
CASE REPORT

Superior Mesenteric Arteriovenous Fistula: an Unusual Cause of Ascites

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ABSTRACT

Superior mesenteric arteriovenous fistulae are rare. They are usually associated with previous penetrating abdominal trauma or surgery. Patients may present with ascites or gastrointestinal bleeding. This report is of a patient with postoperative refractory ascites. Contrast-enhanced computed tomography scan and digital subtraction angiogram showed a communication between the superior mesenteric artery and the dilated superior mesenteric vein. A superior mesenteric arteriovenous fistula can be treated with either surgical ligation or endovascular embolisation, with the latter successfully performed for this patient.

Key Words: Arteriovenous fistula; Embolization, therapeutic; Hypertension, portal; Mesenteric artery, superior

INTRODUCTION

Superior mesenteric arteriovenous fistulae (SMAVF) are rare. They are usually associated with previous penetrating abdominal trauma or abdominal surgery¹ or, more rarely, related to superior mesenteric artery (SMA) aneurysm secondary to connective tissue disease.^{2,3} Patients may present with ascites due to portal hypertension or gastrointestinal haemorrhage secondary to bleeding varices.^{1,3,4} This report is of a patient who presented with refractory ascites 2 months after an abdominal operation.

CASE REPORT

A 67-year-old woman presented with a 4-cm left ovarian mass, identified during a routine medical check in September 2006. Subsequent laparotomy with hysterectomy and bilateral oophorectomy, performed in October 2006, showed this to be a benign ovarian fibroma. Her medical history revealed a previous appendicectomy when she was aged 11 years and caesarean sections when she was aged 22 years and 27 years. She drank alcohol on social occasions.

Soon after the laparotomy, she developed abdominal distension. Clinical examination demonstrated ascites,

which was refractory to repeated paracentesis. Apart from mildly deranged liver function with raised alanine aminotransferase 42 U/L (normal range, 10-40 U/L), aspartate aminotransferase 39 U/L (normal range, 20-48 U/L), alkaline phosphatase 458 U/L (normal range, 50-120 U/L), and γ -glutamyltransferase 183 U/L (normal range, 0-30 U/L), her blood results were normal. Diagnostic paracentesis confirmed the ascites to be a transudate with no evidence of infection or malignancy.

After excluding possible medical causes, the ascites was treated with repeated paracentesis. A contrast-enhanced computed tomography (CT) scan of the abdomen and pelvis, performed in December 2006, demonstrated portal hypertension with gross ascites. There was a fistula between a jejunal branch of the SMA and a dilated superior mesenteric vein (SMV) [Figure 1]. There was also early opacification of the SMV and portal vein with poor and delayed enhancement of the attenuated distal SMA branches. The walls of the small and large bowels, down to the transverse colon watershed, were oedematous and underperfused suggesting mesenteric ischaemia in the SMA territory, while the colon distal to the splenic flexure corresponding to the inferior mesenteric artery (IMA) territory was less oedematous.

Digital subtraction angiograms of the SMA and IMA were performed. SMA angiogram revealed an arteriovenous fistula between the mid-portion of the SMA and SMV, with the presence of venous varices (Figure 2).

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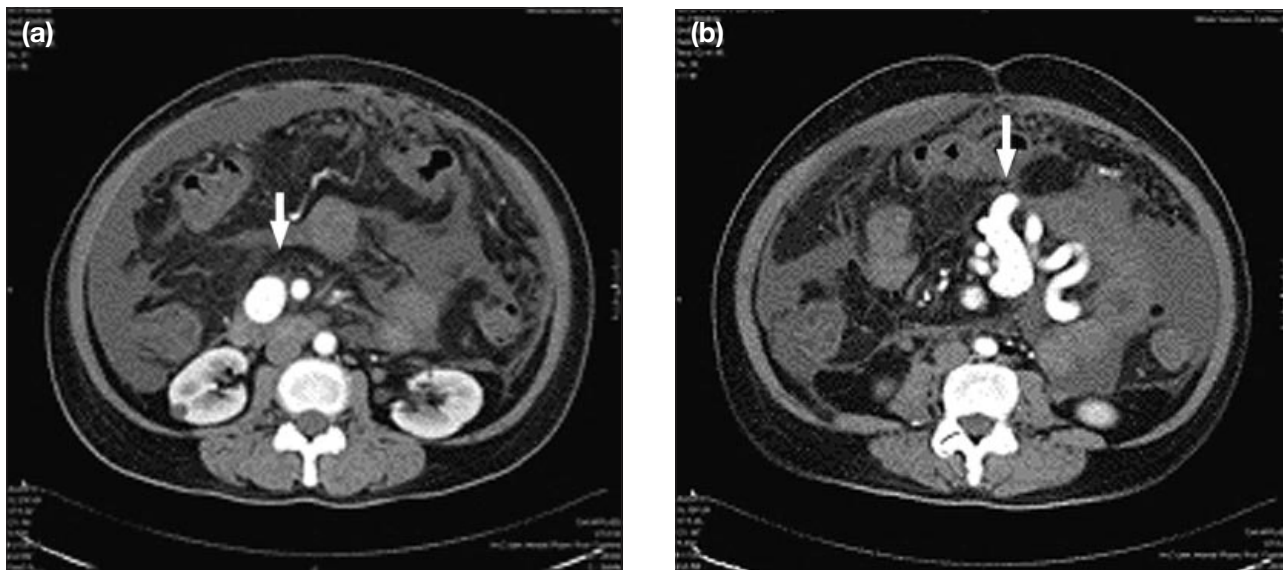


