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Case Report

An incidental finding of splenic vein aneurysm in 59-year-old woman with systemic lupus erythematosus

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ABSTRACT

This case report discusses a rare presentation of a splenic vein aneurysm (SVA) incidentally discovered in a 59-year-old female patient with a history of systemic lupus erythematosus (SLE), interstitial lung disease (ILD), and chronic liver disease (CLD). The patient presented with symptoms of cough and breathlessness, leading to an abdominal ultrasound, which revealed a cystic lesion in the distal pancreas with a venous waveform. Further evaluation through contrast-enhanced computed tomography (CECT) confirmed the diagnosis of SVA, showing a saccular outpouching from the splenic vein without thrombosis or extravasation. Given the patient's asymptomatic status, conservative management and follow-up were advised. SVA is an uncommon entity with potential complications, including thrombosis and rupture. This report adds to the limited literature on SVA, particularly in patients with connective tissue disorders like SLE and conditions of portal hypertension. It underscores the need for further research to establish clearer guidelines for the management and treatment of such aneurysms.

Keywords: Splenic vein aneurysm, SLE systemic lupus erythematous, CLD chronic liver disease, ILD

INTRODUCTION

The splenic vein originates from the hilum of the spleen, located in the upper left quadrant of the abdomen. It runs horizontally, posterior to the pancreas, and merges with the superior mesenteric vein to form the portal vein, which leads to the liver.

SVA is an extremely uncommon condition with only few cases having been reported. It is a rare vascular anomaly with limited documented cases, often discovered incidentally, and its clinical significance, natural history, and optimal management remain areas of ongoing research. A study shows inflammatory changes due to pancreatitis is the most likely cause of aneurysm formation.[1] Peripheral calcifications in venous aneurysms is noted with unknown cause.

Most splenic vein aneurysms are asymptomatic and are usually incidental findings. It causes rupture, thrombosis, or compression of adjacent structures.[2] The mechanism of development is not well understood. Etiology may include inherent weakness of the vessel wall or trauma, inflammation associated with causes by conditions like pancreatitis, CLD, or portal hypertension considered as probable causes.[3]

CASE REPORT

A 59-year-old woman, known case of SLE with associated ILD and CLD came to hospital with complaints of cough and breathlessness. In view of long-term therapy of methotrexate, patient was undergoing routine abdominal imaging as part of her follow-up evaluation. She had no specific gastrointestinal complaints, abdominal pain, or

signs of portal hypertension. Laboratory tests showed mild liver dysfunction but no significant coagulopathy.

USG showed features of CLD with PHTN and a well-defined cystic lesion (3×4 cm) in distal body of pancreas shows venous waveform (Figure A and B) on doppler.

For further characterisation, CECT abdomen was done: finding include CLD with features of PHTN i. e.; multiple collaterals and splenomegaly. A well-defined cystic lesion in distal body of pancreas abutting splenic vein and artery. On contrast arterial phase, lesion shows no enhancement, communication with splenic artery (Figure 2 A and B). In venous and delayed phase, the lesion appears saccular outpunching from splenic vein with contrast filling (Figure 2 C and D). Features consistent with SVA. No evidence of contrast extravasation, no thrombosis noted within the lumen. Adjacent pancreas shows normal parenchymal enhancement also noted.

As the patient had no signs of thrombosis, rupture, or compression of adjacent structures. Given the absence of symptoms and the stable nature of the aneurysm, a conservative approach with periodic imaging follow-up was recommended. The patient remained asymptomatic with no progression in aneurysm size or complications, supporting the role of surveillance over interventional management in select cases.

