Research Paper

Volume : 4 | Issue : 6 | June 2015 • ISSN No 2277 - 8179

A Rare Cause of Splenic Infarction: Plasmodium Vivax Malaria

Medical Science

KEYWORDS : splenic infarct, plasmodium vivax.

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ABSTRACT

Splenic infarct is a rare form of wedge-shaped pathology. It is either segmental or global. It occurs as a result of arterial or venous compromise, and is associated with a heterogeneous group of diseases. Spleen is supplied by splenic artery (branch of coeliac artery) and the short gastric artery (branch of left gastroepiploic artery). Within the spleen, arterial supply is segmental, occlusion of secondary branches results in a wedge-shaped infarct. Plasmodium vivax malaria accounts for a very rare cause of splenic infarcts, eventhough splenic rupture is described. Here we report a case of plasmodium vivax malaria with splenic infarction. Our patient responded well to the conservative treatment offered and was under follow up.

Introduction:

Splenic infarction is a rarely described, and probably underdiagnosed, complication of malaria. Bonnard et al found only eight documented cases of splenic infarction associated with malaria in the literature. The splenic complications of acute malaria include two different prognostic and treatment entities: splenic infarction and splenic rupture. The most frequent causes of splenic infarction include myelofibrosis and haematologic malignant neoplasms[1]. Other causative factors include thromboembolic disease stemming from atrial fibrillation[2],systemic lupus erythematosus complicated by the presence of lupus anticoagulant and anticardiolipin antibodies[3], falciparum malaria[4], septic emboli in endocarditis[5], sickle cell disease[6], and Wegener's granulomatosis[7].

Case report :

A previously healthy 35 year old agricultural labourer, presented with complaints of high grade fever for the past 1 week. Fever was associated with chills, rigors. No complaints of burning micturtion or rash. No complaints of vomiting or loose stools. No h/o any past surgeries. On examination he was moderately built and ill nourished. He was pale with no icterus, cyanosis, clubbing or adenopathy. Cardiovascular and respiratory system was unremarkable. On palpating the abdomen mild splenomegaly was noted extending 1.5 cm below left costal margin and tenderness present on deep palpation in left hypochondrium.

On the day of admission his platelet count was 76000/mm3 and rapid check for malaria antigen was negative. Blood for cultures was drawn and empirical treatment was started. Peripheral smear examination revealed plasmodium vivax schizonts. Antimalarials were added to the treatment. Other investigation profile of the patient is presented below under [Table 1].

On day 2 of admission patient complained of abdominal pain, an ultrasound abdomen revealed splenomegaly and infarction in the spleen. Contrast enhanced CT abdomen revealed multiple splenic infarcts with splenomegaly(fig 1). Investigative protocol for the splenic infarction in lines of 2d echocardiogram, autoimmune profile, hematological studies for abnormal hemoglobin, coagulation profile were performed and all proved to be normal. Cultures of blood and urine were negative. Left with none other than vivax malaria we attributed this as the cause of splenic infarction.

This patient was treated with antimalarials , antipyretics and analgesics, hematinics and he improved well with the treatment. He was under follow up now with improved symptomatology and radiological resolution.

Investigation	Result
Hemoglobin	7.5 gm%
Total count	7400/mm3
Platelet count	76000/mm3
Peripheral smear	Moderate degree of microcytic hypochromic anemia Plasmodium vivax schizonts are noted in the smear.
Reticulocyte count	2%
Renal function tests	Blood urea – 22mg% Serum creatinine – 1.0 mg%
Liver function tests	Serum bilirubin- 3.2 mg% (decreased to 1.2mg% with antimalarials treatment)
Serum electrolytes	Normal
Viral screening	Negative
Widal test	Negative
Blood and urine culture	Sterile
2 D Echo	Normal study (no evidence of any vegetations)
Autoimmune profile	Negative
Coagulation profile	Normal
Hemoglobin electrophoresis	Normal
USG abdomen	Splenomegaly with splenic infarcts
CECT abdomen	Multiple splenic infarcts with moderate splenomegaly

