

Aortocaval fistula – rare complication of ruptured abdominal aortic aneurysms

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Aortocaval fistula (ACF) formation is a rare condition occurring in 0.2 - 1.3% of patients with degenerative aortic aneurysms.¹ This paper describes the presentation and successful management of a patient with such an ACF. We wish to highlight the need to maintain a high index of suspicion for this condition in patients with abdominal aortic aneurysms (AAA), particularly if they present with haematuria or are in congestive cardiac failure.

Case report

A 63-year-old man with a suspected ruptured AAA was transferred to Johannesburg Hospital by helicopter. On computed tomography (CT) scan, an AAA of 10 cm diameter with free fluid in the abdomen was found (Figs 1 - 3). An additional important feature that was evident on these images, but not initially appreciated, was the dilated inferior vena cava (IVC). Risk factors for the AAA included smoking and hypertension. There was a history of leg swelling for a few months and a 2-week history of abdominal pain and distension. On examination, the blood pressure was 154/69 mmHg. There were clear signs of right-sided heart failure, and a large pulsating, tender epigastric mass with a thrill was found on abdominal examination. An ECG demonstrated right-axis deviation with a right bundle-branch block (RBBB). Urine analysis revealed 3+ blood and 1+ protein.

The patient was in the operating theatre within 30 minutes of arrival at the hospital. A high-capacity central venous line and an arterial line were inserted before induction of general anaesthesia. The aneurysm was exposed via a standard generous midline laparotomy. A large amount of ascites but no blood was found on opening the peritoneal cavity. A standard tube graft repair of the AAA was performed except that, upon opening the aneurysm and removing a large intra-aneurysmal thrombus, a gush of deoxygenated blood was noted, leading to the identification of an aortocaval fistula. This was closed with a 3/0 prolene suture.

Postoperatively, the patient was rapidly weaned from the ventilator and inotropes in the ICU. The cardiac failure resolved rapidly; he was discharged from hospital 7 days after surgery. The postoperative course was complicated by an ileus which required re-admission, but this resolved spontaneously. The RBBB was no longer present on the ECG obtained during the re-admission.

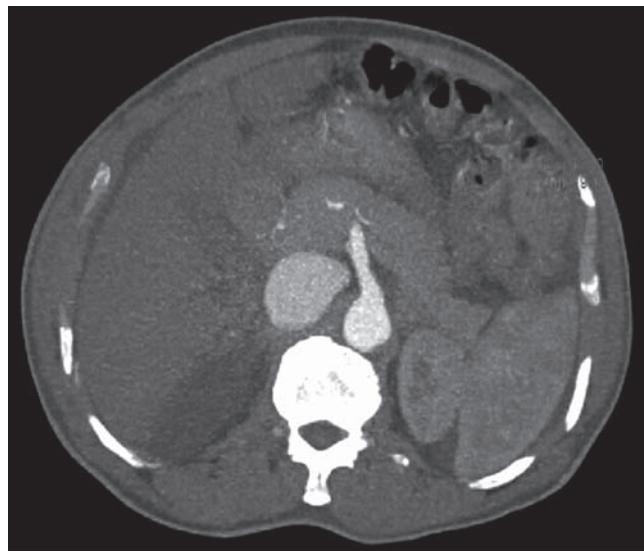


Fig. 1. Early filling of dilated IVC.

Discussion

ACF complicating an AAA (caused by syphilis) was first described by James Syme in 1831.² Today, an ACF is most commonly found in association with spontaneous rupture of a degenerative AAA. Uncommon pathologies such as Ehlers-Danlos syndrome, Marfan's syndrome and mycotic aneurysms have also been described as associated with an ACF.³

Some ACFs are missed owing to the lack of awareness of this condition, while others are asymptomatic and as a result are only discovered incidentally at the time of surgical repair. The onset of symptoms may be acute or insidious.⁴ The usual signs of an AAA with an ACF are high-output congestive cardiac failure (CCF) with a widened pulse pressure and relatively low diastolic pressure (as occurred in this patient), a continuous abdominal murmur or bruit, a pulsatile abdominal mass and renal insufficiency. These signs, however, only occur in approximately half of such patients.⁵ The reasons for this can be understood by considering the pathophysiology of each of these signs:

- Whether a continuous (machinery) murmur or bruit occurs, depends on the degree of turbulence. The degree of turbulence is predicted by Reynold's number, which increases with an increased velocity of blood flow (as occurs in the

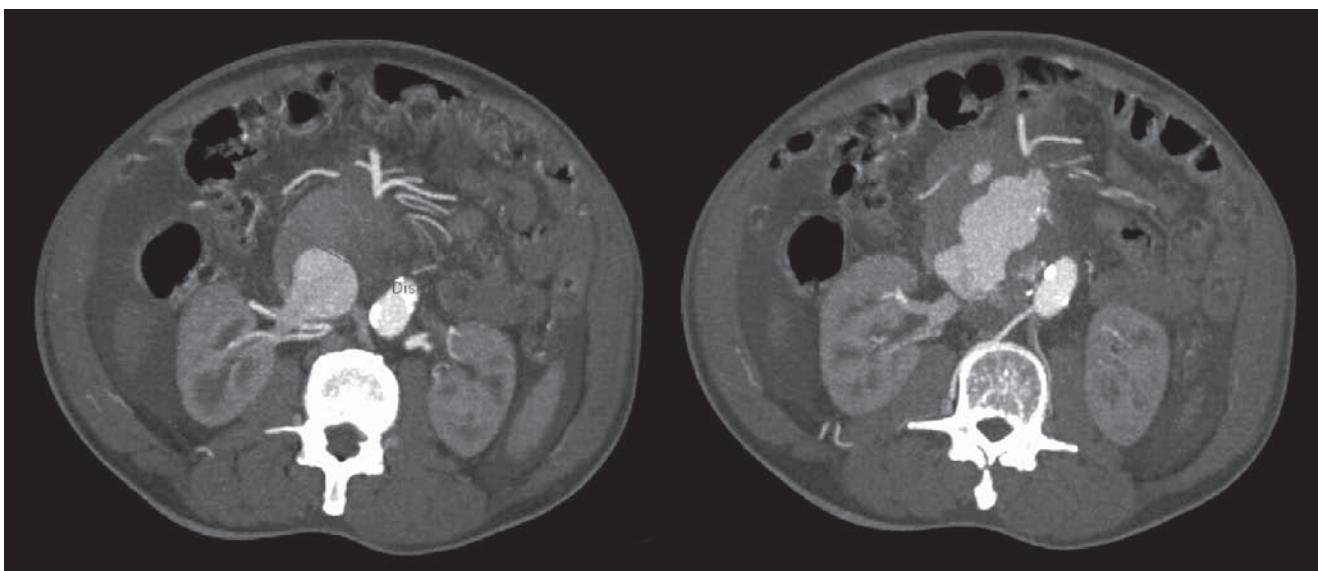


Fig. 2. Early filling of the IVC with irregular outline and large surrounding haematoma and free fluid.

