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Case Report

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A case report of splenic infarction secondary to atherosclerosis of the splenic artery

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Abstract

Splenic infarcts are uncommon and may not be readily considered in the emergency department, as the clinical presentation often mimics acute abdominal pain. While atherosclerotic damage commonly occurs at the abdominal aorta and its branching parts**, it can also affect the splenic artery, potentially leading to splenic infarction. Although it is widely acknowledged that the abdominal aorta and its branches are the most common sites for atherosclerotic damage, atherosclerosis of the splenic artery can also be an etiological factor for splenic infarction, leading to impaired blood flow in the spleen. Splenic infarction represents a clinical scenario that is not frequently encountered, and its presentation can resemble other causes of acute abdominal pain. This case report describes a patient who presented to the emergency department (ED) with abdominal pain and was subsequently diagnosed with splenic infarction secondary to atherosclerosis of the splenic artery.

Keywords: Abdominal Pain, Atherosclerotic, Case Report, Splenic Infarction

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Introduction

Atherosclerosis is the main cause of morbidity and mortality in developed countries. It typically occurs over many years, characterized by the discontinuous development of atherosclerotic plaques with silent periods interrupted by phases of rapid change. Generally, clinical manifestations may become apparent after a long period of silence, presenting as chronic conditions such as angina pectoris, myocardial infarction, intermittent claudication, stroke, or sudden cardiac death. Some individuals may exhibit no signs of atherosclerosis throughout their lifetime (1). Atherosclerotic damage commonly occurs in the abdominal aorta and its branching parts. Although it is widely recognized that the abdominal aorta and its branches are the most common sites for atherosclerotic damage (2-4), less common locations such as the splenic artery can also be affected. Atherosclerosis of the

splenic artery is one of the etiological factors for splenic infarction, leading to impaired blood flow in the spleen (5). Splenic infarction is an uncommon clinical situation, often mimicking other causes of acute abdominal pain (6). It is characterized by vessel obstruction, parenchymal ischemia, and tissue necrosis involving a part or the entirety of the spleen. Etiologies of spleen infarction include splenic artery aneurysm, pancreatitis, septic embolism, and vascular diseases (7, 8). In this case report, we present a case of splenic infarcts secondary to atherosclerosis of the splenic artery in a patient presenting with abdominal pain to the emergency department (7).

Case Report

A 56-year-old previously healthy female was referred to Tohid Hospital in Sanandaj with a three-day history of abdominal pain. The abdominal pain was accompanied by nausea, vomiting, and anorexia. Her vital signs were normal. Suspecting a surgical abdominal emergency, the primary care physician referred the patient to the emergency department and a specialized clinic. There, after assessments by an internist and a surgeon, urgent and necessary laboratory tests, as well as chest, abdomen, and pelvic CT scans with and without contrast, were conducted. Additionally, non-contrast chest, abdomen, and pelvic CT scans, along with MRI, were performed, revealing marked splenomegaly with multiple cystic and mass-like structures in the spleen. These findings were highly suggestive of multiple splenic infarcts along with atherosclerosis of the splenic artery (Figures 1 and 2).