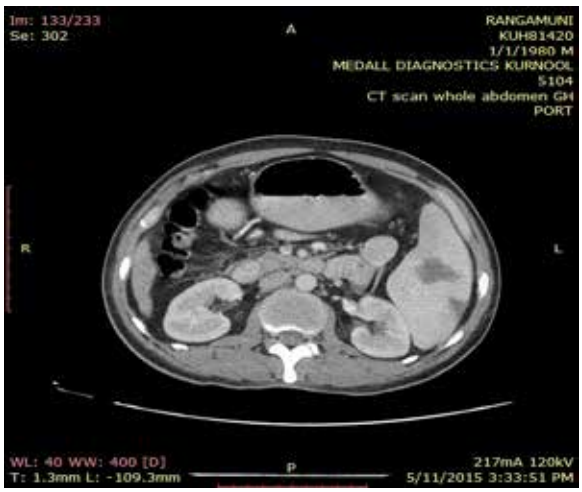

Fig 1: CECT abdomen showing splenic infarcts (wedge shaped) with splenomegaly.

Discussion :

Spleen is supplied by splenic artery (branch of coeliac artery) and the short gastric artery (branch of left gastroepiploic artery). Within the spleen, arterial supply is segmental, occlusion of secondary branches results in a wedge-shaped infarct. Splenic infarction is described but a rare complication of malaria,usually seen with falciparum species[8]. It is very rare to see a splenic infarction in plasmodium vivax.

Etiologies for splenic infarction are as follows

Haematological disorders	**Infections**
Sickle haemoglobinopathies	Meningococcaemia
Polycythaemia vera	Kala-azar (visceral leishmaniasis)
Hypercoagulable state	Infectious mononucleosis
Idiopathic venous thrombosis	Clostridium perfringens
Leukaemia	Campylobacter fetus
Lymphomas	Salmonellosis
Myelofibrosis	Plasmodium vivax
Erythropoietin therapy	Plasmodium falciparum
Emboic disorders	Leptospirosis
Atrial fibrillation	
Endocarditis	**Trauma**
Prosthetic mitral valve	Torsion of wandering spleen
HIV-associated mycobacterial infections	Sclerotherapy of bleeding gastric varices
Paradoxical emboli from right heart	Embolisation of splenic bleed
Infected thoracic aortic graft	Left heart catheterisation via femoral artery approach
Left ventricular mural thrombus	
Autoimmune/collagen vascular diseases	**Miscellaneous**
SLE	Amyloidosis
Ka-wasaki disease	Sarcoidosis
Wegener's granulomatosis	Splenic vein thrombosis
Polyarteritis nodosa	ARDS
Operative aetiologies	
Liver transplant	

In the spleen, the mechanism of infarct is not exactly known . The pathophysiology of splenic infarction in falciparum malaria is thought to be due to sequestration of red cells containing the mature forms of parasites ,rouleaux formation by erythrocytes, infective embolism, low oxygen tension, and out-stripping of massive splenomegaly of its available blood supply. Sequestration occurs in venules of the vital organs predominantly in the white matter of brain, heart, eyes, liver, kidney, adipose tissue and least in skin.. Spleen acts as a principal host defense against malarial parasite by destroying and filtering both uninfected and parasitized red cells. However in plasmodium vivax the pathology is attributed to the tissue avascularity with hypoxia due to the splenomegaly and increased stickiness of parasitized RBC [9]. Splenic enlargement occurs in 95-100% of patients with malaria. Alteration in structure and function of spleen cause splenomegaly. Other complications described in vivax malaria are splenic abscess, rupture, hemorrhage, torsion of spleen [10].

The course of disease was spontaneously favourable, suggesting a relative benignity of this complication. Conservative therapy proves to be effective in many cases and treating the underlying etiology favours a good prognosis. Our patient responded well with the conservative measures and treating underlying cause i.e., malaria.

Conclusion:

Splenic infarction is a rare cause of the abdominal pain in a patient and often overlooked. Prompt evaluation and supportive care proves better results in patient with splenic infarcts. Underlying etiology should be searched and treated for complete resolution of the disease.

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