Splenic Infarction in Malaria

Lt Col AN Prasad*, Lt Col A Singh⁺

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Introduction

Splenic infarction is a rare clinical entity that has received increasing clinical attention in recent years. The aetiology is multifactorial including myelofibrosis, haematologic malignant neoplasms, thromboembolic disease due to atrial fibrillation, rheumatologic disorders, falciparum malaria, adult respiratory distress syndrome, rupture of splenic artery aneurysm, septic emboli in infective endocarditis, sickle cell disease and Wegener granulomatosis [1]. In these conditions, splenic ischemia is induced either suddenly with massive occlusion or vascular discontinuity, or gradually with infiltration of the splenic parenchyma by haematopoietic elements and strangulation of its blood supply.

We describe the case of an elderly female patient in whom splenic infarction occurred during antimalarial treatment for initially uncomplicated acute malaria. Management was restricted to close clinical monitoring and analgesia. We found eight such cases in the literature [2,3]. Clinical outcome was favourable in all the cases.

Case Report

A 57 year old woman presented with sudden onset of left upper abdominal pain which increased following food intake. She had been experiencing fever, chills and nausea since five days. She denied history of dysuria, urgency or frequency. Bowel movements were normal. She had been taking chloroquine for two days. The patient had no significant past/ personal history. On admission her blood pressure was 129/86 mm Hg, pulse 102 beats/minute, respiratory rate 16/minute and temperature 39°C. Physical examination revealed left upper quadrant tenderness in abdomen. There was mild tenderness on percussion of the left flank. Bowel sounds were normal. There was no rebound tenderness or guarding. Liver was palpable three centimeter below subcostal margin and non-tender. There was tender splenomegaly, two centimeter below the left subcostal margin.

On investigations her haemoglobin was 9.8 gm/dl, total leucocyte count 10,600/mm³ (polymorphs 72%, lymphocytes 18%, monocytes 4%, eosinophils 4%, and basophils 2%),

erythrocyte sedimentation rate (ESR) - 32 mm fall in 1st hour, platelet count 1.87 lacs/mm³, blood urea nitrogen 36 mg/dl and serum creatinine of 0.8 mg/dl. Urine examination showed albumin, pus cells 2-3/HPF with no casts or red blood cells. Blood culture, widal test, liver function tests, stool (routine and for occult blood), blood sugar and lipid profile were normal. Peripheral blood smear was negative for malarial parasite. However, serological test for malaria (Standard Diagnostics Bioline rapid immunochromatographic test, with sensitivity of 91.3 % and specificity of 98 %) was strongly positive for both vivax and falciparum malaria.

Ultrasonographic evaluation of the abdomen revealed multiple wedge shaped hypo-echoic areas in the spleen, suggestive of infarction. Other diagnostic possibilities included haematoma, abscess and neoplasm. Kidney was normal and there was no evidence of ascites. Contrast enhanced computed tomography of abdomen revealed multiple wedge-shaped hypodense non enhancing lesions consistent with splenic infarction.

She was treated with intravenous quinine, analgesia, fluids and other supportive measures. During the next 10 days, the patient required steadily reducing doses of oral analgesics. She remained afebrile and recovered completely. Ultrasonography of abdomen was repeated after four weeks, which showed complete resolution of the infarcted areas.

Discussion

Spleen ranks next to the kidney in the frequency with which embolism and infarction occurs. It occurs as a result of arterial or venous compromise and infective endocarditis is the commonest cause. It can occur as a rare presentation of malaria [4]. This complication can occur despite appropriate antimalarial prophylaxis and treatment, as seen in our case. There are no known predictive signs. Clinicians should be aware that left hypochondrial pain occurring during treatment for acute malaria could be due to splenic infarction [5].

The clinical presentation of infarction may vary. They may be discovered incidentally on radiological or postmortem studies, or hemorrhagic shock as a result

*Classified Specialist (Paediatrics), 166 Military Hospital, C/o 56 APO. ⁺Graded Specialist, (Radiology), Military Hospital, CTC, Pune.

Fig. 1 : Ultrasonography of upper abdomen showing a hypoechoic area (wedge shaped) in spleen.

of sub capsular haematoma with rupture into the peritoneal cavity. When splenic infarction occurs secondary to septic emboli in infective endocarditis, upto 90% of patients have no localizing findings. The radiographic characteristics of splenic infarction with either ultrasonography [6] or computed tomography (CT) are well defined, though CT has emerged as the preferred imaging modality for making the diagnosis [7]. When the infarcts are small, the prognosis is usually favourable. The wedge-shaped infarct has its apex towards the hilum and its base beneath the capsule. It is either segmental or global. Spleen is supplied by splenic artery and the short gastric artery. The arterial supply is segmental hence occlusion of secondary branches results in a wedge-shaped infarct [8].

Splenic infarction is a rare complication of malaria which can occur inspite of appropriate antimalarial prophylaxis and treatment.

Conflicts of Interest

None identified

Fig. 2 : Two conical wedge shaped hypo dense lesions seen in CECT scan of upper abdomen, which are non enhancing.

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Events Calendar

07th-08th December 2007

CME on Imaging and Intervention Radiology

Venue: Department of Radiodiagnosis and Imaging,
Armed Forces Medical College, Pune-411040

Contact Person: Col S Bhargava

Professor and Head

Department of Radiodiagnosis & Imaging

AFMC, Pune-411040

Tele No: 020-26306045 (O), 9370144729 (Mobile)

email : sbhargav2@gmail.com