

Splenic Artery Aneurysm in a Patient With Extra Hepatic Portal Vein Obstruction

Khushboo kumari¹, Kundan Kumar², Raja Taha Yaseen Khan³, Danish Kumar⁴, Nida Rasool⁴, Ghazi Abrar⁴, Nasir Hasan Luck⁵

¹ Resident Medical Officer, Department of Hepatogastroenterology, Sindh Institute of Urology and Transplantation, Karachi, Pakistan

² Resident Medical Officer, Department of Nephrology, Sindh Institute of Urology and Transplantation, Karachi, Pakistan

³ Assistant Professor, Department of Hepatogastroenterology, Sindh Institute of Urology and Transplantation, Karachi, Pakistan

⁴ Senior Lecturer, Department of Hepatogastroenterology, Sindh Institute of Urology and Transplantation, Karachi, Pakistan

⁵ Professor, Department of Hepatogastroenterology, Sindh Institute of Urology and Transplantation, Karachi, Pakistan

*Corresponding author: Raja Taha Yaseen Khan, raja_taha101488@hotmail.com

"Cite this Article" Received: 02 January 2026; Accepted: 17 February 2026; Published: 28 February 2026

Author Contributions: Concept: RTYK, NHL; Design: RTYK, NR, GA; Data Collection: KHK, KUK, DK; Analysis: RTYK, DK, NR, GA, NHL; Drafting: KHK, KUK, RTYK, DK, NR, GA, NHL **Ethical Approval:** Sindh Institute of Urology and Transplantation. **Informed Consent:** Written informed consent was obtained from all participants;

Conflict of Interest: The authors declare no conflict of interest. **Funding:** No external funding; **Data Availability:** Available from the corresponding author on reasonable request; **Acknowledgments:** NA

ABSTRACT

Background: Splenic artery aneurysm (SAA) is the most frequently encountered visceral arterial aneurysm. Although portal hypertension is recognized as an associated condition and potential risk modifier, the coexistence of SAA with extrahepatic portal vein obstruction (EHPVO) is uncommon and poses diagnostic and therapeutic challenges. EHPVO is an important cause of non-cirrhotic portal hypertension and commonly manifests with splenomegaly, hypersplenism, gastroesophageal varices, and preserved hepatic synthetic function (1,2). **Case Presentation:** A 37-year-old woman with no established history of cirrhosis presented with recurrent upper gastrointestinal bleeding characterized by hematemesis and melena. Laboratory evaluation demonstrated pancytopenia, including hemoglobin of 7.6 g/dL, total leukocyte count of $2.54 \times 10^9/L$, and platelet count of 79,000/ μL , in the context of marked splenomegaly and portal hypertension. Liver biochemical tests were preserved. Upper gastrointestinal endoscopy demonstrated four columns of Grade II esophageal varices, small GOV2 gastric fundal varices, and mild portal hypertensive gastropathy. Histological assessment of the liver showed only mild portal inflammatory changes without cirrhosis or diagnostic features of autoimmune hepatitis, while transient elastography demonstrated F1–F2 fibrosis. Cross-sectional imaging showed features of extrahepatic portal hypertension, including dilated portal-systemic venous channels, extensive collaterals, and marked splenomegaly. A 2.6×2.3 cm splenic artery aneurysm was identified on contrast-enhanced CT and confirmed by CT angiography. No active aneurysmal rupture or contrast extravasation was seen. Importantly, imaging did not demonstrate definite splenic vein thrombosis attributable to the aneurysm; therefore, a causal relationship between the SAA and EHPVO could not be established. Surgical treatment with splenectomy and aneurysm excision was considered, but intervention was deferred after multidisciplinary assessment because of significant hematologic compromise and perceived procedural risk. Surveillance and reassessment were planned. **Conclusion:** This case demonstrates the unusual coexistence of a clinically relevant SAA with EHPVO and non-cirrhotic portal hypertension. The presence of both disorders should not automatically be interpreted as evidence that the aneurysm caused the portal venous obstruction. Detailed vascular imaging, characterization of portal venous anatomy, exclusion of cirrhosis, and multidisciplinary evaluation are essential. Treatment should be individualized according to aneurysm morphology, rupture-risk profile, portal hypertensive complications, hematologic status, and feasibility of endovascular or surgical intervention. **Keywords:** splenic artery aneurysm, extrahepatic portal vein obstruction, non-cirrhotic portal hypertension, hypersplenism, esophageal varices, gastrointestinal bleeding, splenomegaly

INTRODUCTION

Splenic artery aneurysm is the most commonly reported visceral arterial aneurysm and constitutes a substantial proportion of true visceral artery aneurysms encountered in clinical practice. Many SAAs are discovered incidentally because they remain asymptomatic until identified during abdominal imaging performed for an unrelated indication. Clinically important complications include aneurysmal expansion and rupture, the latter carrying potentially severe hemorrhagic consequences. Factors influencing management include symptoms, aneurysm size and growth, pregnancy or childbearing potential, portal hypertension, liver transplantation status, anatomical characteristics, and overall procedural risk (3,4).

