



ORIGINAL ARTICLE

Splenic infarction as a sentinel sign of underlying pathology: clinical insights from a seven-patient series

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ABSTRACT

Splenic infarction (SI) is a rare condition resulting from vascular occlusion that leads to ischemia and necrosis of the splenic parenchyma. Its diagnosis is often challenging because of its nonspecific clinical presentation, which may include left upper quadrant abdominal pain, fever, and constitutional symptoms. Diagnosis relies primarily on contrast-enhanced computed tomography (CT), which typically reveals characteristic wedge-shaped hypodense lesions extending toward the splenic hilum. SI frequently uncovers serious underlying disorders, including malignancies, hematologic diseases, and cardioembolic conditions. Recent studies report an incidence of approximately 0.016% among hospital admissions, with thromboembolic and hematologic disorders representing the most common etiologies. Therapeutic management is not standardized and should be tailored to the underlying cause, generally combining supportive care, anticoagulation in thromboembolic cases, and treatment of the associated disease. This study describes a series of seven patients diagnosed with SI and aims to analyze their clinical, biological, radiological, etiological, therapeutic, and outcome characteristics. Medical records of patients hospitalized for splenic infarction between 2016 and 2025 were retrospectively reviewed. The cohort included four women and three men, with a mean age of 47 years. Acute abdominal pain was the predominant presenting symptom. Most patients exhibited elevated inflammatory markers, and imaging frequently revealed multiple splenic infarcts, occasionally associated with renal infarction or splenomegaly. The etiological spectrum was broad and included hematologic, autoimmune, infectious, and inflammatory disorders, with multifactorial causes identified in some cases. Management was based mainly on anticoagulation and antibiotic therapy. The overall prognosis was favorable, with clinical recovery observed in the majority of patients (85%).

Keywords: splenic infarction, inflammatory disease, metabolic disease, hematologic disorder, thromboembolic event.

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1. INTRODUCTION

Splenic infarction (SI) is a rare clinical condition that is often difficult to diagnose because of its nonspecific clinical presentation. It can mimic other causes of abdominal pain, and diagnosis is primarily based on imaging, particularly computed tomography (CT), where it is frequently an incidental finding. Although SI is often asymptomatic, it may represent the initial manifestation of serious underlying conditions such as hematologic disorders, malignancies, or cardioembolic events (1-3). The etiologies of SI are diverse and directly influence management, which remains poorly standardized (1,2). Recent reviews highlight that thromboembolic events, including those from atrial fibrillation or infective endocarditis, account for a significant proportion of cases, while hematologic malignancies

and hypercoagulable states are also common. In a large retrospective series, 20% of patients presented with left upper quadrant pain, and in-hospital mortality was 20%, underscoring the potential severity (1).

The objective of this study is to describe the clinical, biological, morphological, etiological, and evolutionary aspects of splenic infarction through a case series.

2. PATIENTS AND METHODS

This retrospective, observational, single-center study was conducted in the Department of Internal Medicine at a hospital in Algiers, Algeria. We analyzed the medical records of patients hospitalized between January 2016 and December 2025. All patients aged over 16 years who presented with splenic infarction were included.

Pregnant women were excluded because, in our hospital, splenic infarction during pregnancy is managed jointly by the obstetrics and internal medicine teams. Their medical records were therefore not available in our records archive, making their inclusion impossible in this retrospective study. Following exclusions, a total of 7 patients with splenic infarction were included in the analysis.

3. RESULTS

We report seven cases of splenic infarction (SI) involving four women and three men, with a mean age of 47 years (range: 28–74 years). Table 1 summarizes patient history, clinical examination findings, laboratory results, imaging features, clinical outcomes, aetiologies, and disease evolution.

