Endovascular repair of delayed traumatic aortocaval fistula

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ABSTRACT

Aortocaval fistula (ACF) is an uncommon condition that can result in a number of adverse clinical sequelae. We describe a case of an ACF that occurred several years after open repair of a penetrating injury of the abdominal aorta and inferior vena cava. Whereas ACF can have sudden and catastrophic presentations, our patient had a subacute presentation of high-output heart failure. We were able to fully correct the vascular injury and heart failure physiology and symptoms with endovascular therapy. (J Vasc Surg Cases and Innovative Techniques 2019;5:467-71.)

Keywords: Aortocaval fistula; ACF; Gunshot wound; Endovascular repair; High-output heart failure

Aortocaval fistula (ACF) is an uncommon condition that can result in a number of adverse clinical sequelae. Common manifestations associated with ACF include back pain, abdominal bruit, congestive heart failure or lower extremity edema, hematuria, and liver and renal failure.[1-7] Less frequent complications include paradoxical pulmonary embolism.[8] The most commonly described cause of ACF is spontaneous rupture of an abdominal aortic aneurysm into the inferior vena cava (IVC), with a reported incidence of 3% to 4% of all ruptured aneurysms.[1] Less commonly, ACFs are caused by trauma or surgical injury or are the result of endovascular procedures. Rarely, ACF due to a congenital anomaly can be manifested in infancy.[9] Davis et al[10] reported an 11% incidence of iatrogenic ACF in a case series of 18 patients during 20 years. We describe a case of an ACF that occurred several years after open repair of a penetrating injury of the abdominal aorta and IVC. The patient is aware of our manuscript submission and has consented to publication of his case and images.

Whereas ACF can have sudden and catastrophic presentations, our patient had a subacute presentation of high-output heart failure. We were able to fully correct the vascular injury and heart failure physiology and symptoms with endovascular therapy.

CASE REPORT

A 41-year-old man with a history of alcohol abuse and abdominal gunshot wound (GSW) with resulting paraplegia of the right lower extremity presented to the hospital emergency department with a chief complaint of bilateral lower extremity pain and swelling. The original injury was incurred 4 years earlier and involved penetration by two bullets of the right flank. At laparotomy by the trauma service, two 1-cm defects in the aorta, one on the left lateral wall and one on the right lateral wall, just above the bifurcation, were initially repaired with polypropylene (Prolene). As this repair did not hold, the repair was redone with polydioxanone (PDS) suture. After the aortic repair, a 2-cm right lateral wall vena cava injury was found just above the iliac confluence and was likewise primarily repaired with absorbable suture. Postoperative recovery was uncomplicated, and follow-up included one office visit with the trauma service 1 month after discharge. He was then released from further follow-up.

The patient did well for approximately 3 years until he noted the gradual onset of bilateral lower extremity swelling and pain, causing him to present to our emergency department. Review of systems was positive for exertional dyspnea and mild orthopnea. On physical examination, he was hemodynamically normal with the exception of mild systemic hypertension. Cardiovascular examination was notable for jugular venous distention, normal S1 and loud S2, with a prominent P2 component, and a right-sided S3. A holosystolic murmur was best appreciated at the left lower sternal border and increased with inspiration. Abdominal examination was notable for a well-healed laparotomy scar, central pulsatile mass, and loud continuous midabdominal bruit. Peripheral pulse examination findings were normal. There were no additional bruits. Lower extremity examination was notable for pitting edema. Chest radiography demonstrated cardiomegaly and pulmonary vascular congestion. The patient was admitted to the cardiology service with the diagnosis of right-sided heart failure.

Transthoracic echocardiography revealed an enlarged right ventricle (RV) with systolic dysfunction as well as moderate to severe tricuspid regurgitation. He had normal left ventricular systolic function, although the ventricular septum was flattened, consistent with pressure and volume overload of the right side of the heart. Of note, there was evidence of high cardiac output on initial echocardiography, as evidenced by turbulent flow in the aortic arch (Fig 1) and a calculated cardiac output by Doppler interrogation of 10.2 L/min. No shunts were detected