Portal hypertension has long been recognized in association with SAA, particularly in patients with advanced liver disease or altered splenic and portal hemodynamics. Proposed mechanisms include

chronically increased splenic arterial flow, changes in vascular resistance, and structural degeneration of the arterial wall; nevertheless, the direction and strength of these associations vary according to the underlying portal hypertensive disorder (3,5). Contemporary vascular guidelines therefore emphasize individualized assessment rather than reliance on aneurysm diameter alone when deciding between surveillance and intervention (3).

Extrahepatic portal vein obstruction is a prehepatic cause of non-cirrhotic portal hypertension characterized by obstruction of the extrahepatic portal venous system, frequently related to chronic portal vein thrombosis and, in established cases, cavernomatous transformation and development of extensive portosystemic collaterals. Patients commonly retain relatively preserved hepatic synthetic function despite clinically significant portal hypertension and may present with recurrent variceal bleeding, splenomegaly, thrombocytopenia, and other manifestations of hypersplenism (1,2,6). Imaging is central to establishing the anatomical diagnosis and determining the extent of portal, splenic, and superior mesenteric venous involvement (1,6).

Coexistence of SAA and EHPVO is uncommon. Individual reports have described patients with EHPVO, portal cavernoma, splenomegaly, and one or more SAAs, but the underlying relationship may vary considerably between patients (7,8). In some circumstances, a large aneurysm may theoretically compress adjacent venous structures or coexist with splenic venous thrombosis; in others, the aneurysm may develop secondary to chronic portal-hypertensive hemodynamic alterations or may simply coexist as an incidental vascular abnormality. Consequently, causality should not be inferred unless an anatomical or thrombotic mechanism is demonstrated.

We report a 37-year-old woman with recurrent variceal gastrointestinal bleeding, marked splenomegaly and hypersplenism in the setting of EHPVO and preserved hepatic function, in whom CT angiography additionally demonstrated a 2.6×2.3 cm SAA. The case is presented according to the CARE case-report framework, with emphasis on clinical chronology, diagnostic reasoning, vascular imaging, therapeutic decision-making, and the limitations of establishing causality from coexistence alone (9,10).

CASE PRESENTATION

Patient Information

A 37-year-old woman presented with recurrent episodes of upper gastrointestinal bleeding consisting of hematemesis and melena. She had no previously established diagnosis of cirrhosis or other advanced chronic liver disease. The recurrent nature of bleeding prompted evaluation for portal hypertension and an underlying hepatic or vascular cause.

No evidence of chronic viral hepatitis was identified during the subsequent diagnostic evaluation. Available information did not demonstrate an established prior diagnosis explaining her portal hypertension before the current work-up.

Clinical Findings

At presentation, the patient was hemodynamically stable. Abdominal examination revealed no reported tenderness or distension. Cardiovascular, respiratory, and neurological examinations were unremarkable.

The overall clinical pattern was dominated by recurrent portal-hypertensive gastrointestinal bleeding and later-supported hypersplenism. EHPVO and other non-cirrhotic causes of portal hypertension can produce significant gastroesophageal varices and splenomegaly despite relatively preserved liver biochemical and synthetic function (1,2).

Diagnostic Assessment

Initial hematologic assessment demonstrated pancytopenia, with hemoglobin of 7.6 g/dL, total leukocyte count of $2.54 \times 10^9/L$, and platelet count of 79,000/ μL . In the setting of marked splenomegaly and portal hypertension, these abnormalities were considered compatible with hypersplenism. However, hypersplenism is a clinicopathological interpretation rather than a laboratory diagnosis in isolation, and other hematologic causes should be excluded according to the clinical context (1,2).

Liver function tests were reported as normal, arguing against advanced hepatic synthetic dysfunction. Viral screening showed non-reactive hepatitis B surface antigen and anti-hepatitis C virus antibodies.

Autoimmune evaluation demonstrated a positive antinuclear antibody at a titer of 1:160 and an immunoglobulin G level of 16 g/L. Anti-smooth muscle antibody and anti-liver-kidney microsomal-1 antibody were negative. Because serological abnormalities alone do not establish autoimmune hepatitis, hepatic histology was undertaken in the context of the broader diagnostic work-up.