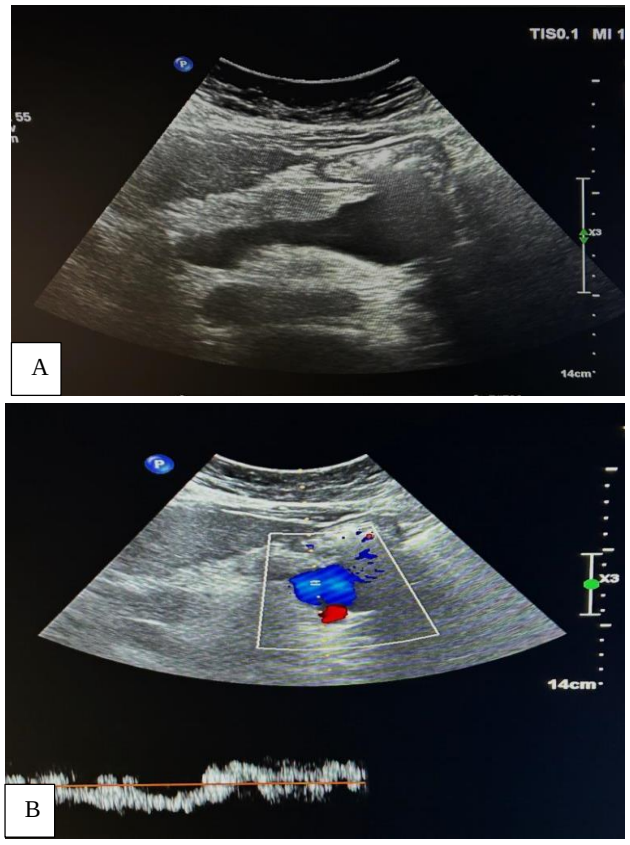

Figure 1 (A and B): USG: A-showing cystic lesion in pancreas. B-cystic lesion showing venous waveform in doppler.

