

Bridging stent repair of type III endoleak causing aortocaval fistula after branched aortic endovascular repair

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A 62-year-old man presented to our department with abdominal pain and diarrhea for 3 weeks on a background of previous branched endovascular repair for a thoracoabdominal aneurysm. A triple-phase computed tomography scan of his abdomen and pelvis showed a large aortocaval fistula caused by a type III endoleak from a dislodged superior mesenteric artery stent. He was successfully treated with a BeGraft (Bentley Innomed, Hechingen, Germany) by using an endovascular technique. (*J Vasc Surg Cases and Innovative Techniques* 2017;3:4-6.)

Very few cases of aortocaval fistulas (ACFs) after endovascular aneurysm repair (EVAR) have been reported in the literature. Most case reports have described an ACF in the context of a ruptured abdominal aortic aneurysm (AAA) or an iatrogenic injury to the inferior vena cava during laparoscopic repair of an AAA. Only four reports of an ACF caused by a persistent type II endoleak have been published. We describe the case of 62-year-old man who presented with a symptomatic ACF caused by a type III endoleak that was successfully managed using an endovascular technique. Consent was obtained from the patient to publish this case report along with the images.

CASE REPORT

A 62-year-old man was referred to our unit with a 2-week history of crampy abdominal pain and diarrhea. His medical history was significant for controlled hypertension and former smoking. His surgical history was consistent with a previous endoluminal repair of a large thoracoabdominal aneurysm using a standard branched Cook endograft (Cook Medical, Bloomington, Ind) with four visceral branches for the celiac, the superior mesenteric artery (SMA), and the right and left renal arteries. He underwent this procedure 4 years before this presentation.

The patient was afebrile and hemodynamically stable. He had been passing four to five watery stools daily for the past 2 weeks. The initial laboratory investigations, namely a full blood count, inflammatory markers, and stool culture, were nondiagnostic for an acute infective process. The patient denied a history of recent travel or previous viral illness.

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A triple-phase computed tomography scan of the thorax, abdomen, and pelvis showed dilated bowel loops measuring up to 6 cm (maximum transverse diameter) in the SMA territory. A dislodged SMA stent was noted causing a large type III endoleak with decompression into the inferior vena cava via a large ACF. The endoleak was arising from the junction of the branch with the connecting stent (*Fig 1*).

There was a marginal expansion of the sac in the interim period from 11 to 11.4 cm. The fistulation meant that the sac size did not increase significantly.

After an informed consent was obtained, the patient was brought forward for an endoluminal repair of the dislodged SMA stent as a semi-elective case. Access was gained via a left axillary cutdown. Because of the tortuous nature of the branches, the SMA stent was initially cannulated. The significant gap from the end of the stent to the native SMA made it difficult for traditional catheters to gain access. A steerable catheter was successfully used to bridge the gap to the native SMA.

A 10-mm × 57-mm BeGraft (Bentley Innomed, Hechingen, Germany) was successfully deployed (*Fig 2*). Selective digital subtraction angiography runs of the SMA showed the type III endoleak was completely excluded. Delayed runs showed no filling of the ACF.

The patient made an uneventful recovery, with complete resolution of his symptoms, and was discharged home 2 days later. Repeat imaging showed sealing of the endoleak and no further fistula flow. The sac size had also stabilized at the 1-month follow-up computed tomography scan (*Fig 3*). At the 6-month follow-up, he was symptom free.

DISCUSSION

An ACF is a rare condition that was first described by James Syme in 1831.¹ In a study reported by Akwei et al, ACF constitutes <6% of all arteriovenous fistulas.² An erosion or spontaneous rupture of the AAA into the vena cava is the most common cause of an ACF.^{3,4} The incidence of an ACF in patients treated with an endoprosthesis for an aortic aneurysm repair is low.

Cardiac preload and venous hypertension are understood to be the sequelae from shunting of blood from a high-pressure arterial system into a low-pressure venous system. This may clinically manifest in some