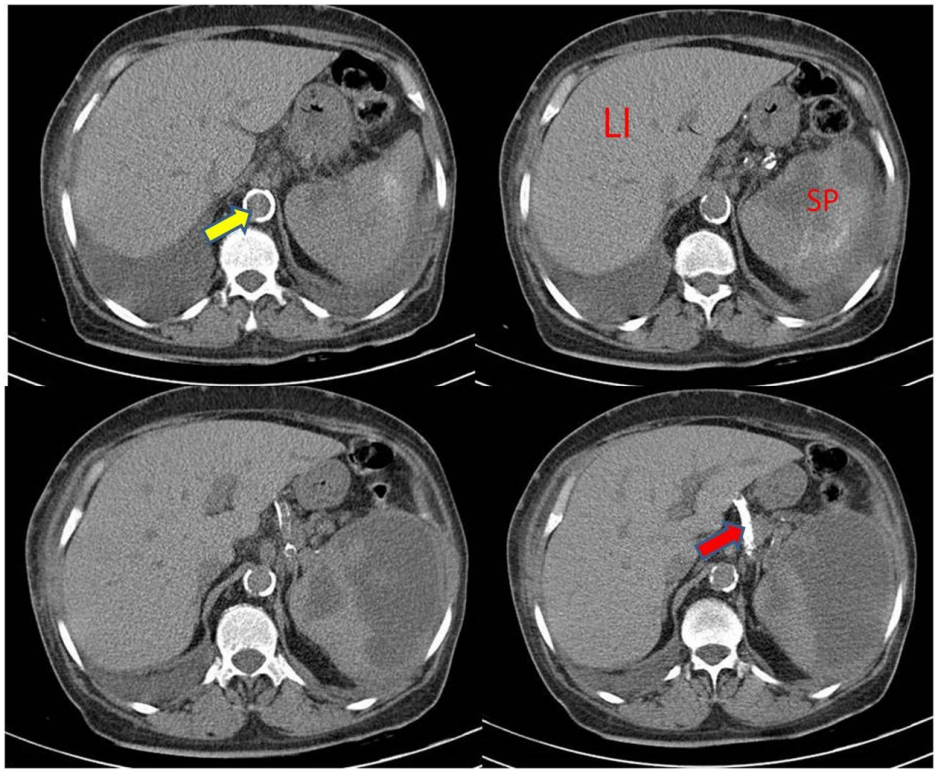

Fig. 1. Axial non-contrast CT scan (16-slice) of the abdominal region revealing atherosclerosis with splenic infarction. Yellow arrow: aorta; red arrow: splenic artery; S: spleen; LI: liver.

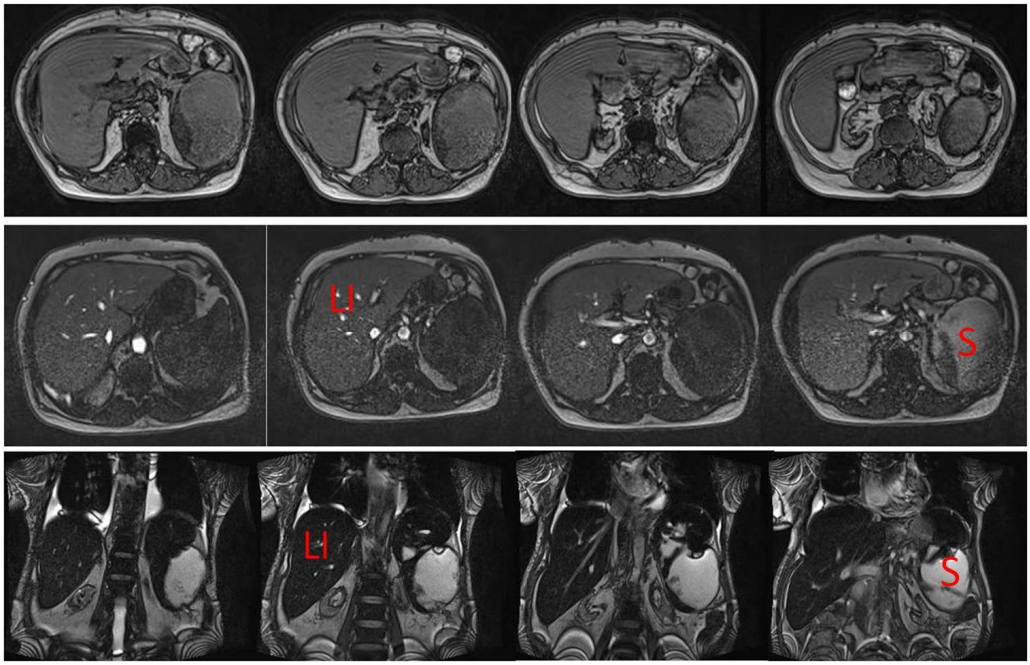

Fig. 2. Axial and coronal non-contrast MRI images of the abdominal region revealing atherosclerosis with splenic infarction. S: spleen; LI: liver.

