

Splenic Infarct of Unknown Etiology - A Rare Presentation.

N. Nand, H. Kumar

Department of Medicine, Pt. B.D. Sharma University of Health Sciences, Rohtak, Haryana, India

Abstract: Splenic infarct is a rare disease entity, which occurs as a result of the occlusion of splenic vessels. Here, we report a 24 year young-female, who presented with fever and pain in abdomen of seven days.

INTRODUCTION

Splenic infarction refers to occlusion of the splenic vascular supply, leading to parenchymal ischemia and subsequent tissue necrosis. The infarct may be segmental, or it may be global, depending upon the site of occlusion. It may be due to a heterogeneous group of diseases, including infiltrative hematologic diseases, thromboembolic conditions, hypercoagulable states; infections and trauma.¹ We report here one case who had pyrexia of unknown origin (PUO), who responded to doxycycline.

CASE REPORT

A 24 years female presented with history of fever and pain in abdomen of seven day duration. Fever was high grade, intermittent and was associated with rigors and chills. She also complained of severe pain in the left upper quadrant of abdomen for the same duration. Pain was associated with multiple episodes of vomiting. There was no history of cough, expectoration, chest pain, yellowish discoloration of sclera, joint pain, decreased urine output and altered bowel habits. Patient was conscious, oriented and mildly anemic. Her pulse was 92/min, average volume, and her blood pressure was 120/84 mmHg. There was no icterus, cyanosis, pedal edema, skin rash and lymphadenopathy. Chest examination revealed bilateral vesicular breathing. Tender spleen was palpable 3 cm below left costal margin. It was firm in consistency. There was no splenic rub. First and second heart sounds were normal in intensity without any audible murmur. Central Nervous System examination was within normal limits. On the basis of history and physical examination, a provisional diagnosis of Pseudo-Typhoid secondary to malaria was made and patient was investigated on the lines of malaria. Complete hemogram showed hemoglobin of 7.5 g%, total leukocyte count (TLC) 7000/mm³ and differential leukocyte count (DLC) showed polymorphs 74, lymphocytes 22, monocytes 2 and eosinophils 2. Platelet count was 1.8lac/mm³. Peripheral blood film examination revealed increased rouleaux formation without any evidence of malarial parasite and sickle cell. Biochemical parameters showed serum bilirubin of 0.8 mg/dl, SGOT of 57IU/Lt, SGPT of 38IU/Lt, serum alkalinephosphatase of 231IU/Lt, prothrombin time of 18 seconds, blood urea of 22mg/dl and serum creatinine of 0.8mg/dl. Urine complete examination was essentially normal. Widal test was negative. Blood and urine culture were sterile. Tests for hypercoagulable state (protein C, protein S, anti-phospholipid antibodies, antithrombin- α_2 , factor-V Leiden mutation, hyperhomocysteinemia) were normal. Hemoglobin electrophoresis did not reveal sickle hemoglobin. Chest skiagram demonstrated a normal heart size. Electrocardiography showed sinus tachycardia. Ultrasonography of abdomen revealed liver size of 18 cm with normal echotexture. Spleen measured 14 cm in size with a hypochoic area of 3.9x2.2 cm. at its lower pole, suggestive of splenic infarcts. Portal vein diameter and splenic vein diameter were 11mm and 8mm, respectively. Gall bladder and bilateral kidneys were normal. Contrast enhanced computed tomography (CECT) scan of abdomen revealed multiple wedge shaped, non enhancing, hypodense lesions in the splenic parenchyma, suggestive of splenic infarcts. Patient was put on chloroquine in antimalarials doses, however she did not respond. She was then put on doxycycline. Patient responded to treatment and demonstrated considerable improvement and became afebrile after four days. Her pain in left upper quadrant persisted for ten more days, for which she was given non-steroidal anti inflammatory drugs. Now, Patient is on our routine follow-up and she is asymptomatic for last 3 month. Repeat USG abdomen after 3 month did not reveal any splenic infarct.

