Conservative management of persistent aortocaval fistula after endovascular aortic repair

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Endovascular repair is a valid alternative for patients with abdominal aortic aneurysms. However, in patients with concomitant aortocaval fistulas, type II endoleaks may result in a persistent communication between the aneurysm sac and the inferior vena cava. In these patients, prompt closure of the persistent fistula has been advocated. We present a patient with an abdominal aortic aneurysm, with aortocaval fistula, who was managed endovascularly. Aneurysm sac shrinkage was observed despite persistent aortocaval communication due to type II endoleak. This case demonstrates that conservative management of type II endoleaks associated with persistent aortocaval fistulas is possible and may result in favorable aneurysm sac remodelling. (J Vasc Surg 2013;58:1080-3.)

Endovascular aneurysm repair (EVAR) is a valid alternative over open reconstruction in patients with (ruptured) abdominal aortic aneurysms (AAAs).[1] However, in patients with concomitant arteriovenous (AV) fistulas, type II endoleaks may result in a persistent communication between the aneurysm sac and the venous system. In the current literature, early closure of the fistula has been advocated due to concerns about future aneurysm-related complications.[2,3] We present an AAA patient with an aortocaval fistula who was managed conservatively and in whom favorable aneurysm sac remodelling was observed, despite a persistent aortocaval communication due to a large type II endoleak.

CASE REPORT

A 61-year-old man presented at the emergency department with acute low back and abdominal pain. The patient had a history of hypertension and chronic obstructive pulmonary disease. Prescription medications included a β-blocker, an angiotensin II inhibitor, and a short-acting β_2-adrenergic receptor agonist. On admission, his heart rate and blood pressure were within normal reference ranges (72 beats/min and 123/87 mm Hg, respectively). Physical examination revealed a tender, pulsatile abdominal mass, without abdominal bruit or thrill. No clinical signs of venous hypertension or heart failure were observed. Laboratory results showed no hepatic or kidney dysfunction. A computed tomography angiography (CTA) demonstrated a large AAA (10 cm in transverse diameter) with an associated aortocaval fistula (Fig 1).

The patient's anatomy was suitable for EVAR, and a bifurcated Endurant endovascular device (Medtronic Inc, Minneapolis, Minn) was used for exclusion. The perioperative final angiography showed an evident type II endoleak from the inferior mesenteric artery, but no further action was taken.

The patient had an uneventful postoperative recovery and was discharged after 5 days. In addition, postoperative renal and hepatic function remained normal.

A postoperative CTA showed persistent communication with the inferior vena cava (IVC) as a result of the type II endoleak (Fig 1). To evaluate the systemic repercussion of the persistent aortocaval fistula, the patient was referred for a formal cardiac evaluation after 3 months. He was asymptomatic, and results of electrocardiography were normal. Echocardiography showed a normal systolic left ventricular ejection fraction, with no signs of increased right atrial filling pressure and a physiologic collapse of the IVC. Consequently, no further action was taken. After 1 year of follow-up, CTA showed shrinkage of the aneurysm sac diameter by 10 mm and an 8% reduction in volume compared with the postoperative CTA,[4] despite the persistent type II endoleak (Fig 2).

DISCUSSION

Persistent AV fistulas after EVAR have been reported, but the natural history of this complication remains unknown; moreover, there is no clear evidence in the current literature supporting treatment over observation. Our case demonstrates that persistent aortocaval communication due to type II endoleak is possible and that a conservative approach may be preferable in the absence of systemic manifestations and in the face of favorable aneurysm sac remodelling.

Aortocaval fistulas are, by definition, high-flow fistulas that may result in complications due to increased cardiac