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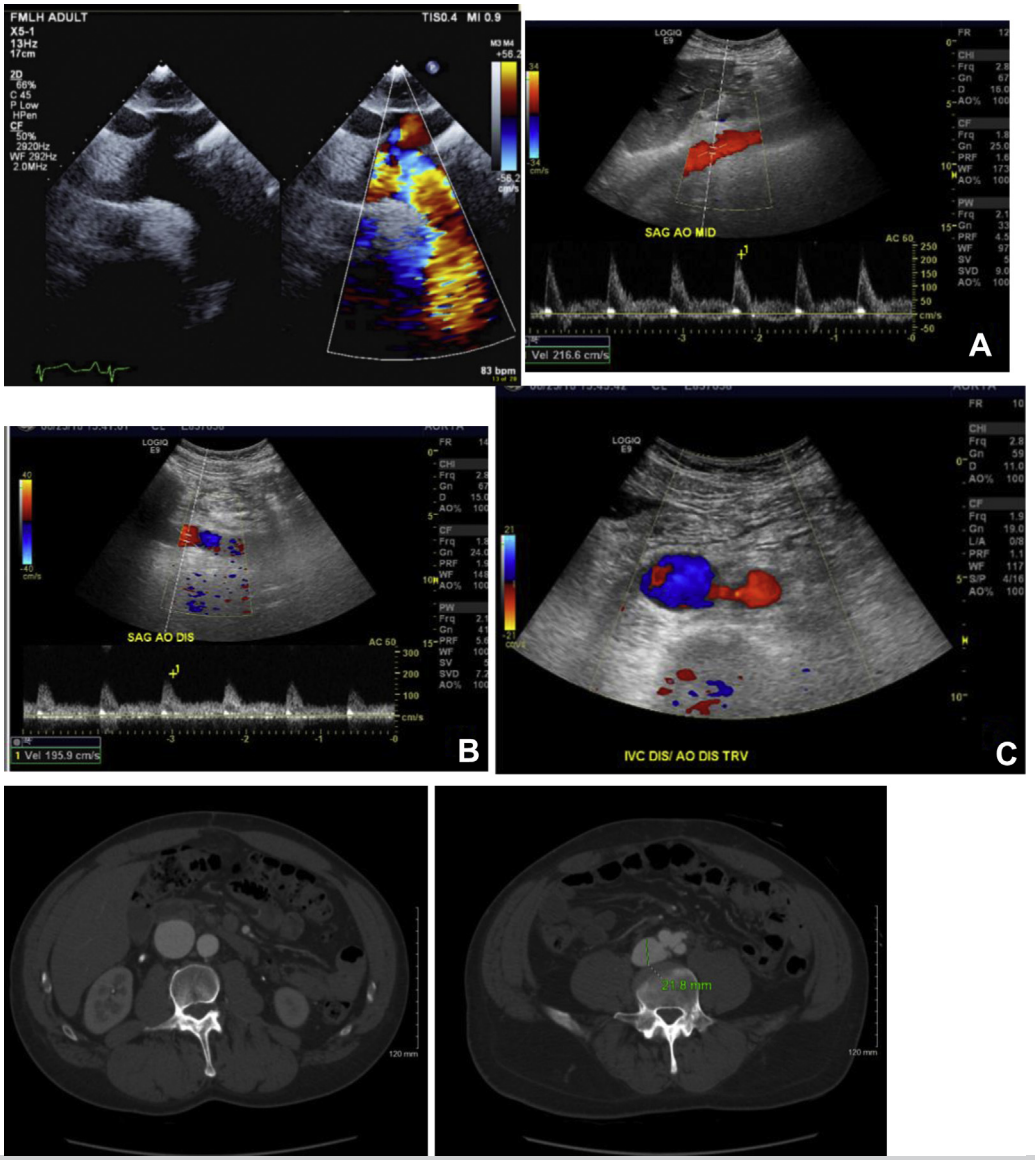

Fig 1. Turbulent flow in the aortic arch suggests a high cardiac output state (*top left*). Duplex ultrasound studies show suprarenal **(A)** and infrarenal **(B)** aortic flow as well as flow highly suggestive of an infrarenal aortocaval fistula (ACF; **C**). Computed tomography images (*bottom*) show marked inferior vena cava (IVC) distention and a serpiginous vessel connecting IVC to aorta.

on imaging. An abdominal ultrasound examination revealed a prominent IVC and hepatic veins and a normal aorta and detected no other abnormality. Left- and right-sided heart catheterization revealed mildly elevated left-sided filling pressures, moderately elevated right-sided filling pressures, small left to right shunt, and high cardiac output of 13.8 L/min by thermodilution method. Based on the patient's history, examination, and right-sided heart catheterization findings, an ACF was suspected and confirmed by vascular ultrasound and computed tomography (CT) angiography (Fig 1).