All patients presented with acute abdominal pain; one patient also reported associated chest pain. Fever was observed in 29% of cases, while anorexia, vomiting, and asthenia were each reported in 14%. Elevated inflammatory markers were found in more than 71% of patients. Splenic infarcts were multiple in 67% of cases, associated with renal infarction in two patients (Fig. 1), and splenomegaly was present in two cases. A single etiology was identified in four patients: myeloproliferative syndrome (Fig. 2), celiac disease, systemic lupus erythematosus, and infective endocarditis (Fig. 3). A multifactorial etiology was identified in two cases: one patient had a history of cholecystectomy with sickle cell trait, obstructive sleep apnea–hypopnea syndrome (OSAHS), and splenomegaly, while the other presented with a flare of Crohn’s disease complicated by perforation and neglected peritonitis. Antibiotic therapy was administered to 71% of patients. Six patients received warfarin, with close clinical and laboratory monitoring. The target INR was tailored to the underlying cause of the splenic infarction. No haemorrhagic complications occurred. Follow-up abdominal CT scans showed complete resolution of the infarction within 6 to 12 months in 67% of cases. In one patient, however, the infarction extended, and splenectomy became necessary. Anticoagulation was maintained long-term for those with a mechanical heart valve or a myeloproliferative syndrome. In the remaining patients, we stopped anticoagulation once their condition was controlled. Overall, 85% of patients had a favorable clinical course. Tragically, one patient died from neurological complications due to septic embolism secondary to infective endocarditis.

4. DISCUSSION

The clinical presentation of splenic infarction (SI) in our series is consistent with the literature, in which left upper quadrant abdominal pain represents the hallmark symptom and may radiate to the left shoulder. Fever, chest pain, and constitutional symptoms may also be present. Notably, SI can constitute the initial manifestation of serious underlying conditions, including hematologic malignancies, atrial fibrillation, infective endocarditis, and autoimmune or infectious diseases. In a case series of 18 patients with radiologically confirmed SI, approximately half presented with abdominal pain, while the remainder were asymptomatic, highlighting the variable symptomatology (1). Additionally, atypical presentations such as acute left-sided chest pain have been reported, posing diagnostic challenges and mimicking cardiac or pulmonary conditions (4). For instance, in an adolescent with hereditary spherocytosis, SI manifested with chest pain, lethargy, and anemia, underscoring its occurrence in younger patients with underlying hematologic disorders (5). Laboratory findings commonly reveal leukocytosis, elevated lactate dehydrogenase (LDH) levels, and increased inflammatory markers. Significant elevations in D-dimer, CRP, and LDH, along with leukocytosis with neutrophilia, anemia, hypoalbuminemia, and liver dysfunction, have been observed. These parameters can serve as prognosis-predicting factors (1). Contrast-enhanced computed tomography (CT) remains the gold standard for diagnosing SI, offering superior sensitivity compared with ultrasound and allowing differentiation from other causes of acute abdomen (1-4). Global splenic infarction, as seen in cases of thoracic aortic mobile thrombus, may be challenging to diagnose on portal venous phase imaging but is better visualized on arterial phase CT.

Table 1. Cases history, examination, laboratory tests, imaging, clinical results, etiology and evolution.

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7
Sexe	Female	Male	Male	Male	Female	Female	Female
Age	37	49	40	70	66	28	42
Past Medical History	Crohn Disease	-	HTA OSAHS SCT	-	Mechanical Mitral Prothesis	Portal End cerebral thrombosis	—
Symptoms	Abdominal pain	Abdominal pain	Abdominal pain	Abdominal pain Fever Chest pain Vomiting Anorexia	Fever Abdominal pain	Abdominal pain	Abdominal pain Arthralgia
Laboratory Finding							
Neutrophils	↑	↑	—	↑	↑	—	—
SR	↑	↑	—	—	↑	—	↑
CRP	+	+	—	+	+	—	+
HB	11	13	10	9	11	10	9
CT finding	Multiple splenic infarct Renal infarct Enlarged spleen	Multiple splenic infarct	Enlarged spleen Large splenic infarct	Aortic thrombosis Multiple splenic infarct Renal infarct	Splenic infarct	Splenic infarct	Splenic infarct
Etiology	Crohn's Disease Flare-up Sepsis Surgery	—	OSAHS SCT Enlarged Spleen	Myeloproliferative syndrome	Endocarditis	Coeliac Disease	Lupus erythematosus
Treatment	Antibiotic anticoagulation	Aspegic	Anticoagulation splenectomy	Anticoagulation Specific treatment	Antibiotic anticoagulation	Anticoagulation Gluten free Diet	Anticoagulation Corticosteroids Hydroxychloro- quine
Evolution	+	+	+	+	-	+	+



Figure 1. Case 4: Abdominopelvic CT showing multiple splenic and renal infarcts.