Figure 1. Contrast-enhanced computed tomography scan showing (a) ascites and early opacification of a dilated superior mesenteric vein (arrow); and (b) arteriovenous fistula or varices (arrow).

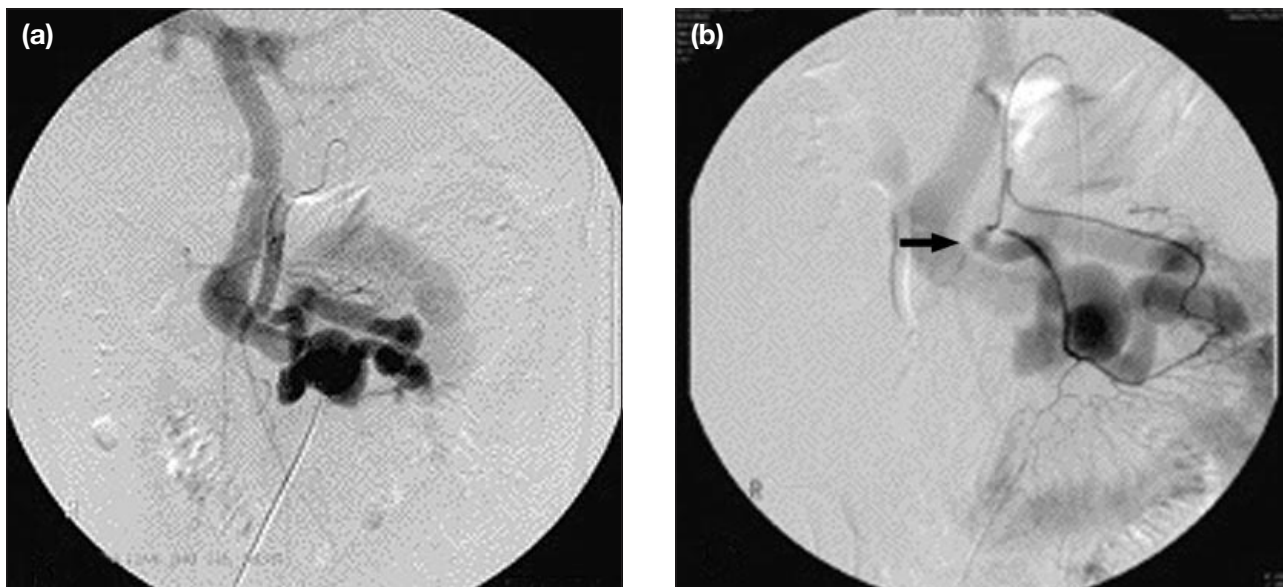


Figure 2. (a) Angiogram of the superior mesenteric artery showing an arteriovenous fistula between the mid-portion of the superior mesenteric artery and the superior mesenteric vein, presence of venous varices, and rapid filling of the superior mesenteric vein and the portal vein; and (b) selective angiogram of the superior mesenteric artery showing one of the superior mesenteric artery branches (arrow) close to the feeding artery of the fistula.

There was rapid filling of the SMV and portal vein via the venous side of the fistula. IMA angiogram was normal.

