominal CT. Control group included 80 patients who had fever and a negative peripheral blood smear whereas 120 healthy people formed normal group. The incidence of splenomegaly, splenic focal low attenuation, and spontaneous splenic rupture were much higher in the malaria group ($P < 0.05$). Although their study showed a high rate of splenic rupture or subcapsular hematoma (3 cases, 8.8%), spontaneous rupture was seen in only one case (2.9%) of malaria [11].

The main deciding factor for management options (conservative v/s splenectomy) is the hemodynamic status of the patient. Low grade injuries (I and II) can be managed conservatively. Even patients with high grade injuries (III and IV) having stable hemodynamic factors can be assigned conservative management and reserving surgery only if hemodynamic instability occurs [12].

CONCLUSION

Spontaneous splenic rupture of typhomalarial spleen is rare and requires high degree of clinical suspicion to arrive at a diagnosis. Most of these cases, like traumatic splenic injuries, can now be managed non-operatively. Splenic salvage should be the aim in the management of these patients.

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