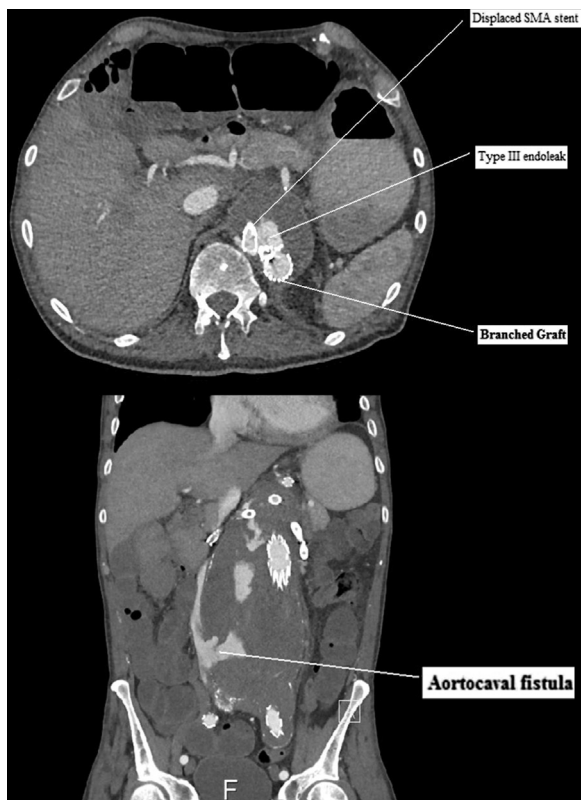


Fig 1. Axial and coronal views of type III endoleak and aortocaval fistula (ACF). SMA, Superior mesenteric artery.

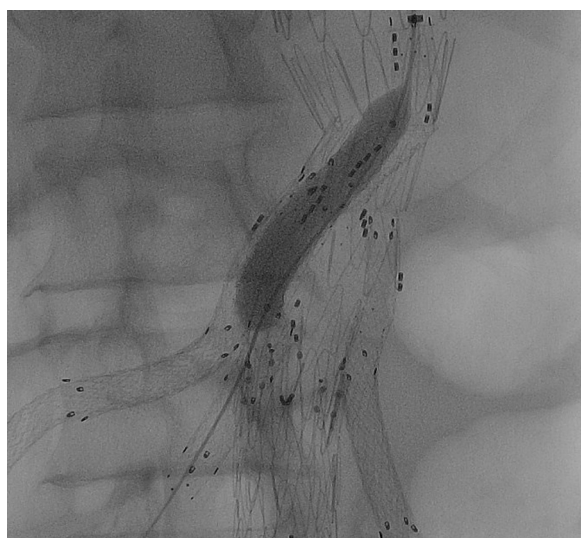


Fig 2. Deployment of BeGraft (Bentley Innomed, Hechingen, Germany) in the superior mesenteric artery (SMA).

patients as an acute lower limb edema or hematuria caused by impaired renal perfusion.^{5,6} Lau et al⁵ have described these symptoms to be prevalent in 50% to 80% patients with a diagnosis of an ACF.

Leon et al⁷ mentions that the symptoms related to an arteriovenous fistula vary according to the size of



Fig 3. Follow-up computed tomography scan of the aorta at 1 month shows resolution of the aortocaval fistula (ACF).

the fistula. Patients with a small fistula could be asymptomatic.

Endovascular and conservative treatment options have been described in the surgical literature for management of ACF. The traditional approach for treatment of an ACF was an open surgical repair, which had a mortality rate of 30%, particularly in patients with cardiac decompression.⁵ The first endovascular repair was performed by Beveridge et al⁸ in 1998, after which other authors have described successful treatment using endovascular techniques.

Van de Luijtgaarden et al⁹ say that in the absence of systemic repercussions, persistent ACFs caused by type II endoleak after EVAR may be managed conservatively and that favorable remodelling of the aneurysm sac might be possible.

In our case, the patient was symptomatic from the type III endoleak due to the dislodged SMA stent. This could have occurred as a result of remodelling of the aorta after aneurysm repair. The bowel symptoms were related to ischemia caused by alteration of arterial supply due to the stent dislodgment and may be related to the venous hypertension. Because the patient's symptoms resolved immediately after the endovascular treatment, this was thought to be most likely cause.

The Bentley Innomed stent is an approved graft in Australia and New Zealand. These grafts are extremely versatile and became popular with the inability to access Atrium V12 stent grafts (Atrium Medical Corp, Hudson, NH) in New Zealand.

In our case, an indirect approach was used to treat the ACF without using an endovenous stent. However a

covered endovenous stent or a direct embolization of the aortic sac by injecting Onyx glue (ev3 Endovascular, Inc, Plymouth, Minn) could be considered if there were an evidence of aortic sac expansion in the future.

CONCLUSIONS

A high degree of vigilance is required in patients presenting with atypical symptoms of abdominal pain. Timely diagnosis and management is important for treatment of an ACF because this has shown to improve patient outcomes.¹⁰ Endovascular treatment is possible for treatment of ACFs even after remodelling of the stent grafts after a previous EVAR.

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