Medical management included initiation of antihypertensives, aggressive diuresis with salt and fluid restriction, and strict monitoring of intake and output. With diuresis and improved blood pressure control, the patient noted improved peripheral edema but persistent dyspnea on exertion (New York Heart Association class II symptom limitation). ACF repair was indicated for symptoms and correction of the vascular injury and right-sided heart failure physiology. Multidisciplinary case review resulted in a consensus recommendation for endovascular repair.

Our standard multidisciplinary approach involving interventional cardiology, interventional radiology, and vascular surgery was employed. Abdominal aortic angiography demonstrated a 7-mm infrarenal aortic defect immediately proximal to the bifurcation with an ACF (Fig 2, *A*) as well as a small pseudoaneurysm arising from the aortic bifurcation. The patient underwent endovascular repair with placement of a 22-40/13-40 bifurcated unibody stent graft (AFX; Endologix, Irvine, Calif) deployed in the

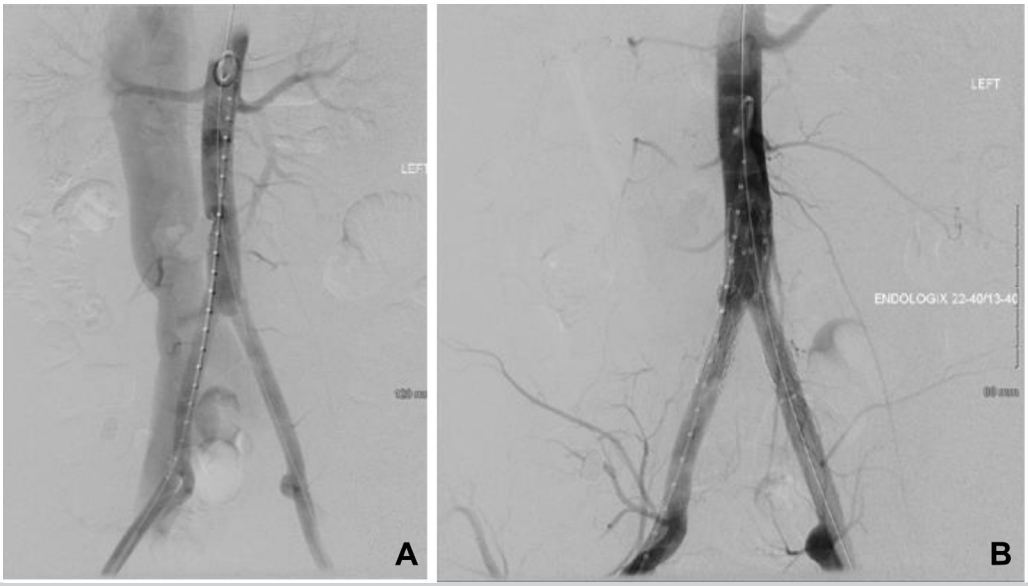

Fig 2. Digital subtraction angiography during procedure showing defect before **(A)** and after **(B)** stent repair.

aorta, followed by kissing balloon angioplasty of the aortic bifurcation. Completion aortography after device deployment demonstrated exclusion of the communication to the IVC (Fig 2, *B*). Hemodynamic measurements were obtained immediately before and after endovascular repair, demonstrating a decrease in cardiac output, right atrial pressure, and mean pulmonary artery pressure after graft deployment. The patient demonstrated marked improvement in blood pressure. Peripheral edema and complaints of exertional dyspnea resolved, and diuretic therapy was discontinued. Follow-up transthoracic echocardiography 1 month later demonstrated reduced RV size, improved RV performance, reduced severity of tricuspid regurgitation, and reduced estimated right-sided pressures. Follow-up CT angiography at 1 month and 8 months demonstrated patency of the endograft and persistent occlusion of the ACF with no other arterial abnormalities.

DISCUSSION

Arteriovenous fistulas can have sudden, catastrophic sequelae.[11] We report a case of chronic ACF causing high-output heart failure with right-sided chamber enlargement, elevated right-sided pressures, peripheral edema, and dyspnea on exertion. Left to right shunt is a known cause of right-sided heart enlargement. Although rare, chronic ACF has been known to cause high-output heart failure.[12] Our patient's history and physical examination findings suggested an arteriovenous fistula and ACF in particular. Despite initial imaging that was negative, persistence ultimately confirmed the diagnosis. The exact cause of the ACF is uncertain. However, based on the index operative report, a left-sided caval injury was probably missed during his laparotomy and repair. It is likely that the aortic repair broke down because of the use of absorbable suture, which in conjunction with a missed injury resulted in ACF. As demonstrated by Lidman and Daniel[13] in their study on healing of arterial anastomoses in rabbits, full-thickness healing of a vascular anastomosis does not occur. The healing process is typified by intimal hyperplasia without regeneration of the media, rendering an anastomosis susceptible to breakdown over years if absorbable suture is used. Therefore, use of permanent suture material for vascular anastomoses is strongly recommended.[13,14] Persistence or growth in size of the ACF led to the high-output physiology and eventual presentation of right-sided heart failure. Recognition and endovascular exclusion of the ACF led to reversal of adverse hemodynamics and positive right-sided heart remodeling. Delayed diagnosis or intervention, on the other hand, could have resulted in irreversible right-sided disease (Eisenmenger syndrome). Medical management without ACF correction is not recommended. As exemplified by our case, regular follow-up of patients suffering vascular trauma is prudent, and the practice of not scheduling follow-up after a single outpatient visit should be discouraged.

