

A Rare Cause of Abdominal Pain: Splenic Artery Aneurysm and Endovascular Treatment

Halil Oktay Usluer¹, Akif Yarkaç², Çağrı Safa Buyurgan², Buğrahan Güldür²,
Ataman Köse², Seyran Bozkurt² and Engin Kara³

¹Siverek State Hospital, Emergency Medicine Department, Sanliurfa, Turkey

²Mersin University Medical Faculty, Emergency Medicine Department, Mersin, Turkey

³Mersin University Medical Faculty, Radiology Department, Mersin, Turkey

Keywords: Abdominal Pain; Coil Embolization; Emergency; Rupture; Splenic Artery

Received: 15 February 2024; Accepted: 29 March 2024

INTRODUCTION

Splenic artery aneurysm (SAA) and splenic artery rupture are rarely seen in the emergency department. The most common visceral artery aneurysm is a SAA, which accounts for about 60–80% of all visceral artery aneurysms. Other common sites include the hepatic artery, the superior mesenteric artery, and the celiac artery [1]. However, SAAs are more prevalent, especially among women and individuals with certain risk factors such as pregnancy, portal hypertension, or connective tissue disorders [2,3]. SAA rupture has been reported to occur between 2% and 10% of SAA cases [4]. A ruptured SAA is an emergency condition and can be fatal if not treated early. Here, we aim to present a case of syncope after abdominal pain due to a ruptured SAA.

CASE REPORT

A 51-year-old male patient presented to the emergency department with abdominal pain and diarrhea, both of which he had been experiencing for 10 days, and dizziness, which had started on the day he arrived at the emergency department, followed by syncope. He had no

known comorbidities. There was no history of previous operations, pancreatitis, or abdominal trauma. Vital signs were blood pressure 65/42 mmHg, pulse rate 72 per minute, and respiratory rate 20 per minute. Physical examination was normal except for diffuse abdominal tenderness. The laboratory findings were as follows: Hgb: 9.4 g/dL, pH: 7.36, HCO₃: 22.0 mmol/L, Be₂-ecf: -2.7 mmol/L.

A 64-slice contrast-enhanced computed tomography (Toshiba Activion 16-Toshiba Medical Systems Corporation, Tochigi, Japan) revealed diffuse, dense free-fluid densities in the perigastric, peripancreatic, and perisplenic areas; faint density increases in the mesenteric adipose tissue that may be compatible with hematoma; and an aneurysm in the splenic artery. The presence of fluid and fat stranding around the aneurysm and around the spleen suggested the diagnosis of aneurysm rupture (Figures 1–3). Aortic aneurysms and dissections were excluded.

An SAA rupture was considered, and intravenous fluid support was administered. The patient underwent emergency surgery by the general surgery department. On exploration, there was diffuse hemorrhagic fluid and coagulum in the abdomen. Approximately 500 cc of hemorrhagic fluid was aspirated. No active bleeding focus could be detected during the operation, and the operation was terminated by placing a drain. The patient underwent an interventional procedure by the Department of Interventional Radiology.

After the placement of a 6F introducer in the right common femoral artery, images were obtained from the celiac superior mesenteric arteries. Subsequently, an attempt was made to reach the splenic artery using 4F and 5F cobra catheters with microcatheter wire complications, but it could not be successfully reached.

Corresponding author:

Halil Oktay Usluer, Siverek State Hospital, Emergency Medicine Department, Sanliurfa, Turkey.

Email: hoktayusluer@gmail.com

© 2024 The Author(s)

This is an open access article published under the terms of the Creative Commons Attribution License (CC BY 4.0), which permits use, distribution and reproduction in any medium, provided the original work is properly cited.



Figure 1 The appearance of an SAA is shown with a black arrow (sagittal plan) (the presence of fluid and fat stranding around the aneurysm and around the spleen suggests SAA rupture).

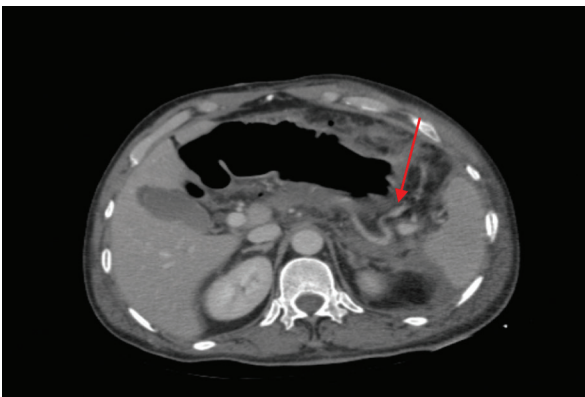


Figure 2 The appearance of an SAA is shown with a red arrow (axial plane) (the presence of fluid and fat stranding around the aneurysm and around the spleen suggests SAA rupture).

