

Transarterial Embolisation of a Giant Splenic Artery Pseudoaneurysm: A Case Report

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ABSTRACT

Giant splenic artery pseudoaneurysm (PSA) is a rare abnormal outpouching from the arterial wall, measuring more than 5 cm in diameter. It is highly prone to rupture and can cause life-threatening haemorrhage and thus, treatment is recommended for all PSAs, irrespective of size and location. Endovascular management with transarterial embolisation of the PSA and the PSA-bearing arterial segment is most commonly performed. Here, the authors present a case of a 54-year-old patient with chronic pancreatitis, who presented with haemodynamic instability and was diagnosed by Computed Tomography (CT) scan as having a giant pseudoaneurysm of the splenic artery with rupture leading to haemoperitoneum. The patient underwent emergency endovascular intervention via right femoral arterial access, with diagnosis confirmed by selective angiograms of the coeliac and splenic arteries. Subsequently, superselective embolisation of the pseudoaneurysm and the lower pole branch of the splenic artery was performed using N-Butyl Cyanoacrylate (NBCA) glue mixed with lipiodol. Postembolisation angiograms confirmed the complete exclusion of the PSA and the lower pole branch, indicating technical success. Postoperatively, there was an improvement in the patient's haemodynamic status and he was eventually discharged with normal vital signs and stable haemoglobin counts. This case demonstrates the rare diagnosis of a giant splenic PSA, managed by an endovascular approach using NBCA glue mixed with lipiodol for embolisation.

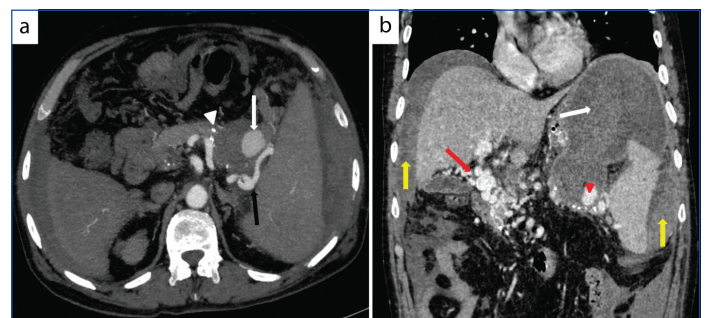
Keywords: Digital subtraction angiography, Glue embolisation, Haemoperitoneum, Pancreatitis, Portal cavernoma

CASE REPORT

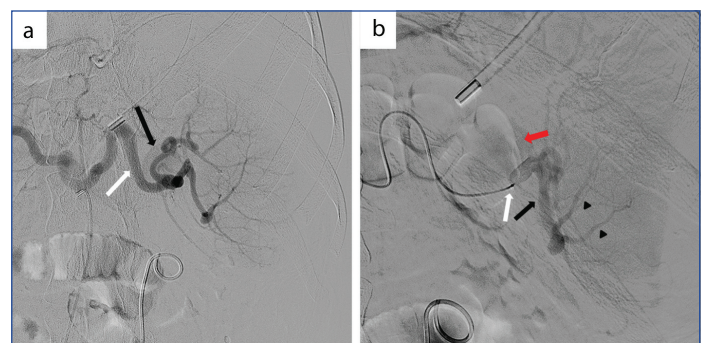
A 54-year-old male patient presented to the emergency department with abdominal pain and giddiness for one day. He had a long-standing history of recurrent abdominal pain for two years, which had been diagnosed as chronic pancreatitis. After further questioning, the patient disclosed a history of melaena one month prior. At an outside hospital, the patient had a haemoglobin level of 4 gm/dL, for which he received blood transfusions and was subsequently referred to the present institute for further management. On evaluation, his haemoglobin was 7.1 gm/dL, and he had tachycardia (heart rate: 120 beats/min) and hypotension (BP: 90/60 mmHg).

CT abdominal angiography was performed, showing a large, well-defined, predominantly hyperdense lesion measuring 18×12×11 cm in the left upper quadrant in the perisplenic region. On arterial phase imaging, a smaller area within the lesion, closely abutting the distal splenic artery, showed arterial filling [Table/Fig-1a,b]. Additionally, mild hyperdense free fluid was noted in the abdominal and pelvic cavities. A diagnosis of a partially thrombosed giant pseudoaneurysm of the splenic artery with rupture leading to haemoperitoneum was made. Other findings on the CT scan included changes of chronic calcific pancreatitis, portal cavernoma and multiple omental and mesenteric collaterals.