Discussion

While the cerebral, coronary, and renal blood flows have been extensively studied by physicians, the mesenteric blood flow has historically received less attention despite its significance. However, although most of the gastrointestinal tract is not composed of vital organs, the intestinal circulation accounts for a significant portion of the cardiac output, and the narrowing of major arteries in intestinal blood flow may result in severe consequences (9). Atherosclerosis is an inflammatory disease that begins in the arterial branching areas. Hemodynamic factors play a role in the localization of atherosclerosis (10, 11). The abdominal aorta is the location for most primary and severe atherosclerotic diseases. The extent of the damages in the aorta may reflect a patient's tendency for atheroma formation (12).

Atherosclerotic plaques are created at the orifice of the blood vessels and within the first 2–3 cm of the main trunk of vessels (2, 13). In a study by Reiner et al., it was found that atherosclerosis plaques develop in the first 9–12 cm of the superior mesenteric artery (4). Another study also found that these plaques develop throughout the splenic artery (4). In the mesenteric, renal, and splenic arteries, fewer plaques are produced than in other vessels (14, 15).

The most common cause of acute mesenteric ischemia in atherosclerotic areas is visceral arterial thrombosis. Superior mesenteric artery occlusion usually occurs in its proximal segment, and the most common part of the gastrointestinal tract affected is the ileocecal area (9). Acute stenosis of the superior mesenteric artery is generally lethal, but chronic stenosis or blockage may be compensated for by the collateral plexus between these vessels, leading specifically to intestinal angina (16, 17).

Atherosclerosis of the splenic artery is one of the etiological agents of splenic infarction, and it might cause impaired blood flow in the spleen and splenomegaly (18). Splenic infarction is mainly caused by the obstruction of the spleen's feeding vessels. It results from arterial or venous occlusion and is

accompanied by a group of heterogeneous diseases. Generally, hematological irregularities and disorders such as leukemias, lymphomas, and myelofibrosis are the causative factors for splenic infarction. These diseases can cause impairment of splanchnic blood flow by abnormal cells or thromboembolic factors that generate vascular blockage (19, 20). Spleen infarction is caused by parenchymal ischemia and tissue necrosis, resulting from the discontinuation of arterial perfusion to the spleen.

The infarction may occur in a part of the spleen or in the whole spleen. Hematological irregularities and disorders that cause the impairment of splenic blood flow with abnormal cells or thromboembolism form the most common (88%) reasons for an infarct (21). It has been reported that the rate of progression of spleen infarction is 50 to 72% in patients with CML and myelofibrosis. Spleen infarction may be caused by factors such as autoimmune diseases, trauma, surgery, and infection (21). It is the first sign of an underlying disease in 16.6% of the patients (22).

Conclusions

The primary condition in this case, splenic infarcts, is usually characterized in patients with atherosclerotic involvement affecting numerous vascular regions. The development of obstructive lesions is an intricate, lengthy process, and signs could manifest in the clinical expression of diseases only at advanced stages. Late detection in these cases considerably increases the mortality rate. This case highlights the importance of considering splenic infarction as a differential diagnosis in patients presenting with abdominal pain, especially those with risk factors for atherosclerosis. Early recognition and appropriate management of this condition are crucial for improving patient outcomes.

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Author Contributions

MBHS supervised the study and drafted the manuscript; SM collected the clinical data.

Compliance with Ethical Standards

Written informed consent was obtained from a legally authorized representative(s) for anonymized patient information to be published in this article. This research has been confirmed by the Research Center of Kurdistan University of Medical Sciences with the file number IR.MUK.REC.1399.082.

Conflict of interest

The authors have no conflict of interest in this study.

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