Embolisation of the SMAVF was performed. Initially, the fistula was selectively cannulated from the SMA using a 5-F Yashiro catheter (Terumo, Tokyo, Japan) [Figure 3a]. A microcatheter was not necessary. Successful embolisation of the SMAVF using fibre coils was subsequently performed to the supplying artery, which was finally occluded (Figure 3b). Fourteen fibre coils

were used (2 at 8 mm x 5 cm; 2 at 10 mm x 5 cm; 6 at 10 mm x 6 cm; 4 at 12 mm x 8 cm). The healthy SMA branches were not sacrificed. A stent was not used for this patient, as one of the SMA branches was found to be in close proximity to the feeding artery of the fistula (Figure 2), rendering stent deployment dangerous.

The patient recovered well and her liver function was normalised. Ultrasound 1 month after the embolisation showed resolution of the ascites. The spectral patterns of the proximal SMA, SMV, and portal vein were

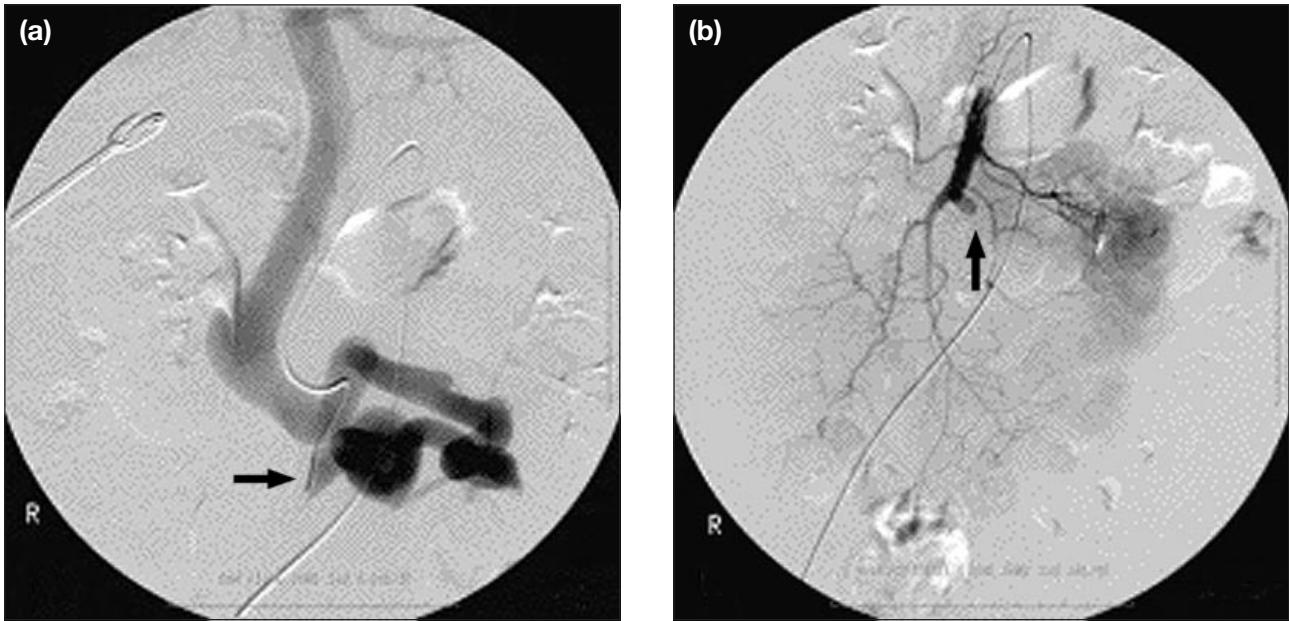


Figure 3. (a) Selective cannulation of the arteriovenous fistula using a 5-F Yashiro catheter (arrow); and (b) successful embolisation of the fistula using fibre coils to the supplying artery, which was occluded (arrow).