In view of the patient's haemodynamic instability, he was taken for an emergency embolisation procedure. Standard percutaneous 5F right femoral arterial access was obtained, and a selective celiac angiogram was performed, which confirmed the presence of a pseudoaneurysm arising from the lower pole branch of the splenic artery [Table/Fig-2a,b]. Superselective cannulation of the lower pole branch of the splenic artery was performed using a Direxion (Boston) microcatheter to avoid non target embolisation of pancreatic branches. Subsequently, embolisation of the pseudoaneurysm and the lower pole branch of the splenic artery was performed using 2 cc of 25% N-Butyl Cyanoacrylate (NBCA) glue mixed with lipiodol. Postprocedure celiac and superior mesenteric artery angiograms showed complete exclusion of the splenic artery pseudoaneurysm from circulation and preservation of the upper pole branch of the splenic artery [Table/Fig-3a,b].

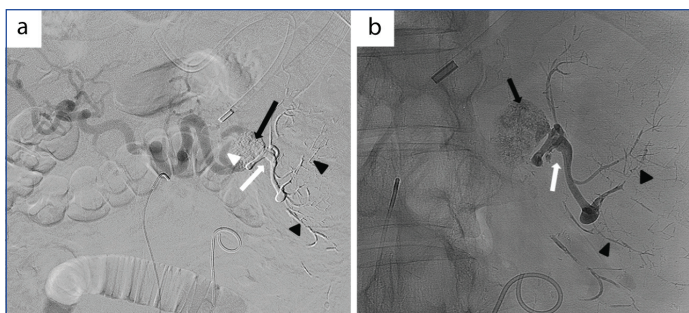


[Table/Fig-1]: (a) Axial arterial phase CECT scan image showing the pseudoaneurysm (white arrow) in the region of pancreatic tail arising from distal splenic artery (black arrow). Note the pancreatic calcification (white arrowhead) consistent with chronic calcific pancreatitis; (b) Portal phase coronal reformatted image showing small enhancing portion (red arrowhead) with larger, partially thrombosed giant pseudoaneurysm sac (white arrow). Note perisplenic and perihepatic haematomas (yellow arrows) and portal cavernoma (red arrow).



[Table/Fig-2]: (a) Frontal projection celiac angiogram showing tortuous course of splenic artery (white arrow) with a pseudoaneurysm (black arrow) at the distal splenic artery in the region of splenic hilum; (b) Frontal projection angiogram demonstrating superselective cannulation of lower pole branch of splenic artery with microcatheter tip (white arrow), lower pole branch (black arrow) and intraparenchymal branches (black arrowhead). Notice opacification of pseudoaneurysm sac (red arrow).

In the postoperative period, the patient was monitored for a further drop in haemoglobin, hypotension, or fever. He complained of mild left hypochondriac pain, which was managed with analgesics. There was a gradual improvement in the haemodynamic status of



[Table/Fig-3]: a) Frontal projection celiac angiogram post embolisation showing complete exclusion of pseudoaneurysm sac (black arrow), lower pole branch of splenic artery (white arrow) and intraparenchymal branches at the lower pole (black arrowheads). Note persistent filling of the upper pole splenic artery branch (white arrowhead); (b) Glue cast pseudoaneurysm sac (black arrow), lower pole branch of splenic artery (white arrow) and intraparenchymal branches at the lower pole (black arrowheads).

the patient. A bedside ultrasound of the abdomen did not reveal any further increase in perisplenic haematoma or haemoperitoneum. He was discharged three days later when his vital signs had returned to normal and his haemoglobin level was stable at 8.3 gm/dL. At one month of clinical follow-up, his abdominal pain and distention had significantly reduced.

DISCUSSION

Pseudoaneurysms are abnormal outpouchings from the arterial wall, which are covered only by the adventitia, whereas true aneurysms contain all three layers of the arterial wall [1]. Splenic artery pseudoaneurysms in cases of pancreatitis are thought to result from damage to the arterial wall by proteolytic pancreatic enzymes; other causes of splenic PSAs include iatrogenic and traumatic injuries, tumours, and peptic ulcer disease [2]. Most splenic artery pseudoaneurysms are small in size, with an average diameter of 2.1 cm at diagnosis; giant pseudoaneurysms measuring more than 5 cm are rare [3,4]. Current guidelines recommend that visceral artery PSAs of any size should be treated due to the lack of normal vascular wall structure, gradually increasing size, and the risk of sudden, massive bleeding [5,6].

