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Case Report of Splenic Infarct with Proximal Splenic Artery and Coeliac Trunk Thrombosis

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Abstract

A thrombus formation in the splenic artery occludes the vascular supply of the spleen, leading to ischemia of parenchyma of spleen and subsequently necrosis – Splenic Infarct. It is often clinically silent, the most common symptom being Pain Abdomen / LUQ (Left Upper Quadrant) pain and the sign being Left Hypochondrium Tenderness. There are a multitude of causes for splenic artery thrombosis and infarction, ranging from hematological disorders and malignancies to embolic disorders, vasculitis, autoimmune and collagen vascular diseases, trauma, systemic inflammatory disorders etc. As the presentation tends to mimic other diseases, a high degree of clinical suspicion is warranted for diagnosis. A contrast enhanced CT scan is the current diagnostic modality of choice. Splenic infarction alone is not an indication for surgery. Non-operative medical management requires close follow up and surgery is indicated for persistence of symptoms and / or complications.

We are reporting the case of a young male who presented to the emergency with 4 days of low grade fever and nausea with sudden onset severe pain in epigastrium and left hypochondrium. Normal lab investigations and USG abdomen were followed up with a CECT-abdomen that revealed splenic infarction and 90% stenosis of celiac trunk and hepatic artery with proximal splenic artery thrombosis.

Keywords: Splenic Infarct; Splenic Artery Thrombosis; Celiac Trunk Thrombosis.

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Introduction

A thrombus formation in the splenic artery occludes the vascular supply of spleen leading to ischemia of parenchyma of spleen and subsequently necrosis - Splenic Infarct. The infarction may involve the entire organ (global) or be localized to a segment. One of the earliest descriptions of splenic infarct was in 1896 in Germany where microscopic splenic infarcts were detected post-spleenectomy in a patient of endocarditis with septic emboli [1].

Splenic infarcts are most often clinically silent. In 1998, Nores and colleagues [2] reported 59 cases treated over a 30-year period at the University of California, Los Angeles (UCLA), and at the Cedars-Sinai Medical Center. In 1986, Jaroch and coauthors [3] identified 75 patients through clinical or autopsy reports at the Cleveland Clinic and found only an additional 77 cases in the literature. Most of the current literature consists of case reports only. However, there is a rising trend in the frequency of number of splenic infarcts identified due to increase in radiological imaging of patients, subsequently leading to an increase in incidental detection of splenic infarcts.

There are a multitude of causes for splenic infarction, majority (88%) comprising of infiltrative haematological diseases that result in congestion of splenic circulation by abnormal cells or obstruction of large vessels by thromboembolic events [2]. The causes may vary ranging from haematological disorders and malignancies to embolic disorders, vasculitis, autoimmune and collagen vascular diseases, trauma, systemic inflammatory disorders etc.

The spectrum of clinical presentation varies from asymptomatic infarction (discovered incidentally) to hemorrhagic shock. The most common symptom is Abdomen Pain in LUQ (Left Upper Quadrant) and the sign is tenderness in Left Hypochondrium. No lab investigations are specific for splenic infarct. Contrast enhanced CT scan is the diagnostic modality of choice [4]. A Gd-MRI clearly identifies area of infracted splenic parenchyma. Presence of luminal bowel gas and morbid obesity render this modality less useful. In a retrospective study of 49 episodes of acute splenic infarction, Antopolsky et al found ultrasonographic scanning to be diagnostically useful in only 18% of patients [5].

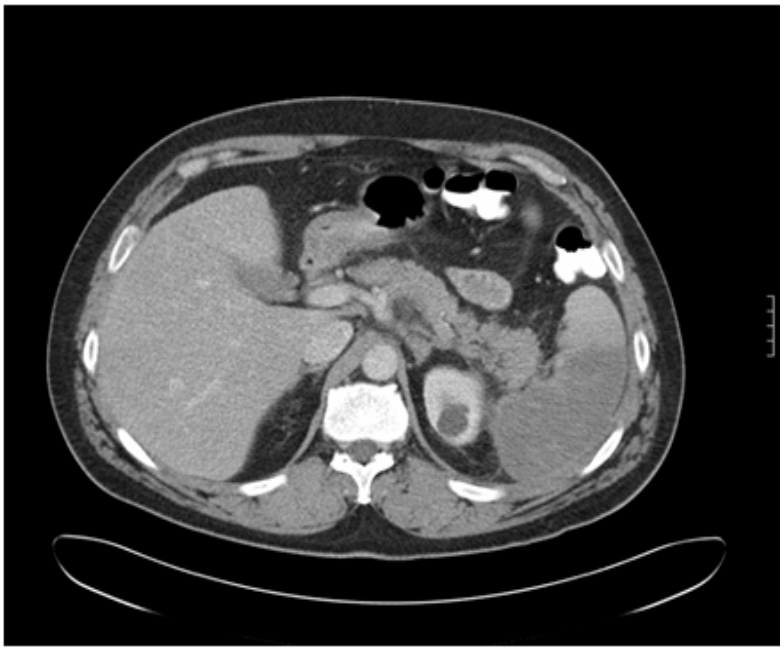
Splenic infarction alone is not an indication for surgery. Non-operative medical management requires close follow up. The mainstay of medical management comprises of adequate analgesia and close follow up. There is no scientifically supported information for the role of antiplatlet drugs and antibiotics. Surgery is indicated for persistence of symptoms and/or complications such as abscess, rupture, haemorrhage or pseudocyst. Because of the small but fatal risk for OPSI (overwhelming post-splenectomy infection), splenic salvage is preferred.