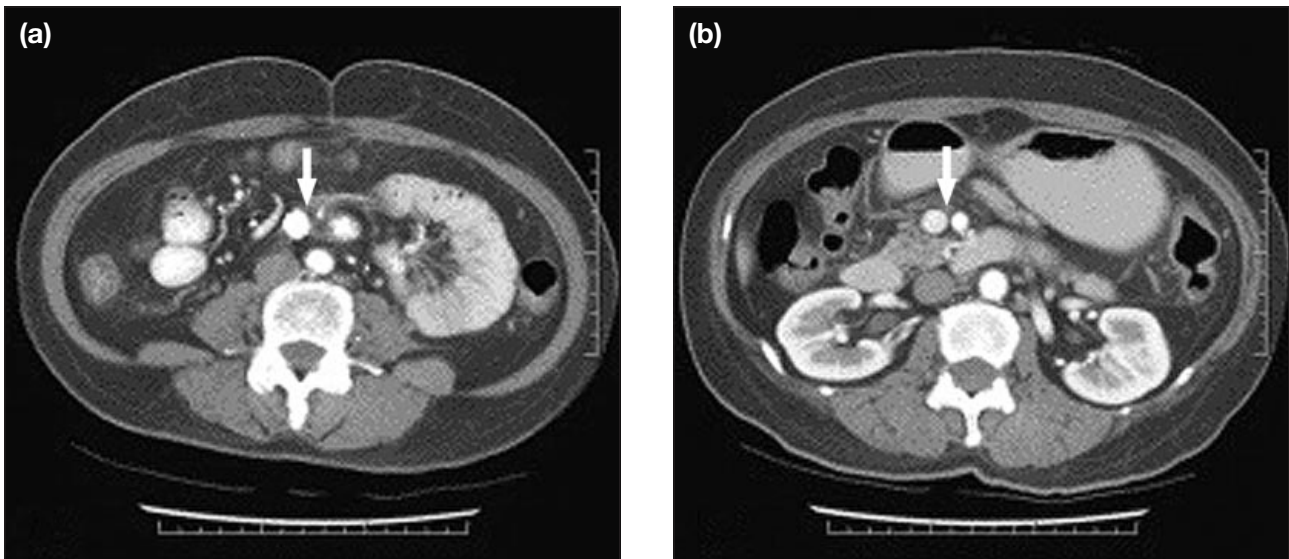


Figure 4. Postembolisation contrast-enhanced computed tomography scan showing (a) no reaccumulation of ascites and embolisation coils in situ (arrow); and (b) patent superior mesenteric artery and superior mesenteric vein (arrow).

normal. Contrast-enhanced CT scan 3 months after the procedure showed a patent SMA and normally opacified SMV and portal vein, with no reaccumulation of ascites (Figure 4). There was no evidence of mesenteric ischaemia.

DISCUSSION

SMAVF are uncommon and are only occasionally reported in the literature. Most SMAVF are acquired, and are believed to be associated with previous penetrating abdominal trauma or abdominal surgery, possibly related to a transfixion suture.⁴ Other SMAVF are congenital,

secondary to arteriovenous malformation, aneurysm, or connective tissue disease.³ Patients may present with abdominal pain, nausea, vomiting, and abdominal distension due to ascites, as well as recurrent gastrointestinal bleeding from varices. Clinical examination shows an abdominal scar due to a previous operation, bruit at the epigastrium, and signs of congestive heart failure.⁴ Gastroscopy can show oesophageal varices secondary to portal hypertension, and colonoscopy may demonstrate oedematous mucosa and punctate areas of erythema, with biopsy showing normal cellular architecture and reactive mesothelial cells.^{1,4}

CT scan is useful to make the diagnosis.^{1,4} Portal hypertension with ascites, SMA-SMV shunting or fistula, poor and delayed enhancement of distal SMA branches, aneurysmal dilatation, and early opacification of the SMV and portal vein, as well as oedematous and hypo-perfused small and large bowel walls are the usual findings.

Digital subtraction angiogram is the gold standard to confirm the diagnosis.^{1,4} This procedure directly shows the arteriovenous fistula with dilated and variceal jejunal or ileal veins, rapid filling of a dilated SMV and portal vein, and retrograde perfusion of the splenic vein, while the SMA branches distal to the arteriovenous fistula are poorly perfused.

Treatment for SMAVF includes endovascular embolisation and surgical ligation.³⁻⁹ The former can be achieved by embolising the feeding fistula with coils or covering the feeding vessel with a stent graft. The main aim of embolisation is to occlude the fistula while preserving the other SMA branches so as not to cause any mesenteric infarct. SMV or portal vein thrombosis is an important but rare complication, for which antithrombotic agents may be necessary.⁴ This complication was not encountered in this patient. The arteriovenous fistula can also be ligated at laparotomy, and small bowel resection may be necessary in the presence of mesenteric ischaemia or infarct. The main aim of both methods is to alleviate the portal hypertension to preserve liver function and to prevent variceal bleeding.

In conclusion, SMAVF is an uncommon cause of ascites, usually secondary to previous abdominal surgery or

penetrating abdominal trauma. Contrast-enhanced CT and angiogram are the imaging modalities of choice. Endovascular embolisation, which was successfully performed for this patient, is an effective treatment.

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