Liver biopsy demonstrated moderately expanded portal tracts with predominantly lymphocytic inflammatory infiltration but no reported interface hepatitis or significant plasma-cell-rich infiltrate. The modified Hepatitis Activity Index was 2/6. These findings did not support active autoimmune hepatitis and did not demonstrate cirrhosis. Transient elastography showed F1–F2 fibrosis, providing additional evidence against advanced cirrhosis. Mild or moderate fibrosis can coexist with non-cirrhotic portal hypertension and should not itself be interpreted as cirrhosis (2,6).

Upper gastrointestinal endoscopy demonstrated four columns of Grade II esophageal varices, without red wale signs, together with small GOV2 fundal varices and mild portal hypertensive gastropathy. These findings established clinically significant portal hypertension and provided a plausible source for the patient's recurrent upper gastrointestinal bleeding. Gastroesophageal varices are among the most characteristic clinical consequences of chronic EHPVO (1,2). Abdominal ultrasonography demonstrated marked splenic enlargement and features of portal hypertension. A reported splenic measurement of 79 mm was documented in the available record; however, because the imaging plane and measured splenic dimension were not specified, this value should not be interpreted as a standard craniocaudal splenic length. Cross-sectional CT more clearly demonstrated substantial splenic enlargement.

Contrast-enhanced CT of the abdomen showed features of portal hypertension, including enlargement of the portal-systemic venous circulation, dilatation of the splenic and superior mesenteric venous systems, extensive portosystemic collateral vessels, and marked splenomegaly. The hepatic veins and inferior vena cava were reported as patent and morphologically normal, reducing the likelihood of a post-hepatic obstructive process. An additional vascular abnormality was identified: a 2.6×2.3 cm splenic artery aneurysm. On axial contrast-enhanced imaging, the lesion appeared as a rounded, intensely enhancing vascular structure associated with the splenic arterial circulation. CT angiography subsequently confirmed the aneurysm and demonstrated its relationship to the adjacent splanchnic vasculature (Figure 1).

No active contrast extravasation was demonstrated, and there was no radiological evidence of acute aneurysmal rupture. Importantly, the available imaging description did not demonstrate definite splenic vein thrombosis or direct venous compression attributable to the aneurysm. For that reason, the SAA was considered to coexist with EHPVO rather than being definitively established as its cause. The imaging study also reported multiple regenerative-appearing hepatic nodules. Because regenerative nodular change may occur in vascular liver disorders and non-cirrhotic portal hypertension, these findings were interpreted in conjunction with the preserved liver function, low fibrosis stage, and biopsy findings rather than as radiological evidence of cirrhosis (2,6).

A subsequent upper gastrointestinal endoscopy demonstrated three columns of Grade I esophageal varices, healed mucosal ulceration, and no active source of bleeding. The apparent reduction in variceal grade should be interpreted in the context of interval endoscopic management and clinical evolution;

the available source information does not permit precise attribution of this change to a specific intervention.

Differential Diagnostic Considerations

The principal diagnostic problem was to differentiate cirrhotic portal hypertension from non-cirrhotic portal hypertension caused by a vascular obstruction. Cirrhosis was considered less likely because liver biochemical tests were preserved, biopsy showed no cirrhotic architecture, inflammatory activity was minimal, and transient elastography showed only F1–F2 fibrosis. The combination of significant portal hypertensive manifestations with comparatively preserved hepatic parenchyma is typical of prehepatic or presinusoidal non-cirrhotic portal hypertension (1,2).

Autoimmune hepatitis was also considered because of the positive ANA and modestly elevated IgG. However, negative ASMA and anti-LKM-1 antibodies, the absence of interface hepatitis and plasma-cell-rich inflammation on biopsy, minimal histological activity, and lack of advanced fibrosis did not support autoimmune hepatitis as the cause of the portal hypertension. The remaining findings favored an extrahepatic vascular mechanism. EHPVO is typically diagnosed by demonstrating chronic obstruction of the extrahepatic portal vein, frequently with collateralization or portal cavernoma (1,6). The present case was clinically and radiologically managed as EHPVO; however, precise reporting of the site and extent of portal vein obstruction, portal-vein patency, presence or absence of cavernomatous transformation, Doppler flow direction, and involvement of the superior mesenteric and splenic veins would further strengthen anatomical confirmation and should be provided from the original radiological report if available. The SAA represented an additional diagnostic issue. Although SAA has been described in patients with portal hypertension and EHPVO, coexistence does not establish causality (7,8). As no definite aneurysm-related splenic vein thrombosis, direct compression, or other specific mechanism was identified, the most defensible interpretation was SAA associated with, rather than proven to have caused, EHPVO.

