

# Acute Abdominal Pain with an Unusual Etiology: Splenic Infarction in a Diabetes Patient

Metin Yalaza<sup>1</sup>, Mehmet Tolga Kafadar<sup>2</sup>, Gürkan Değirmencioğlu<sup>3</sup>, Ahmet Türkan<sup>4</sup>, Meral Şen<sup>5</sup>

<sup>1</sup>Clinic of General Surgery, Division of Surgical Oncology, Ankara Numune Training and Research Hospital, Ankara, Turkey

<sup>2</sup>Clinic of General Surgery, Mehmet Akif İnan Training and Research Hospital, Şanlıurfa, Turkey

<sup>3</sup>Department of General Surgery, Turgut Özal University School of Medicine Ankara, Turkey

<sup>4</sup>Clinic of General Surgery, Dr. Münif İslamoğlu State Hospital, Kastamonu, Turkey

<sup>5</sup>Clinic of General Surgery, Ankara Medsentez Policlinic, Ankara, Turkey

Splenic infarction is a rare clinical event caused by reduced blood flow to the spleen. Its presentation can mimic other causes of acute abdominal pain. The most frequent causes of splenic infarction include thromboembolic events, malignant hematologic neoplasms, and vasculitides. A few reports of single or multiple cases of diabetes-induced splenic infarction have been published in medical journals. Herein, we report a 67-year-old patient with diabetes-associated splenic infarction who presented to an emergency department with acute abdominal pain.

**Keywords:** Acute abdomen, splenic infarction, diabetes mellitus

## INTRODUCTION

Splenic infarction is a relatively rare event in which a portion of the spleen dies due to interruption of the blood supply to the affected tissue for any reason. An interruption in the blood supply can be caused by thrombosis, emboli, changes in blood pressure, twisted blood vessels, trauma, and blood disorders (e.g., leukemia and abnormal blood coagulation). The severity of symptoms depends on the amount of splenic tissue affected (1). Herein, we present a patient with splenic infarction secondary to diabetes mellitus who subsequently developed sterile peritonitis as a cause of acute abdominal pain.

## CASE PRESENTATION

A 67-year-old woman with diabetes mellitus was admitted to our hospital complaining of acute abdominal pain in the upper left quadrant, nausea, and vomiting lasting 2 days. Her medical history included congestive heart failure, hypertension, and diabetes mellitus. She was treated with spironolactone. Physical examination on admission revealed palpable splenomegaly. There was tenderness in all quadrants, with guarding and rebound tenderness. During laboratory investigations, the following results were revealed: white blood cell count, 15,700/mm<sup>3</sup> (neutrophils, 91%); hematocrit, 43%; hemoglobin, 14.1 g/dL; platelet count, 245,000/mm<sup>3</sup>; fasting blood glucose, 320 mg/dL; C-reactive protein, 9.63 mg/dL (normal, 0–0.8 mg/dL); lactate dehydrogenase, 1130 U/L (normal, 240–480 U/L); and aspartate transaminase, 61 U/L (normal, 0–38 U/L). There was no gross pathology in her echocardiogram. Computed tomographic (CT) images of the abdomen depicted regions of low attenuation in the spleen that were consistent with acute infarction over nearly three-fifths of the spleen (Figure 1). The patient was transferred to the operating room for emergency surgery. During the surgery, an area of infarct (8x6 cm) was found in the spleen and a total splenectomy was performed. The postoperative period was uneventful and the patient was discharged on postoperative day 5. Informed consent was obtained from the patient who participated in this case.

## DISCUSSION

Splenic infarction secondary to diabetes mellitus is a rarely encountered clinical event. Although there are numerous causes of splenic infarct, the majority (88%) of cases involve either infiltrative hematologic diseases, which causes congestion of the splenic circulation by abnormal cells, or thromboembolic conditions, which obstruct larger vessels (2).



**FIGURE I.** Abdominal computed tomographic images reveal several areas of low attenuation in the spleen, the largest of which is located superolaterally and posteriorly

The cause of the infarct varies with age; an associated hematologic disorder is more common in patients aged <40 years, whereas an embolic event is more common in patients aged >41 years (3). The clinical spectrum ranges from asymptomatic infarction to hemorrhagic shock and acute abdomen, as in our patient. Approximately one-third of splenic infarcts are clinically occult. In a 10-year retrospective study, Antopolsky et al. (1) examined the clinical presentations in 49 episodes of acute splenic infarction. The most common symptom was abdominal or left flank pain (80% of episodes), while the most common sign was tenderness in the upper left quadrant (35% of episodes). In 16.6% of patients, splenic infarction was the presenting symptom of an underlying disease. Antopolsky et al. (1) also reported that risk factors for splenic infarction were present in 71% of patients. The risk factors included atrial fibrillation in 23% of patients and a history of previous splenic infarction in 8% of patients. Essential hypertension, diabetes mellitus, chronic obstructive pulmonary disease, and chronic heart failure were present in 31%, 23%, 8%, and 8% of patients, respectively. Other symptoms include fever and chills, nausea and vomiting, pleuritic chest pain, and left shoulder pain (Kehr's sign) (1). Septic thromboemboli may result in splenic abscesses, which present with sepsis and left upper abdominal pain. In the case series reported by Nores et al. (2), most of the patients with thromboembolic infarction were symptomatic: 70% of patients with emboli were febrile, and 86% of individuals with thrombosis had abdominal pain.

