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# Clinical Spectrum of Splenic Infarction, a South Indian Perspective

ORIGINAL

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## Abstract

**Background:** Splenic infarction is a rare condition which is presently detected with increasing frequency on imaging studies, but our knowledge about its presentation and etiology is still inadequate. The clinical profile has evolved tremendously since its original description. Case reports with malarial etiology are preponderant in the Indian literature review. The study objective was to identify the prevailing clinical spectrum of splenic infarction in our tertiary care center.

**Methods:** Retrospective review of in-patients clinically diagnosed with splenic infarction in our hospital, from June 2013 to May 2015, was conducted. Details regarding age, gender, etiology of the infarct, underlying diseases and diagnostic tests were obtained from the electronic medical database. Imaging studies were analyzed for the infarct pattern and its complications.

**Results:** Splenic infarction was identified in 25 patients, constituting 0.015% of hospital admissions. Mean age was 43 years. Male to female ratio was 2.5:1. Abdominal pain was the cardinal symptom in only half the patients. 32% complained of fever. Abdominal tenderness was observed in 24%. 6 patients had splenomegaly. Leukocytosis occurred in 60%.Thromboembolic disorders were the predominant cause, followed by pancreatic disorders. 4 previously healthy patients (16%) were diagnosed with concealed fatal diseases. Single infarcts were noted in 16 (64%) patients, of which wedge shaped (36%) lesions were prevalent. Associated other visceral infarcts were present in 6 (24%) patients. Splenic abscess developed in 3 patients (12%), and one underwent splenectomy.

**Conclusion:** The foremost etiology in our center was thromboembolic disorder, in comparison with infectious causes. The mainstay therapy is treatment of the underlying disease.

**Keywords**
Splenic Infarct, Abdominal Pain, Splenomegaly, Embolus, Abscess.

 

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## Introduction

Splenic infarction is a rare clinical entity caused by occlusion of the splenic vascular supply. Etiology encompasses a heterogeneous group of disorder like hematological diseases, embolic disorders, hypercoagulable states, infections and trauma [1]. Despite being a relatively common radiological finding, only scanty case series have been reported. Most of the Indian literature on splenic infarction emerges from case reports emphasizing its presentation as a complication of infections [2-7]. A few case series published were limited to malaria and sickle cell trait [8, 9, 10]. The classical presentation of splenic infarction is left upper abdominal quadrant pain, tenderness and swelling accompanied by a peritoneal friction rub [11]. Our study objective was to determine the clinical profile of splenic infarction.

## Material and Methods

We conducted a retrospective review of patients admitted in Sri Ramachandra University from June 2013 to May 2015. Patients with clinically diagnosis of splenic infarction complemented with radiological imaging were included. Demographic details, presenting complaints, physical examination and investigations were collected from the electronic medical record. Imaging studies retrieved from the database were discussed with the radiologist.

## Results

We identified 25 cases of splenic infarction from the 168,572 admissions during the 2 year study period, accounting to a prevalence of 0.015%. 18 were males and 7 were females. Mean age was 43 years (range of 3 to 76). 56% of the patients presented with abdominal pain. Only 5 (20%) had localized left upper quadrant pain. 8 (32%) patients complained of fever. On examination, abdominal tenderness was elicited in 6 (24%) patients. Splenomegaly were present in a quarter of patients (24%). Typical features of splenic infarction with left upper abdomen pain, tenderness and splenomegaly, described by Osler, was found only in 2 patients (8%).

Blood investigations revealed anemia in 84%, leukocytosis in 60% and thrombocytosis in 8%. Elevated alanine transaminase and alkaline phosphatase levels were observed in 36% and 40% respectively. LDH levels were raised in 6 out of 7 patients. **Table 1** shows the demographic and clinical features of the cases.