The aneurysm was then localized with a combination of microcatheter wires placed proximal to the celiac artery using a long introducer with a steerable tip, and the aneurysm and the adjacent splenic segment artery were embolized with coils of 7 × 150 mm, 5 × 150 mm, 5 × 8 mm, 6 × 11 mm, 4 × 10 mm, 3.5 × 8 mm, 3 × 10 mm, and 3 × 8 mm (Balt Extrusion, Montmorency, France). There were no complications, and splenic artery flow improved after the procedure (Figure 4). The patient, who underwent open surgery and subsequent interventional procedures, was followed in terms of fluid therapy,



Figure 3 The appearance of an SAA is shown with a red arrow (coronal plane) (the presence of fluid and fat stranding around the aneurysm and around the spleen suggests SAA rupture).

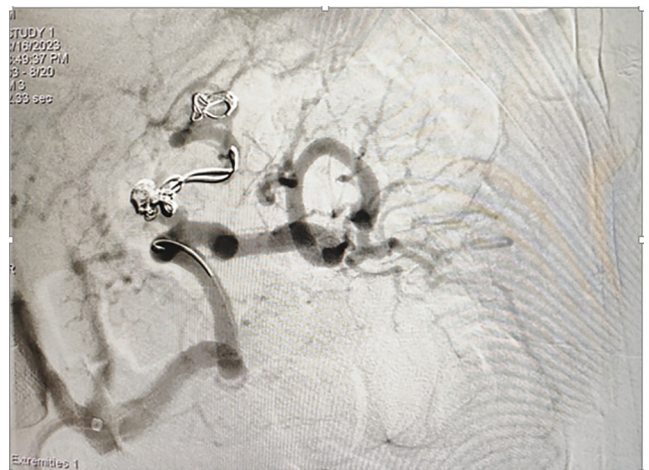


Figure 4 Coil embolization in the interventional procedure performed on the patient.

antibiotic therapy, and hemodynamic observation. The patient remained stable throughout the follow-up. After 14 days of hospitalization, outpatient follow-up was recommended and the patient was discharged.

Ethical Approval and Informed Consent

Ethics committee approval was not required for our case. Informed consent was obtained from the patient.

DISCUSSION

Although the exact etiology of SAAs is unknown, the most common pathologic finding is a defect of elastic fiber and smooth muscle loss in the tunica media.

Increased blood flow in the splenic artery seems to be a factor related to aneurysm development; therefore, these aneurysms are more common in fibromuscular dysplasia, portal hypertension, infection, congenital anomalies, liver transplant patients, and patients with pancreatic malignancy [3]. Especially during pregnancy, weakening of the vessel wall due to hormonal changes and increased splenic blood flow may predispose to aneurysm development [5]. It may be secondary to the above-mentioned causes or congenital. According to the International Registry of Acute Aortic Dissection (IRAD), women with vasculopathy are at risk of pregnancy-related vascular dissection. The presence of concomitant hypertension in pregnant patients with vasculopathy is seen as a risk factor for aneurysm development [6]. Our patient was diagnosed with SAA at a young age despite the absence of any predisposition and was subsequently investigated, but no risk factor was found. In a published case report, a 53-year-old woman who died of SAA without any risk factor was found to have possible atherosclerosis and fibromuscular dysplasia findings on histologic examination performed during an autopsy. It is also thought that α -1 antitrypsin deficiency may play a role in the etiology [7].

Patients with SAAs are frequently asymptomatic, but in 20% of cases, there are vague symptoms such as left upper quadrant pain, nausea, epigastric discomfort, and sometimes back pain [8,9]. In our case, the aneurysm manifested itself only with mild abdominal pain, which was present for 10 days, but after rupture, it led to hospitalization with hypotension and syncope.

Generally, aneurysms larger than 3 cm are rare. However, aneurysms larger than 10 cm have been reported in the literature. In aneurysms larger than 5 cm, the risk of rupture increases [9]. In our case, the aneurysm diameter was approximately 9 mm, and although the risk of rupture was low, the patient presented with a rupture clinic. In rupture, the sudden onset of severe abdominal pain, hypotension, or hypovolemic shock is observed and may lead to life-threatening outcomes. Rupture is the most fatal clinical condition in SAA, occurring in 3–9% of cases, and the mortality rate associated with rupture is reported to be up to 36%. Our patient was hypotensive on admission, and although rupture was detected, the patient, with a high amount of intra-abdominal hemorrhage, required prolonged follow-up (14 days). He was discharged from the hospital in good health after successful treatment.

Radiologic imaging methods are essential for the diagnosis. Calcifications can be seen in the left upper quadrant on direct abdominal radiographs. It may not always be possible to visualize the splenic artery with ultrasonography. Computed tomography and magnetic resonance imaging methods can be used to determine the localization, morphology, and other pathological

findings that may accompany the aneurysm. In the literature, it has been reported that aneurysms are most commonly seen in the middle 1/3 of the splenic artery [3]. In our case, the aneurysm was located in the middle 1/3 of the splenic artery, and computed tomography helped us in the diagnosis.