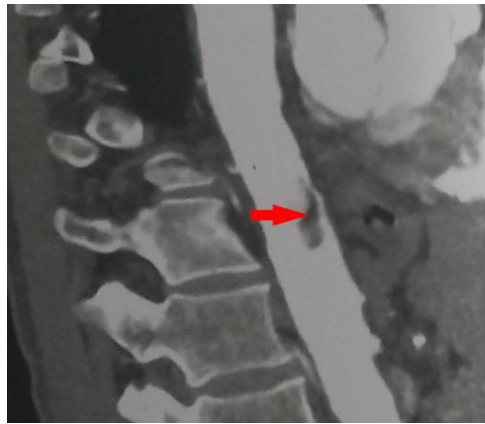


Figure 2. Case 4. Thoracoabdominal AngioTDM showing floating thrombus at the thoracic aorta.

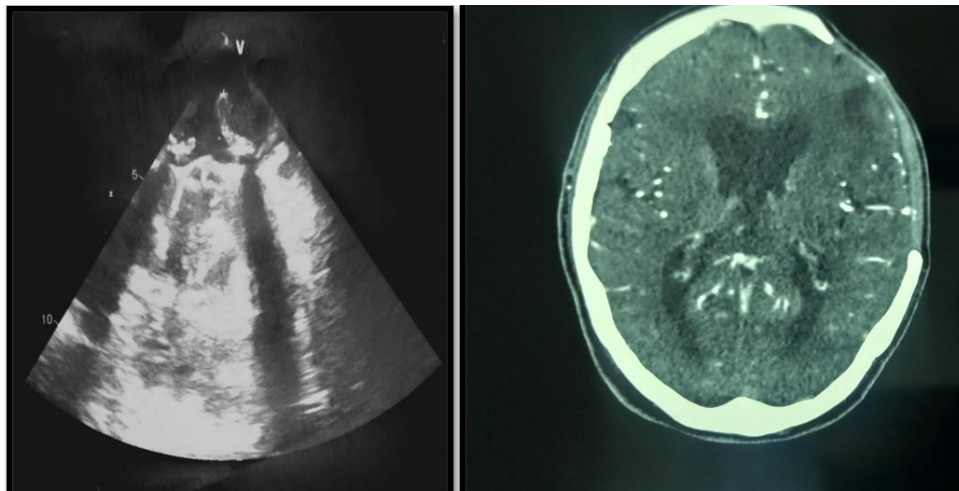


Figure3. Case 5. Left: transoesophageal echocardiography showing findings on a mechanical mitral valve prosthesis, right: Cerebral CT showing a range of right frontal ischemia, in a patient with an infectious endocarditis complicated by a splenic infarct.

Our case series highlights the wide etiological spectrum of splenic infarction, which can be systematically classified into thromboembolic, hematologic, infiltrative, and infectious causes, each associated with distinct and well-established pathophysiological mechanisms. An almost systematic review of the literature from 1991 to 2022, encompassing 11 cohort studies and 867 clinical cases, identifies the most frequent causes as emboligenic heart disease, hematological malignancies, solid neoplasia, and certain infections (2). Embolic phenomena are exemplified by infective endocarditis, in which valvular vegetations lead to arterial occlusion by septic or bland emboli, as well as by local vascular events such as thrombosis or spontaneous dissection of the celiac artery. Splenic infarction has been reported as the presenting manifestation of infective endocarditis in a pregnant woman without prior cardiovascular risk factors, emphasizing the need for echocardiographic evaluation in such cases (7). Atrial fibrillation can also cause simultaneous cerebral and splenic infarctions through cardioembolic mechanisms (9).