It is possible that the splenic infarct in our patient was caused by cardiac-related embolization because she had chronic atrial fibrillation and was on oral anticoagulant therapy. However, the echocardiogram revealed no evidence of cardiac-related thromboembolism. Furthermore, all of the postoperative hematologic tests were within the normal ranges. It is unclear whether diabetes itself was the cause of sterile peritonitis in our patient. However, it is well known that severe vascular diseases, extensive atherosclerosis, and thromboembolic events are common in patients with advanced diabetes. Therefore, the prolonged diabetic

state may have facilitated the formation of extensive atherosclerotic lesions in blood vessels, including the splenic artery, which might have led to splenic infarction and resulted in culture-negative peritonitis (4).

The abdominal contrast-enhanced CT scan is the best and most advantageous diagnostic procedure of choice for splenic infarction. It is also more advantageous for the identification of other pathologies. The possibility of splenic infarct should be considered in patients at risk and with non-specific upper left quadrant pain, and a CT scan should be performed (5).

Splenic infarction is caused by ischemic events in the spleen. Patients with diabetes mellitus often have impaired vascular endothelial function, including altered vasomotor activity, vascular smooth muscle cell dysfunction, overproduction of inflammatory cytokines and chemokines, impaired platelet function, and abnormal coagulation. These abnormalities lead to increased vasoconstriction, thrombosis, and inflammation, which may cause splenic infarction (6). It is notable that, owing to the absence of complications (e.g., abscess or pseudocyst formation, hemorrhage, and rupture), splenic infarction was the only cause of acute abdominal pain in our patient.

## CONCLUSION

In this case report, we have described a rare presentation of splenic infarction in a patient with diabetes mellitus. As a rare cause of acute abdominal pain, splenic infarction is likely to contribute to the heterogeneous clinical manifestations of diabetes mellitus. Surgery is indicated for patients with complications and for patients with acute abdominal pain.

**Informed Consent:** Written informed consent was obtained from patient who participated in this study.

**Peer-review:** Externally peer-reviewed.

**Author contributions:** Concept - M.Y., M.T.K.; Design - M.Y., M.T.K., G.D.; Supervision - M.Y., M.T.K., A.T.; Resource - M.Y., M.T.K., G.D.; Materials - M.Y., M.T.K., G.D.; Data Collection and/or Processing - M.Y., M.T.K., M.S.; Analysis and/or Interpretation - M.Y., M.T.K., A.T.; Literature Search - M.Y., M.T.K., A.T., M.S.; Writing - M.Y., M.T.K.; Critical Reviews - M.Y., M.T.K., M.S.

**Conflict of Interest:** No conflict of interest was declared by the authors.

**Financial Disclosure:** The authors declared that this study has received no financial support.

## REFERENCES

1. Antopolsky M, Hiller N, Salameh S, Goldshtein B, Stalnikowicz R. Splenic infarction: 10 years of experience. *Am J Emerg Med* 2009; 27: 262-5. [\[CrossRef\]](#)
2. Nores M, Phillips EH, Morgenstern L. The clinical spectrum of splenic infarction. *Am Surg* 1998; 64: 182-8.

3. Jaroch MT, Broughan TA, Hermann RE. The natural history of splenic infarction. *Surgery* 1986; 100: 743-50.
4. Cox M, Li Z, Desai V, Brown L, Deshmukh S, Roth CG, et al. Acute nontraumatic splenic infarctions at a tertiary-care center: causes and predisposing factors in 123 patients. *Emerg Radiol* 2016; 23: 155-160. [\[CrossRef\]](#)
5. Joshi SC, Pant I, Shukla AN, Anshari MA. Splenic infarct as a diagnostic pitfall in radiology. *J Cancer Res Ther* 2008; 4: 99-101. [\[CrossRef\]](#)
6. Erarslan E, Bozkurt A, Yüksel I, Demir HD. Spontaneous splenic infarction in an elderly cirrhotic patient with multiple comorbidities. *Turk J Gastroenterol* 2012; 23: 596-8. [\[CrossRef\]](#)