The options for mechanical correction include open and endovascular surgery. Surgical correction is invasive and is associated with significant morbidity and mortality with corresponding risks, costs, and delayed recovery.[7] Endovascular options, which may be favored to avoid laparotomy, to limit blood loss, and to allow local anesthetic,[14] include use of venous and arterial covered stents, vascular plugs including atrial septal defect closure devices, and aortic endografts.[15] Whereas surgical treatment of ACF had been the mainstay of therapy for several years, endovascular repair has recently been employed more frequently with good success

rates.[7,16-23] Close attention must be paid to the presence of superficial vein dilation, safe visualization of the renal arteries, and hemodynamic perturbances on stent graft deployment.[14] A literature review[16] published in 2016 analyzed 26 endovascular ACF repair cases. These repairs included several endograft types. An endoleak was reported in half the patients, with 38% of those cases requiring intervention. There were five deaths (19%) but no ruptured aneurysms. Complications included ischemia, stent prolapse, thrombus, and pulmonary embolism. There is concern that CT angiography may not appreciate endoleaks on completion angiography, and additional need for grafting of the IVC has been reported.[24] Of note, in this literature review, only nontraumatic ACF cases were examined.[16] In traumatic ACF cases due to GSW, a rare but potential complication is bullet embolization, which is likely to require open surgical repair.[25]

In our case, the diagnosis and treatment plan were respectively made and initiated by the cardiology service. The endograft planning and implantation were performed by interventional radiology and vascular surgery services using a co-surgeon model, congruent with our multidisciplinary institutional approach to aortic endografting during the last 20 years. This multidisciplinary team approach helped guide complex decision-making. The location of our patient's ACF near the aortoiliac bifurcation and the presence of an associated aortic pseudoaneurysm favored the use of a bifurcated unibody aortic endograft. Long-term sequelae from this approach were believed to be minimal, and the patient has done well in follow-up, with follow-up imaging confirming continued fistula occlusion. Had a type II endoleak been noted on follow-up imaging, our typical process of repair of endoleak would be followed and would be based on the anatomic characteristics; the transcaval approach may be technically more challenging, given his previous injuries, but this approach is rarely required.

The advancement of endovascular options for treatment of complex cardiac and vascular disease and the application of these technologies to aging and diseased populations may increase the future prevalence of iatrogenic ACF. Techniques such as transaortic valve replacement and transcatheter mitral valve replacement involve insertion of large-bore arterial and venous sheaths and increase the possibility of both recognized and unrecognized vascular injury, although to our knowledge no such case has been reported to date. The transcaval access technique purposely creates an ACF to enable passage of large-bore sheaths from the IVC into the aorta in patients with prohibitive arterial obstructive disease.[26] Although the ACF track is closed at the end of the procedure with placement of a vascular plug, complete closure is not always obtained. Long-term durability of partial or complete closure is not known.

High-output heart failure and ACF are both relatively uncommon conditions. Spencer et al[27] reported two cases of chronic ACF as a late complication of GSW. One patient declined intervention and was lost to follow-up, and another patient underwent surgical repair. Only four other cases of traumatic ACF treated with endovascular stent repair have been reported in the literature, three of which were repaired several years after the initial insult.[7,18,22] We encourage others to highlight and publish cases of salvage endovascular treatment of long-term delayed-onset complications of prior open surgical treatment of the aorta.

CONCLUSIONS

We report a case of subacute high-output heart failure resulting from a traumatic ACF. In the appropriate clinical setting, ACF should be considered a potential cause of right-sided heart failure. Vigilance is necessary and can help lead to proper diagnosis. It is our hope that our case report not only aids in diagnosis and appropriate therapeutic choices in similar clinical scenarios but also stimulates further discussion and research.

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