Timeline

Clinical stage	Findings and actions
Initial presentation	Recurrent hematemesis and melena in a 37-year-old woman without known cirrhosis
Initial laboratory assessment	Hemoglobin 7.6 g/dL, leukocyte count $2.54 \times 10^9/L$, platelets 79,000/ μL ; preserved liver biochemical profile
Etiological work-up	HBsAg and anti-HCV negative; ANA 1:160 and IgG 16 g/L; ASMA and anti-LKM-1 negative
Initial endoscopy	Four Grade II esophageal variceal columns, small GOV2 fundal varices, mild portal hypertensive gastropathy
Liver assessment	Liver biopsy: mild portal lymphocytic inflammation, no interface hepatitis/plasma-cell infiltrate, HAI 2/6; FibroScan: F1–F2 fibrosis
Ultrasound/CT assessment	Splenomegaly, portal-hypertensive collateralization and enlarged splanchnic venous structures
Incidental vascular finding	2.6 × 2.3 cm splenic artery aneurysm identified on contrast-enhanced CT
CT angiography	SAA confirmed; no active extravasation; portal-hypertensive changes and marked splenomegaly demonstrated
Repeat endoscopy	Three Grade I esophageal variceal columns, healed ulcers, no active bleeding
Multidisciplinary review	Splenectomy with aneurysm excision considered; intervention deferred because of hematologic compromise and procedural risk
Planned follow-up	Conservative surveillance with reassessment after approximately 3 months

Exact calendar dates were not available in the source clinical information; the timeline is therefore presented according to clinical sequence in accordance with CARE reporting principles (9,10).

Therapeutic Intervention

The case was reviewed multidisciplinary because two clinically relevant problems required simultaneous consideration: chronic portal hypertension with recurrent variceal bleeding and a 2.6-cm SAA. The aneurysm diameter was clinically important. Contemporary Society for Vascular Surgery guidance recommends treatment decisions according to aneurysm type, size, symptoms, growth, pregnancy-related considerations, associated portal hypertension, and procedural feasibility rather than applying an indiscriminate threshold to all visceral aneurysms (3). Earlier literature frequently used 2 cm as an

intervention threshold, whereas current guideline-based decision-making for true SAA is more nuanced and incorporates patient-specific rupture risk and anatomical suitability (3–5).

Open operative management, including splenectomy with aneurysm excision, was considered. However, the patient had significant pancytopenia, including severe anemia and thrombocytopenia, in association with marked splenomegaly and portal hypertension. These factors were considered to increase operative and perioperative risk.

Following multidisciplinary discussion, immediate surgery was therefore deferred. The patient was managed conservatively with planned close clinical and imaging surveillance and reassessment approximately three months later.

An important limitation of the original therapeutic documentation is that the reason for not performing an endovascular procedure, such as coil embolization, plug embolization, or covered stent exclusion, was not specified. Current practice increasingly uses endovascular treatment for anatomically suitable SAAs because it avoids laparotomy and splenectomy in selected patients, although feasibility depends strongly on aneurysm location, arterial tortuosity, neck morphology, collateral supply, available expertise, and the requirement to preserve splenic perfusion (3,11,12). This consideration should therefore be explicitly documented if information from the multidisciplinary discussion is available.

Similarly, the source material did not provide sufficient detail regarding the exact acute and secondary prophylactic treatment of the patient's variceal bleeding. Management of EHPVO-related gastroesophageal varices generally includes endoscopic therapy and individualized pharmacological or portal-decompressive strategies, while portal venous recanalization, surgical shunting, or other interventional techniques may be considered according to anatomy and recurrent complications (1,2,13). These details should be added if documented in the patient's medical record.

Follow-Up and Outcomes

At the time represented by the available clinical data, the patient was hemodynamically stable and had no evidence of active aneurysmal rupture or ongoing gastrointestinal hemorrhage. Repeat endoscopy demonstrated lower-grade esophageal varices and no active bleeding source.

Because definitive aneurysm treatment had been deferred, close clinical and radiological surveillance was planned, with reassessment after approximately three months. Surveillance was intended to evaluate aneurysm stability, recurrent gastrointestinal bleeding, hematologic status, splenic enlargement, and the feasibility of delayed endovascular or operative intervention.