**Table 1.** Demographic and clinical features of the cases.

| Demographics | | |
|---|---|---|
| Mean age (years, range) | 43 (3-76) | |
| Male/total | 18/25 | 72% |
| Presenting symptoms | | |
| Left upper quadrant pain | 5/25 | 20% |
| Other abdominal pain | 9/25 | 36% |
| Fever | 8/25 | 32% |
| Nausea and vomiting | 2/25 | 8% |
| Physical examination | | |
| Abdominal tenderness | 6/25 | 24% |
| Splenomegaly | 6/25 | 24% |
| Blood investigations | | |
| Hemoglobin <12 g/dl | 21/25 | 84% |
| White blood count >12,000/dl | 15/25 | 60% |
| Platelet count > 5.5 lakhs/dl | 2/25 | 8% |
| LDH > 250 | 6/7 | |
| Alanine transaminase > 40 U/L | 9/25 | 36% |
| Alkaline phosphatase > 130 U/L | 10/25 | 40% |
| Imaging | | |
| Infarcts on CT | 20/25 | 80% |
| Infarcts on ultrasound | 4/25 | 16% |
| Infarcts on MRI | 1/25 | 4% |
| Complications | | |
| Abscess | 3/25 | 12% |
| Outcome | | |
| Splenectomy | 1/25 | 4% |
| In-hospital mortality | 2/25 | 8% |

LDH: lactate dehydrogenase, CT: computerized tomography, MRI: magnetic resonance imaging.

The predominant etiology was thromboembolic disorders (n=4). Pancreatitis was reported in 3 cases. Other causes included hematological malignancy and liver diseases in 3 patients each. Polycythemia vera was diagnosed in 1 patient. Sepsis was responsible for the infarcts in 3 cases. Vascular disorders and trauma were found in 2 patients each. Infective endocarditis was present only in 1 patients, and atrial fibrillation was documented in 1. 6 patients had dual predisposing factors for splenic infarction. **Table 2** & **3** shows the etiological profile of patients with splenic infarction.

**Table 2.** Clinical features of patients with splenic infarction

| Case No. | Age | Gender | Presenting complaint | Chronic medical conditions | New diagnosis |
|---|---|---|---|---|---|
| 1 | 24 | F | Abd pain | Liver cirrhosis - Autoimmune | Arteriovenous fistula |
| 2 | 36 | F | LUQ abd pain | DM | Aortic, hepatic artery & splenic vein thrombosis; splenic abscess |
| 3 | 72 | M | LUQ abd pain | CVA | Aortic, inferior mesentery, ileocolic, femoral artery thrombosis; bowel gangrene |
| 4 | 76 | M | Dysphagia | DM | Ca Esophagus, portal vein thrombosis |
| 5 | 36 | M | Abd pain | Chronic pancreatitis | Hypovolemic shock, WOPN |
| 6 | 18 | M | LUQ abd pain, fever | | SLE, EBV |
| 7 | 44 | M | Fever | | Viral fever |
| 8 | 54 | M | Fatigability | Lymphoma | Pneumonia |
| 9 | 34 | M | Limb pain | DM | Splenic and renal injury, humerus fracture, necrotizing fasciitis, pneumonia |
| 10 | 54 | M | Anorexia | DM,HTN,CVA | Ca Esophagus |
| 11 | 3 | F | Joint pain, fever | Leukemia | Splenic abscess |
| 12 | 58 | M | Abd pain, fever | Polycythemia, Hepatitis B | Viral fever |
| 13 | 55 | M | Abd distension | Liver cirrhosis | ARF |
| 14 | 47 | M | Abd pain, vomiting | Ca Stomach | Nodal metastases |
| 15 | 60 | M | Abd pain, weight loss | | Metastatic Ca Pancreas |
| 16 | 56 | M | Abd pain, vomiting | | Chronic pancreatitis, splenic vein thrombosis, pancreatic fluid collection |
| 17 | 48 | M | Abd pain, anorexia | | Portal vein, splenic vein, SMV & IMV thrombosis |
| 18 | 47 | M | Abd pain, fever | | Pancreatitis, AF, ARF |
| 19 | 11 | M | Altered sensorium | Wilson's disease, PHT, Hypersplenism | Post embolization syndrome, pancreatitis, pneumonia, urinary tract infection |
| 20 | 55 | F | LUQ abd pain | | Vasculitis, obstructive sleep apnea |
| 21 | 8 | M | Fever | Leukemia | Mucormycosis, burst abdomen, pneumonia |
| 22 | 50 | F | Abd pain | Metastatic adenocarcinoma | Aortic thrombus, liver & splenic abscesses, pneumonia |
| 23 | 29 | F | Fever | | AFLP, Invasive mucormycosis, gastric perforation, MODS |
| 24 | 42 | F | LUQ abd pain, fever | RHD, HTN | Infective endocarditis, renal infarct |
| 25 | 46 | M | Dyspnea | RHD, DM, HTN, AF | Liver cirrhosis |