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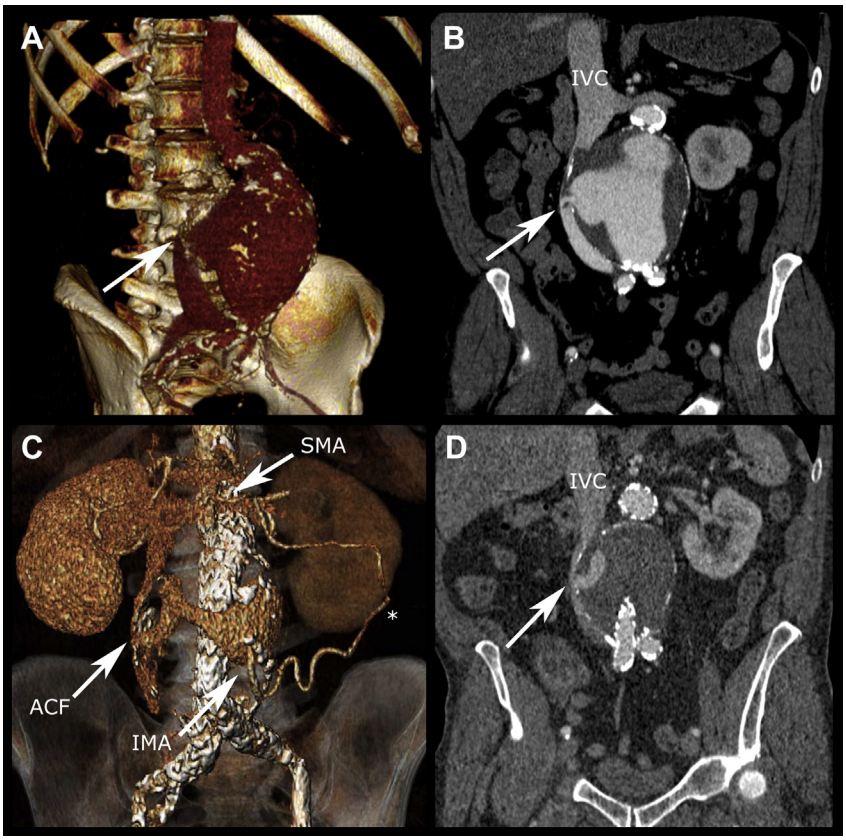

Fig 1. A preoperative **(A)** luminal three-dimensional reconstruction and a **(B)** coronal view of the computed tomography angiography (CTA) show a 10-cm large abdominal aortic aneurysm (AAA) with associated aortocaval fistula (*ACF*, *white arrowheads*). A postoperative **(C)** reconstruction and a **(D)** coronal view show the persistent aortocaval fistula at the 1-year follow-up. *Artery of Drummond; *IMA*, inferior mesenteric artery; *IVC*, inferior vena cava; *SMA*, superior mesenteric artery.

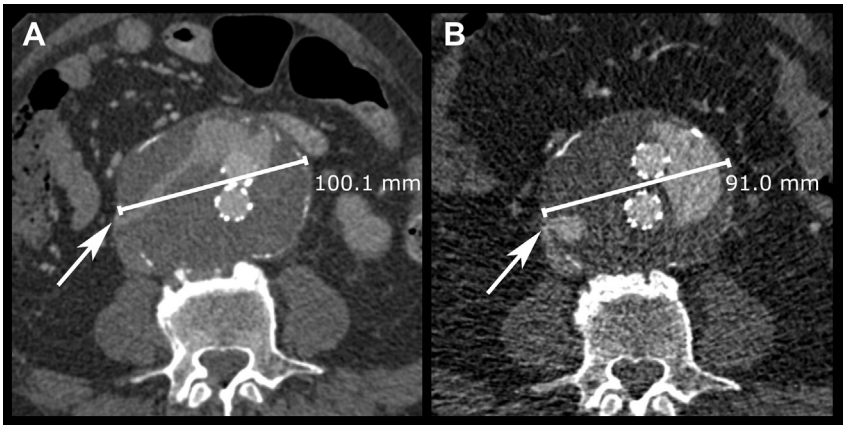

Fig 2. Postoperative computed tomography angiography (CTA) at **(A)** 6 weeks and at the **(B)** 1-year follow-up shows a 10-mm decrease in diameter. Notice the large type II endoleak associated with a persistent aortocaval fistula (*white arrowheads*).

output and venous hypertension.[5-7] Treatment is therefore advised. Patients with aortocaval fistulas have traditionally been treated with open repair, with mortality rates ranging from 16% to 66%.[5] Because EVAR has been adopted as valid treatment for (ruptured) AAAs,[1] also in the presence of aortocaval fistulas, persistent communication between