Longer-term follow-up data were not available in the supplied record. In accordance with the CARE guideline, the final submitted report would be strengthened by adding, if available, the patient's subsequent clinical status, recurrent bleeding events, interval hemoglobin and platelet counts, repeat CT/CTA aneurysm diameter, aneurysm growth or stability, and whether definitive vascular or portal-decompressive treatment was subsequently performed (9,10).

DISCUSSION

This case illustrates the uncommon coexistence of SAA and EHPVO in a young woman with recurrent gastroesophageal variceal bleeding, marked splenomegaly, pancytopenia compatible with hypersplenism, and preserved hepatic parenchymal function. The diagnostic importance of the case lies in distinguishing a vascular cause of portal hypertension from cirrhosis and, separately, in avoiding an unsupported assumption that the SAA itself caused the extrahepatic portal venous obstruction.

EHPVO is an important cause of non-cirrhotic portal hypertension, particularly among children and young adults in regions where vascular and prothrombotic causes of portal hypertension remain prevalent. The obstruction occurs at a prehepatic level, and chronic disease may result in cavernomatous

venous transformation and extensive portosystemic collateralization. Because hepatic parenchymal function is usually preserved, patients may remain clinically compensated except for complications such as variceal hemorrhage, massive splenomegaly, hypersplenism, portal biliopathy, and, in selected cases, thrombosis extending into the splenic or superior mesenteric venous circulation (1,2,6,13).

The patient's clinical phenotype was consistent with this pattern. She had recurrent hematemesis and melena, gastroesophageal varices, portal hypertensive gastropathy, marked splenic enlargement, and pancytopenia despite relatively preserved liver biochemical tests and absence of cirrhosis on biopsy. The mild F1–F2 fibrosis seen on transient elastography does not conflict with a diagnosis of non-cirrhotic portal hypertension because the defining distinction is the absence of established cirrhosis rather than the complete absence of fibrosis (2,6).

The positive ANA and elevated IgG represented a potentially distracting component of the diagnostic work-up. Autoimmune serological abnormalities can be encountered without meeting diagnostic criteria for autoimmune hepatitis. In this patient, the absence of interface hepatitis and plasma-cell-rich inflammatory activity, very low histological activity, preserved liver function, negative ASMA and anti-LKM-1 antibodies, and lack of advanced fibrosis made autoimmune hepatitis an unlikely explanation for the portal hypertension. The case therefore demonstrates the importance of integrating serological, histological, imaging, and clinical findings rather than assigning causality to an isolated positive antibody test.

The second major finding was the 2.6×2.3 cm SAA. SAA is the most frequently encountered visceral arterial aneurysm, and portal hypertension is a recognized associated condition. Chronic increases in splenic arterial blood flow and altered splanchnic hemodynamics have been proposed as mechanisms contributing to aneurysm development in patients with portal hypertension (3–5). This raises an important alternative causal model: instead of the SAA producing portal hypertension, pre-existing portal hypertension may have contributed to the development or enlargement of the SAA.

A particularly relevant report by Pathak et al. described SAA associated with extrahepatic portal venous obstruction, emphasizing both the rarity of the combination and the complexity of determining the direction of causality (7). More recently, Sharma et al. described a young woman with chronic extrahepatic portal vein thrombosis, portal hypertension, splenomegaly, thrombocytopenia, esophageal varices, and multiple large SAAs who ultimately underwent splenic artery embolization, portosystemic shunting, and splenectomy (8). These cases illustrate that SAA can occur in the hemodynamic environment created by chronic extrahepatic portal hypertension and may require combined vascular and portal-hypertensive management.

Earlier case literature has also proposed venous compression or splenic venous thrombosis secondary to large aneurysms as a mechanism of segmental or extrahepatic portal hypertension. However, such mechanisms should only be invoked when demonstrated by imaging. In the present patient, no definite splenic venous thrombosis attributable to the aneurysm was described and no direct aneurysmal compression of the portal venous system was established. Consequently, the original assertion that the aneurysm caused EHPVO has been intentionally removed. The case is more accurately characterized as SAA coexisting with EHPVO.

This distinction is clinically relevant because it changes both interpretation and management. If the aneurysm were directly obstructing a major portal venous channel, treatment of the aneurysm might theoretically address an etiological component of the portal hypertension. By contrast, when EHPVO is chronic and anatomically independent of the aneurysm, treatment of the SAA addresses rupture risk but does not necessarily correct the underlying portal hypertension. Chronic EHPVO may require separate endoscopic, portal-recanalization, TIPS-related, or surgical-shunt strategies when clinically indicated (1,2,13).