LUQ: left upper quadrant, abd: abdominal, DM: diabetes mellitus, HTN: hypertension, PHT: portal hypertension, CVA: cerebrovascular accident, Ca: carcinoma, RHD: rheumatic heart disease, AF: atrial fibrillation, WOPN: walled off pancreatic necrosis, SLE: systemic lupus erythematous, EBV: Ebstein-Barr virus mononucleosis, SMV: superior mesentery vein, IMV: inferior mesentery vein, ARF: acute renal failure, AFLP: acute fatty liver of pregnancy, MODS: multi organ dysfunction syndrome

**Table 3.** Etiology of splenic infarction.

| Underlying cause | Total no. of cases | New underlying diagnosis |
|---|---|---|
| Hematologic malignancy | 3 | 0 |
| Solid tissue malignancy | 3 | 2 |
| Aortic thrombosis | 3 | 2 |
| Pancreatitis | 3 | 0 |
| Liver disease | 3 | 0 |
| Sepsis | 3 | 0 |
| Vascular disorder | 2 | 2 |
| Trauma | 2 | 0 |
| Infective endocarditis | 1 | 1 |
| Portal vein thrombosis | 1 | 0 |
| Autoimmune | 1 | 1 |
| **Total** | 25 | 8 |

Computerised tomography was diagnostic modality in 20 patients (80%). Single (64%) and multiple (36%) infarcts were the two distinguishing patterns. The mean age of cases in both the groups was 42 years. Wedge shaped infarct was prevalent in 9 (36%), and was common in patients presenting with abdominal pain and fever. This observation could not be explained. Hypoechoic and near total patterns were found in 20% and 8% respectively. Multiple infarcts were more frequent in patients more than 40 years (n=8), with leukocytosis and thromboembolic etiology. **Figures 1**, **2** & **3** shows the different patterns of splenic infarction. Perisplenic collaterals were noted in 5 patients. Associated visceral infarcts of kidney or liver was reported in 6 (24%) cases.

Splenic abscess gradually developed in 3 patients (12%) with aortic thrombus. This could be due to infection of the liquefied infarct. One patient underwent splenectomy and the others were managed with antibiotics and analgesics. In-hospital mortality occurred in 2 patients succumbing to the multi-organ dysfunction.