Hematologic disorders represent a major risk category, as myeloproliferative neoplasms promote a hypercoagulable state and sickle cell trait induces vaso-occlusive phenomena, particularly under conditions of hypoxia or dehydration. A comprehensive systematic review of 90 case studies on splenic infarction in sickle cell trait patients revealed that most cases occurred in males (64%), with a mean age of 27 years, often triggered by high-altitude exposure or strenuous activity, and typically managed conservatively with

favorable outcomes (12). Hereditary spherocytosis, another hematologic condition, can present with splenic infarction in adolescents. In addition, systemic inflammatory conditions such as inflammatory bowel disease (IBD) generate a prothrombotic environment driven by active disease, systemic inflammation, and associated nutritional deficiencies, which may lead to hyperhomocysteinemia or deficiencies in natural anticoagulants such as proteins C and S, as observed in celiac disease. Splenic infarction has been described as a rare manifestation of celiac disease, potentially due to thromboembolism in the celiac artery. In Crohn's disease, thrombotic events extend beyond the spleen, as evidenced by cases of acute myocardial infarction shortly after diagnosis, highlighting the hypercoagulable state in active IBD (8). Obstructive sleep apnea–hypopnea syndrome (OSAHS) is increasingly recognized as a significant prothrombotic condition, as chronic intermittent hypoxia and hypercapnia induce endothelial dysfunction, oxidative stress, and systemic inflammation, thereby contributing to the multifactorial pathogenic landscape of SI. (2-13)

In systemic lupus erythematosus without secondary antiphospholipid syndrome, thrombotic events may occur in up to 20% of cases, though SI is an exceptional event. Multifactorial complications may result from lupus-related vasculitis involving the splenic arterioles with inflammatory occlusion of these vessels due to local inflammation triggered by complement activation; furthermore, systemic inflammation induces a prothrombotic state, which may promote *in situ* thrombosis. Thrombotic microangiopathy may also contribute to splenic ischemia. Nephrotic syndrome, another recognized prothrombotic condition in lupus, was not observed in our patient, who presented with secondary Sjögren's syndrome. A rare case of a 23-year-old female with recently diagnosed SLE and negative antiphospholipid antibodies presented with abdominal pain due to splenic infarcts, managed successfully with steroids and hydroxychloroquine. Similarly, splenic infarction can be the sole manifestation of antiphospholipid syndrome in patients with rheumatoid arthritis, underscoring the overlap of autoimmune conditions (13-15).

Management of SI is primarily supportive and includes analgesia, hydration, and close monitoring for complications. Anticoagulation is indicated in cases with a clearly identified thromboembolic etiology in association with treatment of the underlying aetiology. Surgical intervention is reserved for complicated or refractory cases. Overall prognosis is generally favorable; however, mortality increases in the presence of comorbidities such as cirrhosis or advanced hematologic disease (1,2). In a case series, one-third of patients died, with prognostic factors including leukocytosis, decreased lymphocytes, prolonged activated partial thromboplastin time, decreased fibrinogen, elevated LDH, and liver dysfunction (1). A multicenter retrospective study on SI in emergency departments identified key prognostic factors, emphasizing the need for risk assessment to predict outcomes (3). Recent multicenter studies suggest that anticoagulant therapy may improve long-term survival without significantly increasing bleeding risk (16).

This case series underscores the importance of splenic infarction as a diagnostic marker, emphasizing the need for clinical vigilance, prompt CT confirmation, and a thorough etiological workup to ensure optimal patient outcomes.

Our study's main limitation is its retrospective, single-centre design and the small number of patients. This inevitably limits statistical power, the representativeness of the sample, and the generalisability of our findings. Splenic infarction is rare. For that reason, this series offers clinically meaningful descriptive data on presentation, aetiology, treatment, and outcome. These results must, however, be interpreted with caution.

5. CONCLUSION

Although rare, splenic infarction should be considered in the differential diagnosis of acute or subacute abdominal pain, particularly when initial evaluation is inconclusive. Diagnosis relies primarily on imaging, namely contrast-enhanced computed tomography. Management is directed toward addressing the underlying etiology rather than the infarction itself. Consequently, a comprehensive etiological workup is essential, including the investigation of uncommon causes such as autoimmune and systemic inflammatory diseases in order to guide appropriate treatment and improve patient prognosis.

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