Treatment options for splenic artery PSAs include transarterial embolisation techniques, ligation of the splenic artery and resection of the PSA or splenectomy, with or without distal pancreatectomy along with resection of the PSA [7]. The selection of treatment options is made taking into consideration the age of the patient, location and dimensions of the PSA, complications, adequacy of collateral flow to the liver and clinical severity [8]. With the improvement of Digital Subtraction Angiography (DSA) technology and equipment, endovascular treatment is now considered the first choice in most cases, as it has a high success rate, low morbidity, a shorter hospital stay with rapid recovery and can be performed under local anaesthesia [8-10]. While open surgical options have a higher risk of perioperative complications and mortality, they can be considered in haemodynamically unstable patients [8] or in cases where endovascular embolisation is not feasible.

The most common endovascular technique is the sandwich technique, which involves selective catheterisation of the aneurysm-bearing arterial segment and embolisation with coils just distal and proximal to the lesion to avoid reformation via collateral circulation [11,12]. Alternatively, the sac-packing technique with coils, glue embolisation of the parent artery, vascular plug, or stent-graft placement may also be employed [12]. The sac-packing technique of coil embolisation has the advantage of preserving the splenic artery distal to a pseudoaneurysm. However, it should be performed with great caution due to the high risk of rupture and aneurysm recanalisation. Vascular plugs can be used as an alternative to coils; however, as plugs are bulky and stiff, their use in distal splenic artery aneurysms is limited. Splenic aneurysms and pseudoaneurysms can be treated effectively using stent grafts, which exclude the

pseudoaneurysm sac while maintaining vascular patency [13,14]. However, like vascular plugs, stent grafts are bulky, which limits their use in tortuous splenic artery anatomy and distal splenic artery aneurysms. NBCA glue undergoes rapid polymerisation on contact with ionic solutions such as blood and saline, which can result in non target proximal or distal embolisation or adhesion of the microcatheter to the glue cast. The rate of polymerisation can be adjusted by changing the ratio of glue to lipiodol, thus requiring the operator to have specialised training and experience [15].

Borzelli A et al., reported a case of recurrent chronic pancreatitis with pseudocysts and a giant pseudoaneurysm of the splenic artery, associated with spleen invasion and concomitant Arteriovenous Fistula (AVF), which was treated successfully by transcatheter embolisation with multiple microcoils [16]. Meyer NS and Ullrich L, described a case of an 80-year-old male with a grade IV splenic laceration, giant splenic artery PSA, and an AVF component, managed with open splenectomy due to haemodynamic instability, high risk of decompensation, and lack of definitive endovascular treatment due to the large size of the pseudoaneurysm and AVF component [17].

In the present case, the patient presented with active bleeding and haemodynamic instability; hence, the endovascular embolisation procedure was preferred. Glue was chosen as the embolising agent in this case due to the distal location of the aneurysm and the tortuous anatomy of the proximal splenic artery. Additionally, initial CT revealed portal cavernoma formation and multiple omental and mesenteric collaterals favouring portal hypertension. By using glue, authors were able to embolise the splenic artery branches to the lower pole of the spleen, which is expected to have a beneficial effect on portal hypertension.

CONCLUSION(S)

Giant splenic artery pseudoaneurysms are rare abnormal outpouchings from the arterial wall, which are covered only by the adventitia and measure more than 5 cm in diameter. The clinical case described here is notable because of the significant size of the distal splenic artery pseudoaneurysm, which had ruptured, leading to haemoperitoneum and the planning of endovascular NBCA glue embolisation, considering the associated issues of chronic pancreatitis and portal hypertension. With the ongoing improvement in microcatheters, coils and more trackable stent grafts, endovascular embolisation can be considered as the first-line treatment for splenic artery pseudoaneurysms, offering both constructive (preserving the vessel) and destructive (occluding the vessel) treatment options.

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PLAGIARISM CHECKING METHODS: [\[Jain H et al.\]](#)

- Plagiarism X-checker: Feb 12, 2025
- Manual Googling: Mar 27, 2025
- iThenticate Software: Mar 29, 2025 (11%)

ETYMOLOGY: Author Origin
EMENDATIONS: 6

AUTHOR DECLARATION:

- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. Yes

Date of Submission: **Feb 08, 2025**
Date of Peer Review: **Mar 06, 2025**
Date of Acceptance: **Mar 31, 2025**
Date of Publishing: **May 01, 2025**