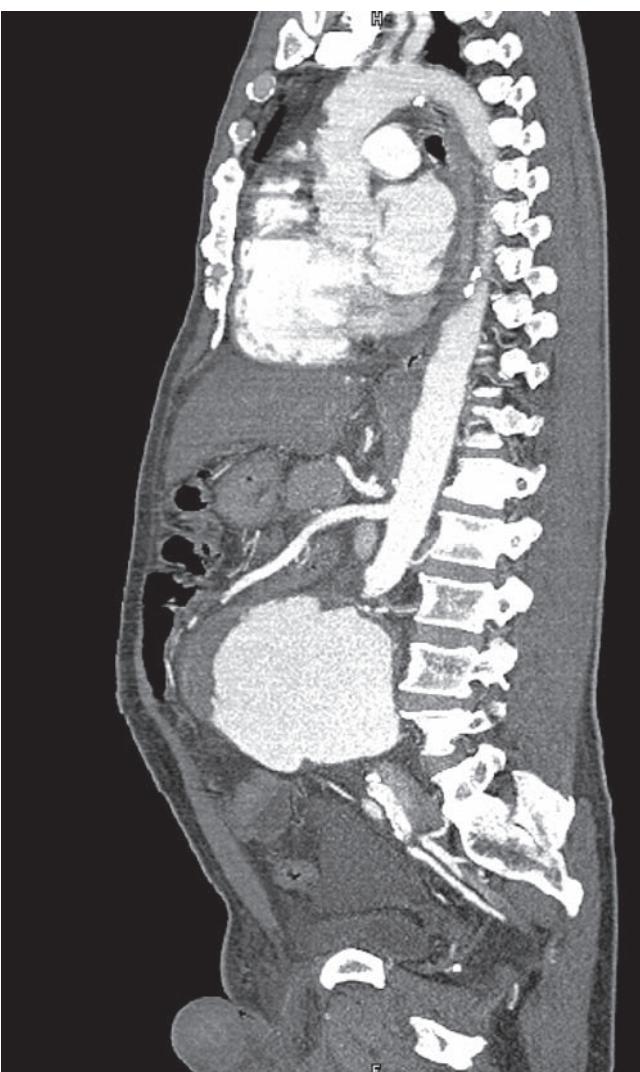


Fig. 3. Infrarenal AAA.

hyperdynamic circulation characteristically found in such patients), a wide diameter of the fistula (i.e. rather than a

narrow or thrombosed fistula) and decreased blood viscosity (e.g. anaemia).⁶

Similarly, the tendency for developing CCF is dependent upon the size of the fistula (and therefore the degree of the shunt which causes an increase in the cardiac preload) and the ability to compensate for this (which is related to the individual's pre-existing cardiac reserve).⁷

Patients with an ACF commonly have renal dysfunction which improves significantly after repair of the fistula.⁸ The pathophysiology of the renal dysfunction is unclear. It may not simply be the result of decreased renal blood flow caused by the heart failure but may also stem from increased central venous pressure which decreases the renal perfusion pressure, or redistribution of renal blood flow.⁹

Haematuria is not usually associated with symptomatic AAA and, as a result, it has been suggested that haematuria in a patient with an AAA should be regarded as having been caused by an ACF until proven otherwise.¹⁰ The haematuria associated with an ACF may be the result of rupture of venous varicosities in the bladder, or may be of a renal origin.

ACF is a rare cause of priapism but should be considered in elderly patients with high-flow priapism, not on erectile dysfunction medications, and especially if the individual is known to have an AAA.¹¹

Paradoxical pulmonary embolism is a surprisingly rare association of ACF.¹²

A contrast-enhanced CT scan has become the standard method for making a definitive pre-operative diagnosis in haemodynamically stable patients. The diagnosis may also be made with magnetic resonance imaging (MRI), duplex Doppler ultrasound or digital subtraction angiography.¹³ The most distinctive finding on a CT scan is a rapid flow of contrast from the aorta into the dilated IVC.¹⁴

The standard approach to the repair of an ACF is open surgery. The major hazards of AAA repair in the presence of an ACF are arterial and pulmonary emboli (thrombus, atherosclerotic debris and air) and excessive bleeding. To prevent pulmonary embolisation from occurring, the IVC should be controlled before opening the aneurysm by intra-vascular balloons inserted via the femoral vein or by manual compression – dissection of the IVC and iliac veins is no

longer advocated¹⁵ because ACFs usually occur in the distal IVC just above the confluence of the iliac veins (because the aorta is relatively fixed at this point and closely applied to the IVC at this level),¹⁶ which makes mobilisation of the vena cava hazardous. The fistula is usually closed primarily using non-absorbable sutures from within the aneurysmal sac, although there have been reports of repair with a Dacron patch, IVC ligation or aortic exclusion (the AAA is excluded from arterial circulation which subsequently thromboses due to stasis).¹⁷

Recently, endovascular stent grafts have been shown to be an alternative definitive treatment option¹⁸ and have been suggested as a potential 'bridging' procedure to allow cardiac recompensation, restoration of associated organ failure and reduction of perioperative risk for subsequent open repair.¹⁹

Postoperatively, one should be aware of the risk of IVC thrombosis due to the IVC repair.²⁰

Conclusion

ACFs are rare complications which frequently present with unusual symptoms and signs. They should, however, be actively excluded in patients with AAA who also are in CCF or have haematuria. In addition, they require special intraoperative consideration which may necessitate appropriate changes to the usual operative technique.

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