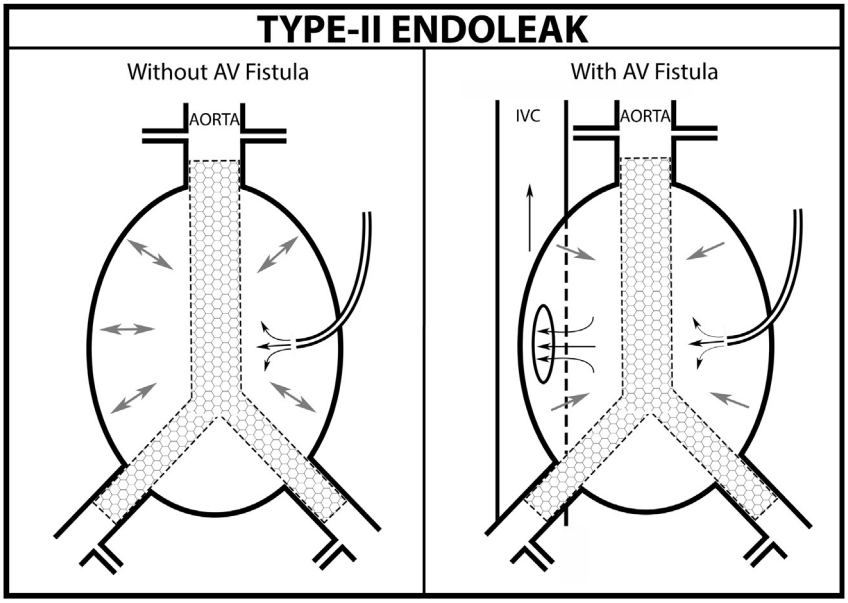

Fig 3. Different dynamics are shown in the aneurysm sac between a type II endoleak **(left)** without and **(right)** with an arteriovenous (*AV*) fistula. *IVC,* Inferior vena cava.

the aneurysm sac and the IVC may occur due to a type II endoleak. The question remains if—and how—we should manage these persistent fistulas.

Type II endoleak is defined as persistent retrograde flow into the aneurysm sac from the inferior mesenteric, lumbar, or other arteries, without attachment-site leak.[8] They are classified as (a) simple, with one patent branch or (b) complex, with two or more patent branches.[9] These endoleaks are considered important because they may result in repressurization of the excluded aneurysm sac, with consequent growth and, ultimately, rupture. However, in the presence of persistent caval communication, type II endoleaks are subject to different flow and pressure dynamics caused by the connection with the low-pressure venous system (2-8 mm Hg).[10] The connection causes a pressure gradient, resulting in a decrease of in-sac pressure (Fig 3), mimicking the concept of aneurysm sac fenestration.[11] Owing to the low-pressure nature, this special type of type II endoleak has a very low probability of aneurysm sac growth. This was confirmed in our patient, in whom we observed significant aneurysm sac shrinkage at 1 year (Fig 2).

Several cases of endovascular treatment for AAA patients with aortocaval fistulas have been described.[12-15] However, only four case reports of persistent aortocaval fistulas have been published to date. Burke et al[2] reported a patient with a type II endoleak 4 days postoperatively, which was immediately treated with a cuff in the IVC and glue in the aneurysm sac. Kopp et al[3] reported a similar patient who underwent secondary coiling of a persistent type II endoleak. Lastly, Vetrhus et al[15] suggested that persistent fistulas in this context tend to resolve spontaneously, which did not occur in our patient. The fistulas in their two patients resolved on-table in one patient and after 4 weeks in the other, without further intervention.[15] Taken together, the chance of a persistent aortocaval fistula after EVAR is rather small but is considerable due to the high frequency of type II endoleaks after EVAR.

We treated our patient conservatively because no systemic repercussions of the AV fistula were present and secondary interventions for type II endoleaks are generally reserved for persistent endoleaks (≥6 months) associated with aneurysm sac enlargement.[16] However, we acknowledge that conservative management in our patient might result in complications. Increased cardiac output and, ultimately, heart failure may develop from AV fistulas. Nevertheless, we considered this risk was relatively small because the arterial component of the fistula in our patient resulted from retrograde flow from the inferior mesenteric artery. Moreover, a physiologic collapse of the IVC was observed, emphasizing the low-pressure nature of the fistula. Nonetheless, careful and continued cardiac evaluation is required to ensure early identification of this possible complication.

In addition, embolization may occur as a result of dislodgment of aneurysm sac debris; however, we believe that the risk for clinically significant embolization is minute. First, the chance of embolization from untreated AAAs is very small.[17] Second, closing an AV fistula with an implant may also induce thromboembolism. Lastly, the chance of embolization due to manipulation during secondary intervention to close the AV fistula is probably higher than the embolization risk itself.

Because compression of the IVC was observed, thrombosis could occur. Therefore, implantation of an endograft in the IVC could be considered to exclude the AV fistula and treat IVC compression. However, this compression is

frequently observed in EVAR patients, and caval thrombosis is anecdotic. Also, stent graft placement in a significantly compressed IVC may yield a suboptimal result, with a risk of endograft collapse or acute stent thrombosis, or both.

CONCLUSIONS

Our case shows that in the absence of systemic repercussions, persistent aortocaval fistulas due to type II endoleak after EVAR may be managed conservatively and that favorable remodeling of the aneurysm sac is possible. Nevertheless, close observation, including periodic cardiac evaluation, is mandatory. Secondary intervention may be reserved for patients with persistent aortocaval fistulas combined with aneurysm sac enlargement or systemic manifestations.

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