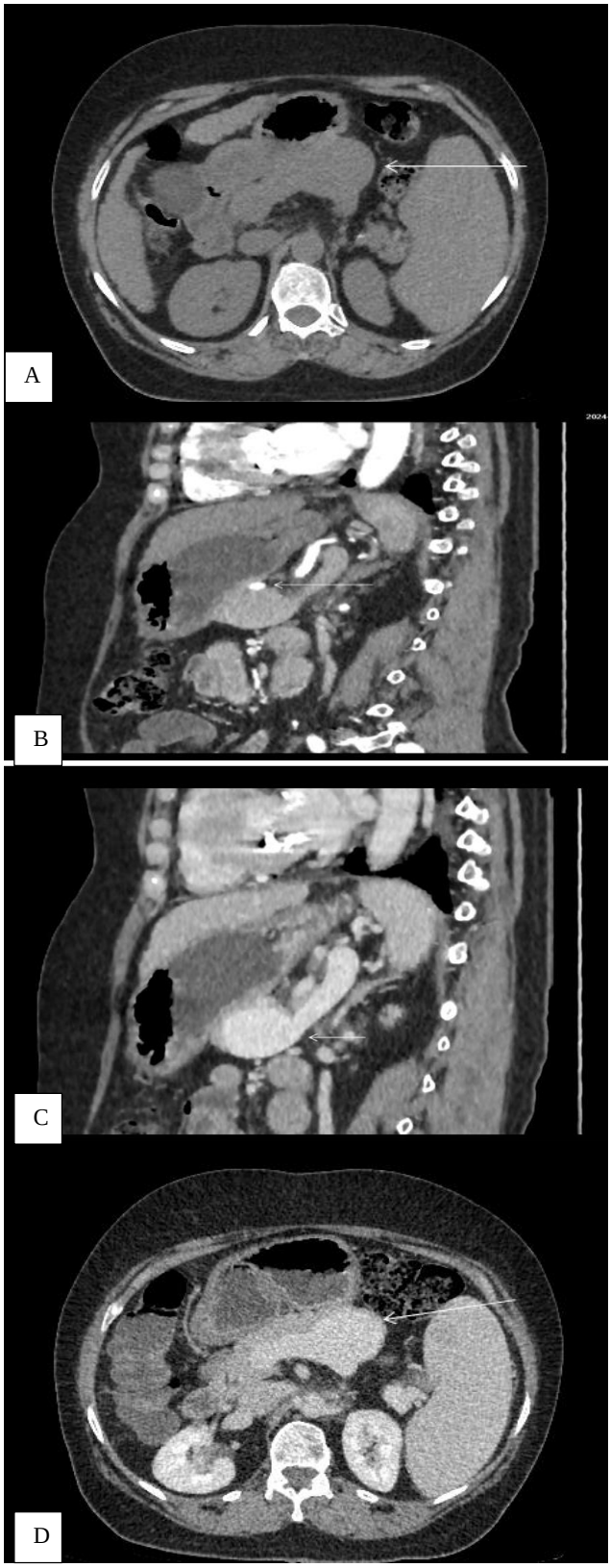

Figure 2 (A-D): CECT A-(plain axial section) of cystic lesion noted in distal body of pancreas. B-(arterial phase sagittal section) No arterial phase enhancement. C and D-(venous and delayed phase sagittal section) lesion shows venous phase enhancement and communication with splenic vein.

DISCUSSION

Abdominal venous aneurysms are rare. In the portal venous system, they could arise from the extra- or intrahepatic portion of the portal vein or splenic vein. Extrahepatic portal venous aneurysm and SVA is an uncommon condition. The etiology of SVA is thought to be due to abnormal weakness of the venous wall which can be due to congenital causes, secondary to PHTN, localised inflammation.[4]

A portal vein aneurysm (PVA) is the abnormal focal saccular or fusiform dilatation of the portal venous system, and it is defined as a PV diameter exceeding 19 mm in cirrhotic patients and 15 mm in a normal liver. It is a rare vascular abnormality, representing 3% of all venous aneurysms in the human body. The location of a PVA can be extrahepatic or intrahepatic. Extrahepatic PVAs often occur in the main trunk of the PV, the splenomesenteric confluence, at the level of the PV bifurcation, the main branches of the PV, the splenic vein (SV) and the superior mesenteric vein (SMV).[5]

Liebowitz and Rousselot believed that a congenital or developmental mechanism was responsible for the condition. This theory claims that a congenitally weakened area of the vessel wall may become dilated under normal pressure of the portal venous system, similar to arterial aneurysm formation in branching sites.[6]

The pressure within the portal system depends on the resistance to blood flow and volume of blood entering the system. Most forms of splenic, pancreatic, small intestinal, gastric, and colonic inflammatory diseases are associated with increased portal blood flow. Therefore, portal system aneurysm seems unlikely to be the result only of PHTN, even if long standing.

We believe that in our case, Connective tissue disorder, SLE and associated CLD resulted in weakening of the walls of the splenic vein with increased PHTN led to SVA. Patient had no acute aneurysm-related symptoms. Reported complications include thrombosis and rupture of aneurysms.

Management of SVAs can be conservative with serial non-invasive imaging versus plication and aneurysm excision.

When excision of SVA is difficult, a decompression procedure may be the best option to drain the aneurysm associated with portal hypertension.

In symptomatic aneurysms and presence of complications are sound indications for intervention. Surgery has been the standard treatment of portal aneurysms and includes aneurysmorrhaphy, aneurysmectomy, splenectomy, spleno-renal shunt, and distal pancreatectomy. Kwon et al reported the first endovascular treatment in a traumatic SVA through trans splenic approach using a Viabahn stent.[3] Evidence indicates that rupture risk is low due to low portal venous pressure; however, risk is increased in the presence of portal hypertension. No defined management guidelines currently exist.[7]

CONCLUSION

SVA are rare. They can be congenital or acquired. They are often associated with connective tissue disorder, cirrhosis and PHTN, and their presentation includes abdominal pain and other non-specific symptoms, or discovered incidentally. Watchful waiting is an appropriate treatment, except when complications occur. Most common complications include thrombosis and rupture.

Because the incidence of these aneurysms is low, the exact indication for intervention and the type of treatment is not well defined. There is currently no standardized procedure for the management of low-risk visceral venous aneurysms, highlighting the need for further research to establish optimal guidelines for diagnosis, monitoring, and intervention. Hence reporting this case will add data for a development of effective guideline in future.

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