Management of SAA has evolved substantially. Historically, asymptomatic aneurysms larger than approximately 2 cm were frequently offered intervention. The 2020 Society for Vascular Surgery guideline provides more condition-specific recommendations and emphasizes aneurysm size, growth, symptoms, rupture, pregnancy considerations, pseudoaneurysm status, comorbidity, and anatomy (3). Consequently, the statement that all SAAs larger than 2 cm require treatment is overly simplistic and should be avoided.

Nevertheless, the patient's aneurysm deserves careful consideration because of several contextual factors. She was relatively young, the aneurysm measured approximately 2.6 cm, and she had significant portal hypertension. Portal hypertension is frequently regarded as an important consideration when estimating aneurysm-associated risk, especially in patients undergoing liver transplantation or those with substantial alterations in splenic arterial flow (3–5). Conversely, her severe anemia, thrombocytopenia, leukopenia, extensive portal collateralization, and marked splenomegaly increased the complexity of conventional surgery.

The original surgical proposal of splenectomy with aneurysm excision is understandable, particularly if the aneurysm was distal or located near the splenic hilum. However, contemporary endovascular techniques merit explicit consideration. Coil embolization, vascular plugs, parent-artery embolization, and covered stent exclusion can provide effective aneurysm control in appropriately selected patients. Recent observational data demonstrate high technical success for endovascular treatment, although splenic infarction, post-embolization syndrome, endoleak, arterial dissection, and need for reintervention can occur (11,12). A 2025 series reported successful endovascular exclusion in the large majority of treated patients, supporting the feasibility of such approaches when anatomy is favorable (12).

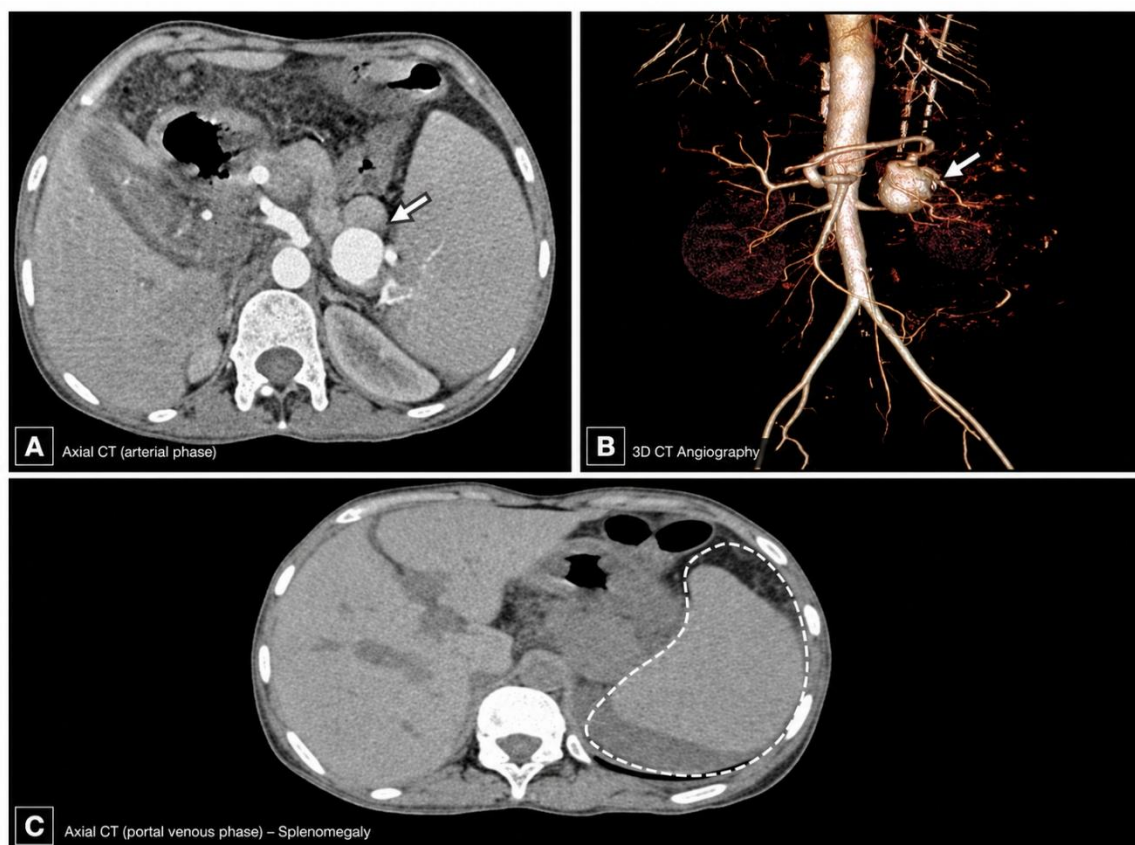


Figure 1. Composite CT and CT angiographic findings. (A) Axial contrast-enhanced CT demonstrating a 2.6 × 2.3 cm splenic artery aneurysm (arrow). (B) Three-dimensional CT angiographic reconstruction showing the aneurysm and its relationship to the adjacent splanchnic vasculature (arrow). (C) Axial abdominal CT demonstrating marked splenomegaly in the setting of extrahepatic portal vein obstruction and portal hypertension.