DISCUSSION

Splenic infarction refers to occlusion of the splenic vascular supply, leading to parenchymal ischemia and subsequent tissue necrosis. The infarct may be segmental, or it may be global, involving the entire organ. It is the result of arterial or venous compromise and is associated with a heterogeneous group of diseases. The etiologies of splenic infarct are malignant hematologic disorders (leukemia, lymphoma & myelofibrosis), benign hematologic disorders (hypercoagulable states - protein C or protein S deficiency, oral contraceptives, lupus anticoagulant, erythropoietin therapy, idiopathic venous thrombosis, polycythemia vera & sickle hemoglobinopathies), thromboembolic disorders (endocarditis, atrial fibrillation, prosthetic mitral valve, paradoxical emboli from right heart & left ventricular mural thrombus), autoimmune/collagen vascular disease, trauma, surgery and miscellaneous (splenic vein thrombosis, pancreatitis, amyloidosis, sarcoidosis,

pancreatic cancer, malaria, salmonella, adult respiratory distress syndrome & postpartum toxic shock syndrome). The vast majority (88%) of splenic infarcts, are because of either infiltrative hematologic diseases or thromboembolic conditions.²⁻⁵

The pathophysiology of splenic infarction in infiltrative hematologic diseases and myelofibrosis include the congestion of the splenic circulation by abnormal cells. Where as, in sickle cell disease, crystallization of the abnormal hemoglobin during periods of hypoxia or acidosis is responsible for it. In malignant hematologic diseases (eg, chronic myeloid leukemia), relative hypoxia as a result of increased splenic oxygen requirements secondary to an increased splenic mass, coupled with a decreased oxygen-carrying capacity secondary to the anemia of hypersplenism, leads to infarction.³

Thromboembolic events are also frequent cause of splenic infarcts. Splenic embolization may result from a variety of cardiovascular conditions, including a left atrial or ventricular mural thrombus that formed as a result of acute myocardial infarction or atrial fibrillation, or that developed from complications of cardiac catheterization or bacterial endocarditis. A report on 108 patients with left-sided endocarditis undergoing valvular surgery revealed a 19% incidence of splenic infarction; in almost half of the study's patients with infarction, the diagnosis was made incidentally on a computed tomography (CT) scan.⁵ In this patient, there was increased rouleaux formation on peripheral blood film. However, there was no evidence of hypercoagulable state on the basis of investigations.

The clinical spectrum of splenic infarct varies from asymptomatic infarction to hemorrhagic shock (secondary to massive subcapsular hemorrhage with free rupture into the peritoneal cavity). Approximately one third of splenic infarcts are clinically occult. The most common presenting symptom is left upper quadrant abdominal pain (70%). Additional symptoms include fever and chills, nausea and vomiting, pleuritic chest pain, and left shoulder pain (Kehr sign). Septic thromboemboli may result in splenic abscesses, which present with sepsis and left upper abdominal pain. Laboratory tests are not diagnostic for splenic infarction except mild leucocytosis, thrombocytosis and anaemia.² CECT scan is the current diagnostic modality of choice. Other modes of diagnosis include ultrasonography, magnetic resonance imaging (MRI) and radioisotope scan.⁶ Principal mainstay of medical therapy is analgesia with either narcotics or non-steroidal anti-inflammatory agents and close follow-up. The infarcted spleen can be left in situ, and the patient is observed, as there is sufficient collateral flow in spleen. Surgery is indicated only in the presence of complications, i.e., sepsis, haemorrhage, splenic abscesses, pseudocyst formation, and rupture of spleen.² Our patient responded to treatment with doxycycline. It is difficult to pinpoint whether it was antibacterial or antimalarials effect of doxycycline, which was responsible for the clinical improvement.

REFERENCES

- Antopolsky M, Hiller N, Salameh S, et al. Splenic infarction: 10 years of experience. *Am J Emerg Med.* Mar 2009; 27(3):262-5.
- Nores M, Phillips EH, Morgenstern L. The clinical spectrum of splenic infarction. *Am Surg* 1998; 64(2):182-8.
- Hayashi H, Beppu T, Okabe K, et al. Risk factors for complications after partial splenic embolization for liver cirrhosis. *Br J Surg.* Jun 2008; 95(6):744-50.
- Gupta BK, Sharma K, Nayak KC, et al. A case series of splenic infarction during acute malaria in northwest Rajasthan, India. *Trans R Soc Trop Med Hyg.* Jan 2010; 104(1):81-3.
- Ting W, Silverman NA, Arzouman DA, et al. Splenic septic emboli in endocarditis. *Circulation.* Nov 1990; 82(5 Suppl):IV105-9.
- Joshi SC, Pant I, Shukla AN, et al. Splenic infarct as a diagnostic pitfall in radiology. *J Cancer Res Ther.* Apr-Jun 2008; 4(2):99-101.