Case Report

A 45 yr old male presented to the ER with complaints of sudden onset very severe pain abdomen since morning (4-5hrs), mainly in the epigastrium and left hypochondrium, non-radiating, not associated with any aggravating or relieving factors, associated with fever, low grade, not associated with chills past 3-4 days and nausea and is not associated vomiting, loose motions, chest pain, syncope, sweating, SOB and cough. On arrival to the ER, patient is talking with no apparent respiratory distress and is haemodynamically stable, with P=86/min, BP=130/80, T= 98.4^{o}F (36.9^{o}C), RR=18/min, Spo2=99% on R, ARBS = 176 mg/dl. On systemic examination of the patient, there was tenderness present over the epigastrium and left hypochondrium with no palpable mass/ organomegaly and Bowel sounds were present, rest of the systemic examination was absolutely normal

Management in the Emergency department included intravenous cannulation, symptomactic pain medications and Investigations which were ECG,CBC, LFT, KFT, Blood cultures, S. Amylase, S. Lipase, PS for MP, Typhidot and USG whole abdomen was done which was absolutely normal. In view of persistence of pain abdomen in spite of adequate analgesia, associated nausea and a normal USG study without any GB or Hepatobiliary pathology, Acute Pancreatitis was suspected. Pt kept NPO, iv fluid, iv antibiotics, iv analgesics, iv antiemetics, iv antacids, supportive managementSurgical consultation was taken. The surgical specialist evaluated the patient and a CECT Whole Abdomen was planned.

Image 1: CECT abdomen showing hypodense area within the spleen—splenic infact



CECT Whole Abdomen (Fig. 1) Enlarged spleen with a Hypodense lesion within the splenic parenchyma– suggestive of Splenic Infarct. The splenic artery also does not enhance, suggestive of thrombosis. A cardiology consultation was taken for abdominal thrombus / Ischaemia. Patient was planned for a detailed 2D ECHO, CT Angiography, USG Abdominal Doppler (portocaval) and ANA. 2D- ECHO revealed LVEF 55% with no RWMA, Doppler – lower limb which was normal arterial and venous Doppler studies *Additional labs* included **C**-ANCA <6 u / ml (negative) P-ANCA <6 u / ml (negative) Phospholipids 207 mg / dL (151-264 mg / dL) Thrombin time 20 sec (16-23 sec) with no evidence of lupus anticoagulant and Cardiolipin antibodies normal. CT Angiography revealed 90% stenosis of Celiac trunk and Hepatic artery with proximal Splenic artery thrombosis. Patient was administered Inj. Clexane 0.6ml s / c stat and planned for DSA. The patient was Discharged on Request in stable condition on conservative management (anti platlets).

Discussion

Acute occlusion of the splenic artery results in infarction of the splenic parenchyma. As the spleen receives its blood supply from both splenic arteries (from celiac plexus) and short gastric arteries (from left gastroepiploic artery), occlusion of the main splenic artery may be compensated by collaterals that often preserve some or all of the splenic parenchyma. Within the spleen, the arterial supply is segmental. Occlusion of these secondary branches results in the classic wedge-shaped infarct.

Splenic infarction is often clinically silent and the presentation tends to mimic other diseases. Hence, a high degree of clinical suspicion is warranted for diagnosis. In patients presenting with left upper quadrant pain, fever, chills, nausea, vomiting, pleuritic chest pain and left shoulder pain, infarction of the spleen should be considered. Rapid diagnosis is the key to salvage the spleen. Multitude of causes need to be considered, especially, hematological diseases, thromboembolic states, vasculitis (SLE with lupus anticoagulant or antiphospholipid antibodies), cocaine abuse, trauma etc.

Not all cases require surgical intervention and the call for splenectomy should be taken judiciously, taking into consideration the lifelong risk of OPSI. Majority of cases are managed medically.

At one end of the spectrum of prognosis lies clinically occult splenic infarcts without any sequel while at the other end is high mortality associated with splenectomy. Asplenic individuals are at high risk of developing OPSI and require regular clinical follow up.

Although the incidence of splenic infarcts are rare but still quite prevalent and has been seen in adult populations. This case tells us that a simple case of pain abdomen which was initially thought to be a pancreatitis can turn out to be a case of splenic infarct and it carries along with it a whole set of complications. A high index of clinical suspicion has to be maintained for considering this among the differential diagnosis of patients with non specific abdominal pain.

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