The decision to manage this patient conservatively should therefore be interpreted as an individualized risk-benefit decision rather than as a general management recommendation for an aneurysm of this size. Ideally, the report should specify whether interventional radiology reviewed the aneurysm anatomy, whether embolization or stent-graft exclusion was technically feasible, whether the patient declined intervention, and what surveillance protocol was selected. Without these data, the reasoning for conservative management remains incompletely documented.

The severe cytopenias are also clinically relevant. Hypersplenism secondary to chronic portal hypertension frequently produces thrombocytopenia and leukopenia through increased splenic sequestration, while recurrent gastrointestinal blood loss may contribute to anemia (1,2). However, describing pancytopenia as definitively caused by hypersplenism would require exclusion of other hematological causes. The revised report therefore uses the more appropriate wording “compatible with” or “consistent with hypersplenism.”

The regenerative-appearing hepatic nodules identified on CTA also warrant cautious terminology. Alterations of hepatic perfusion in vascular liver disease can produce regenerative changes in the absence of cirrhosis. Such nodules should not automatically be equated with cirrhotic nodularity, particularly when biopsy and transient elastography do not demonstrate advanced fibrosis. Current concepts of non-cirrhotic portal hypertension emphasize precisely this potential discordance between severe portal hypertensive manifestations and relatively preserved hepatic architecture (2,6).

Another limitation is incomplete anatomical description of the portal venous obstruction. A high-quality report of EHPVO should identify whether the main portal vein was narrowed, chronically thrombosed, or completely occluded; whether cavernomatous transformation was present; whether the splenic and superior mesenteric veins remained patent; and the direction and velocity of portal flow on Doppler ultrasonography. This information has direct implications for both diagnostic certainty and potential portal-vein recanalization or shunt procedures (1,6,13). These details should therefore be retrieved from the original imaging archive if possible before final publication.

The clinical implications of the present case are consequently twofold. First, severe portal hypertension in a patient with preserved liver function should prompt careful evaluation for non-cirrhotic vascular causes rather than presumptive labeling as cirrhosis. Second, discovery of an SAA in a patient with EHPVO should trigger detailed vascular characterization, but an etiological relationship should only be claimed when direct anatomical evidence supports it. This distinction reduces diagnostic overstatement and facilitates rational multidisciplinary planning.

Strengths and Limitations

The principal strength of this report is the documentation of an unusual vascular association in a patient with clinically significant non-cirrhotic portal hypertension. The case includes endoscopic findings, hepatic histology, transient elastography, CT, CT angiography, hematologic findings, and multidisciplinary therapeutic assessment, allowing relatively broad diagnostic correlation.

Several limitations remain. First, the exact anatomical morphology of the extrahepatic portal vein obstruction—including portal vein patency, the presence or absence of cavernomatous transformation, Doppler flow characteristics, and the extent of portomesenteric involvement—was not completely specified in the available data. Second, the precise anatomical segment, morphology, neck characteristics, mural thrombus status, and calcification of the SAA were not documented. Third, the rationale for choosing surveillance rather than an endovascular intervention was incompletely recorded. Fourth, details of acute and secondary prophylaxis for variceal bleeding were unavailable. Finally, long-term follow-up after the proposed three-month reassessment was not provided. These limitations prevent firm conclusions regarding causality and long-term outcome.

Patient Perspective

A patient-perspective statement was not available in the clinical information supplied for preparation of this report. If the patient provides a perspective regarding recurrent bleeding, treatment options, concern regarding surgery, or surveillance, it should be incorporated before submission in accordance with CARE recommendations (9,10).

Informed Consent

Written informed consent for publication of the clinical details and radiological images should be confirmed and documented before journal submission. The final manuscript should state: "Written informed consent was obtained from the patient for publication of this case report and the accompanying anonymized clinical images."