Although SAA is usually asymptomatic, it should be treated early as it can lead to complications such as rupture, which can result in mortality. Treatment is recommended when the aneurysm diameter is greater than 2 cm [10]. As pseudoaneurysms have a higher risk of rupture, they should be treated as soon as they are detected, regardless of their diameter. The ideal treatment modality is to maintain flow in the splenic artery and disable the aneurysm by protecting the spleen [3,5], although there are studies reporting that beta blocker agents may reduce the risk of rupture. In the treatment of SAA, depending on the localization of the aneurysm, splenic artery ligation by open surgery or laparoscopic method, ligation of the aneurysm, arterial reconstruction after partial or total aneurysmectomy, and/or splenectomy are planned [7,10].

In our case, these techniques were not applied because active bleeding was not detected at laparotomy. There are opinions advocating that treatment can be performed with percutaneous interventional techniques such as transcatheter embolization, drug-coated stent graft application, or percutaneous coil or thrombin injection after aneurysms are detected on CT angiography. In patients with high operative risk, and especially in patients with bleeding, effective results can be obtained in a short time with arterial embolization using a coil [5]. McDermott et al. reported a success rate of 85% with transcatheter arterial embolization in patients with SAA [11]. Similarly, Guillon et al. reported a 92% technical success rate in endovascular treatment with embolization and stenting in patients with SAA [12]. In practical applications, coil embolization should be the first endovascular method of choice in SAAs.

CONCLUSION

SAA and associated aneurysm rupture are rare yet fatal clinical conditions. Open surgery, laparoscopic surgery, and endovascular treatments are viable options for managing these patients. However, the mortality and morbidity rates associated with surgical interventions are notably high. Conversely, endovascular therapies are increasingly favored due to their less invasive nature, ease of performance, lower complication rates during the procedure, and higher success rates.

Endovascular methods should be prioritized, especially when the patient is within reach of a tertiary care center, when the primary care hospital lacks appropriate surgical expertise, or when transportation of the patient is challenging.

Acknowledgements

The study was performed at Mersin University Faculty of Medicine Hospital. The study has not been previously presented at any forum or meeting.

Ethics Statement

- (1) All the authors mentioned in the manuscript have agreed to authorship, read and approved the manuscript, and given consent for submission and subsequent publication of the manuscript.
- (2) The authors declare that they have read and abided by the JEVTM statement of ethical standards including rules of informed consent and ethical committee approval as stated in the article.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

Funding

The authors received no financial support for the research, authorship, and/or publication of this article.

REFERENCES

- [1] Pulli R, Dorigo W, Troisi N, Pratesi G, Innocenti AA, Pratesi C. Surgical treatment of visceral artery aneurysms: a 25-year experience. *J Vasc Surg.* 2008;48(2):334–42.
- [2] Madoff DC, Denys A, Wallace MJ, et al. Splenic arterial interventions: anatomy, indications, technical considerations, and potential complications. *Radiographics.* 2005;25 Suppl 1:S191–211.
- [3] Larraín D, Fava M, Espinoza R. Splenic artery aneurysm: case report. *Rev Med Chil.* 2005;133:943–6.
- [4] Pescarus R, Montreuil B, Bendavid Y. Giant splenic artery aneurysms: case report and review of the literature. *J Vasc Surg.* 2005;42(2):344–7.
- [5] Dereli Y, Gedik HS, Karaca OG, Ucak A, Yilmaz AT. Coil embolization of splenic artery aneurysm: case report. *Ulusal Vaskuler Cerrahi Dergisi.* 2014;23(3):202–6.
- [6] Braverman AC, Mittauer E, Harris KM, et al. Clinical features and outcomes of pregnancy-related acute aortic dissection. *JAMA Cardiol.* 2021;6(1):58–66.
- [7] Martin D, Teixeira Farinha H, Dattner N, Rotman S, Demartines N, Sauvain MO. Spontaneous non-traumatic splenic artery aneurysm rupture: a case report and review of the literature. *Eur Rev Med Pharmacol Sci.* 2018;22(10):3147–50.
- [8] Dolar E, Uslusoy H, Kiyici M, et al. Rupture of the splenic arterial aneurysm due to Behcet's disease. *Rheumatology (Oxford).* 2005;44(10):1327–8.
- [9] Agrawal GA, Johnson PT, Fishman EK. Splenic artery aneurysms and pseudoaneurysms: clinical distinctions and CT appearances. *AJR Am J Roentgenol.* 2007;188(4):992–9.
- [10] Hogendoorn W, Lavidá A, Hunink MG, et al. Open repair, endovascular repair, and conservative management of true splenic artery aneurysms. *J Vasc Surg.* 2014;60:1667–76.
- [11] McDermott VG, Shlansky-Goldberg R, Cope C. Endovascular management of splenic artery aneurysms and pseudoaneurysms. *Cardio Vasc Interv Radiol.* 1994;17(4):179–84.
- [12] Guillon R, Garcier JM, Aberqel A, et al. Management of splenic artery aneurysms and false aneurysms with endovascular treatment in 12 patients. *Cardio Vasc Interv Radiol.* 2003;26(3):256–60.