**Figure 1:** Wedge shaped infarct in blunt trauma.

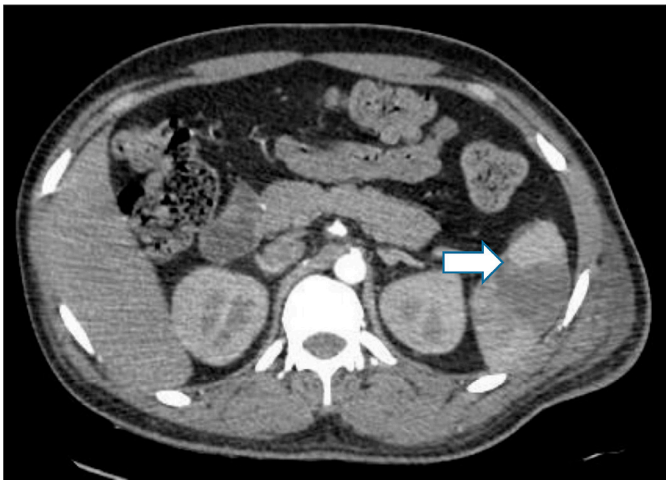


**Figure 2:** Multiple infarcts in thromboembolus.

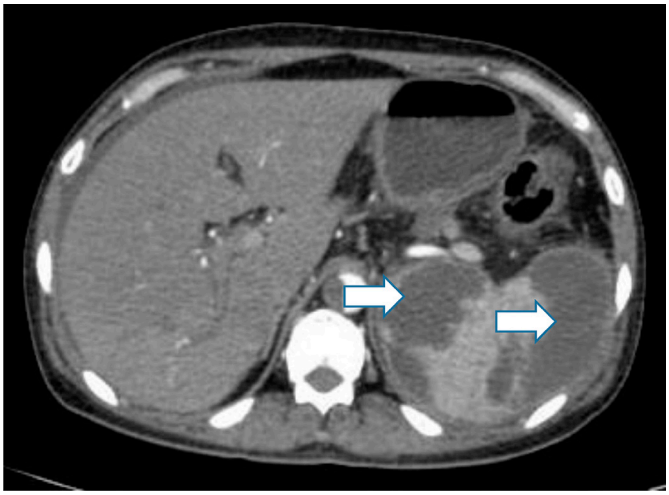
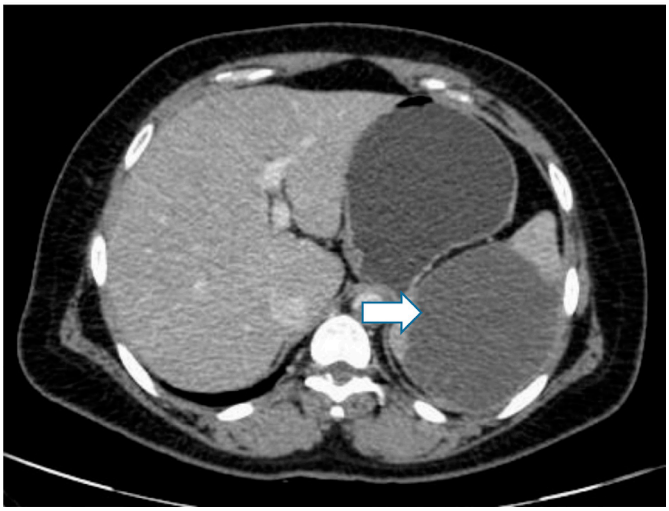


**Figure 3.** Near total infarct in thromboembolus.



## Discussion

Splenic infarction occurs due to arterial or venous compromise and is associated with a heterogeneous group of disorders. Although the frequency of detection has enhanced due to increased radiologic imaging of patients over the recent years, the exact incidence may be underestimated. Often the etiology and diagnosis of the infarction are overlooked. The prevalence rate in our study was 0.015% which is similar to a study by Ami et al [12].

Numerous etiologies have been included since its original description by Osler. The majority (88%) are infiltrative hematologic diseases causing congestion of the splenic circulation by abnormal cells, or thromboembolic disorders that produce obstruction of larger vessels. It is reported as a rare complication of malaria as highlighted in a few Indian case reports [2, 4, 5, 6]. Our study shows a wide range of etiological distribution, in which thromboembolic disorders was the predominant (20%). This observation was consistent with international studies [12, 13].

Recent studies have shown that the clinical presentation varies from asymptomatic to hemorrhagic shock [14, 15]. Abdominal pain was the foremost presenting symptom (56%), as against 80% reported in previous series [12, 13, 16]. Left upper quadrant tenderness was elicited in 24% in comparison with 35%. Infrequently the splenic infarct can be the presenting symptom of some important underlying life threatening disorder. Approximately 20% of the cases had such presentations [12, 16]. 4 patients in our study (16%) had no prior predisposing conditions.

Although leukocytosis, thrombocytosis and anemia were observed in few cases, laboratory tests are not diagnostic of splenic infarction. Ultrasonogram is useful when splenic parenchyma is not obscured by bowel gas or morbid obesity. Computerised tomography is the diagnostic modality of choice [17]. Other methods include gadolinium enhanced MRI, splenic scintigraphy with radio-labelled colloids, and RBC scans.

The mainstay of management is pain relief with NSAIDS or narcotic analgesics, due to tendency for complete healing with sufficient collateral flow. Complications occurred in 7 % to 20 % in previous studies. Sepsis, abscess, hemorrhage, pseudocyst formation and rupture warrant surgical intervention [18, 19].

The limitations in our study are relatively small number of identified cases, and possibly missing out a large number of asymptomatic patients and those with atypical presentations in whom imaging were not performed.

## Conclusion

Our study is probably the first in India to analyze the wide spectrum of etiology in splenic infarction. Infectious etiology was noted in less than one third of the patients. Clinical presentation is less apparent in contrast to other recent series, thereby emphasizing the need for a high clinical suspicion. Evaluation may bring forth the diagnosis of a fatal underlying disease. Medical management remains the mainstay treatment.

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