CONCLUSION

Splenic artery aneurysm may coexist with EHPVO and clinically significant non-cirrhotic portal hypertension. In the present case, recurrent variceal bleeding, marked splenomegaly, pancytopenia compatible with hypersplenism, preserved liver biochemical function, mild hepatic fibrosis, and absence of cirrhosis supported a predominantly vascular cause of portal hypertension. CT angiography additionally identified a 2.6 × 2.3 cm SAA.

Importantly, the available imaging did not demonstrate direct aneurysmal compression or splenic venous thrombosis sufficient to establish that the SAA caused EHPVO. The relationship should therefore be described as an association rather than a proven causal mechanism. Detailed characterization of portal venous anatomy and aneurysm morphology is essential because each condition may require separate therapeutic consideration.

Although splenectomy with aneurysm excision was considered, significant hematologic compromise contributed to a decision for surveillance. Contemporary management should also consider endovascular exclusion when technically feasible. Multidisciplinary assessment involving hepatology, gastroenterology, vascular surgery, interventional radiology, and hematology is particularly valuable when the rupture risk of an SAA must be balanced against portal-hypertensive and hematologic procedural risks.

REFERENCES

1. Khanna R, Sarin SK. Idiopathic portal hypertension and extrahepatic portal venous obstruction. *Hepatol Int.* 2018;12(Suppl 1):148-167. doi:10.1007/s12072-018-9844-3.
2. Gioia S, Nardelli S, Ridola L, Riggio O. Causes and management of non-cirrhotic portal hypertension. *Curr Gastroenterol Rep.* 2020;22:56. doi:10.1007/s11894-020-00792-0.
3. Chaer RA, Abularrage CJ, Coleman DM, Eslami MH, Kashyap VS, Rockman C, et al. The Society for Vascular Surgery clinical practice guidelines on the management of visceral aneurysms. *J Vasc Surg.* 2020;72(1S):3S-39S. doi:10.1016/j.jvs.2020.01.039.
4. Abbas MA, Stone WM, Fowl RJ, Gloviczki P, Oldenburg WA, Pairolero PC, et al. Splenic artery aneurysms: two decades experience at Mayo Clinic. *Ann Vasc Surg.* 2002;16(4):442-449.
5. Keschenau P, Kaisaris N, Jalaie H, Grommes J, Kotelis D, Kalder J, et al. Management strategies for true and dissecting visceral artery aneurysms. *J Cardiovasc Surg (Torino).* 2020;61. doi:10.23736/S0021-9509.19.11036-1.
6. Meena B, Rudra OS, Sharma D, Sarin SK. Noncirrhotic portal hypertension: current trends and future directions. *Curr Opin Gastroenterol.* 2025. doi:10.1097/MOG.0000000000001106.
7. Pathak N, Poddar E, Shrestha S, Kumari K, Ranjan R, Sharma D, et al. Splenic artery aneurysm with extrahepatic portal venous obstruction: a case report. *Int J Surg Case Rep.* 2022;99:107568. doi:10.1016/j.ijscr.2022.107568.

8. Sharma V, Gatmaitan R, Neale M, Samra JS. Management of multiple splenic artery aneurysms in the setting of portal hypertension and splenomegaly. *BMJ Case Rep.* 2025;18. doi:10.1136/bcr-2024-260823.
9. Gagnier JJ, Kienle G, Altman DG, Moher D, Sox H, Riley D; CARE Group. The CARE guidelines: consensus-based clinical case reporting guideline development. *J Med Case Rep.* 2013;7:223. doi:10.1186/1752-1947-7-223.
10. Riley DS, Barber MS, Kienle GS, Aronson JK, von Schoen-Angerer T, Tugwell P, et al. CARE guidelines for case reports: explanation and elaboration document. *J Clin Epidemiol.* 2017;89:218-235. doi:10.1016/j.jclinepi.2017.04.026.
11. Rinaldi LF, Brioschi C, Marone EM. Endovascular and open surgical treatment of ruptured splenic artery aneurysms: a case report and systematic literature review. *J Clin Med.* 2023;12(18):6085. doi:10.3390/jcm12186085.
12. Zhang X, Wang M, Li M, He H, Yang C, Shu C. Technical and clinical outcomes of endovascular treatment for splenic artery aneurysms. *BMC Surg.* 2025. doi:10.1186/s12893-025-03422-6.
13. Patel RK, Tripathy T, Sasidharan R, Gupta S, Nayak H, Pattnaik B, et al. Endovascular management of extrahepatic portal vein obstruction (EHPVO): expanding beyond surgical shunts. *Abdom Radiol (NY).* 2026. doi:10.1007/s00261-026-05695-8.