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CASE REPORT

Acute Global Testicular Infarction Post-EVAR from Cholesterol Embolisation can be Mistaken for Torsion

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Introduction: Endovascular aneurysm repair (EVAR) is the most commonly used approach for treatment of abdominal aortic aneurysms (AAA). Testicular infarction is a rare complication of EVAR. A novel case of acute global testicular infarction post-EVAR from cholesterol embolisation mimicking torsion is presented.
Report: A 75 year old man developed acute right testicular ischaemia requiring orchidectomy following EVAR of an infrarenal aortic aneurysm. The patient was initially diagnosed with testicular torsion as the aetiology of the infarction; however, on re-analysis of histopathology it was found to be secondary to cholesterol emboli.
Discussion: In patients complaining of groin/scrotal pain following EVAR, it is worth considering testicular ischaemia whether secondary to cholesterol embolisation or gonadal occlusion. Clinicians should be aware that clinical and radiological findings can mimic torsion as this affects management and outcome.

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INTRODUCTION

The prevalence of abdominal aortic aneurysm (AAA) rises sharply with increasing age, affecting approximately 4–7% of men and 1–2% of women over the age of 65 years.[1] Rupture is associated with high mortality unless surgical repair can be performed urgently. Currently, endovascular aneurysm repair (EVAR) is the preferred method of surgical repair for abdominal aneurysms, owing to the low peri-operative morbidity and mortality.[1] In this report the case of a 75 year old man, who had an acute global infarct of his right testis secondary to cholesterol embolism following EVAR of infrarenal aortic aneurysm, which was initially diagnosed as testicular torsion, is presented. Only four cases of testicular ischaemia/infarction following EVAR have been reported in the literature, with only one being secondary to histologically confirmed cholesterol emboli.[2–5]

REPORT

A 75 year old man underwent EVAR for repair of a 5.4 cm asymptomatic infrarenal AAA. The patient's past medical history included ischaemic heart disease and coronary artery bypass graft in 1995, hypercholesterolaemia, bowel resection secondary to chronic appendicitis, and previous transurethral resection of prostate. The patient was an ex-smoker. After pre-operative investigations and anatomical assessment of the aneurysm and access, the patient was deemed suitable for EVAR (Fig. 1A).

The patient underwent an uncomplicated procedure involving a bilateral percutaneous approach via both common femoral arteries with 5,000 units of heparin given intra-operatively, and preservation of both internal iliac arteries. Immediately post-procedure it was noted that the patient began to complain of right groin pain. Examination did not reveal any haematoma or bruising; however, the patient was tender over the inguinal canal and scrotum. On palpation the abdomen was soft and non-tender. Assessment of vital signs was within normal parameters, and biochemistry revealed a normal haemoglobin and lactate. The working diagnosis at this stage was presumed to be pain secondary to the arterial puncture. The patient was commenced on patient controlled analgesia overnight with a view to obtain a computed tomography angiogram of the aorta if pain persisted, increased, or if there was any clinical deterioration.

The patient had ongoing pain the next day, but with some improvement. On re-examination the patient had a tender right testicle. There was no scrotal or inguinal swelling or tenderness. An urgent ultrasound of the scrotum was

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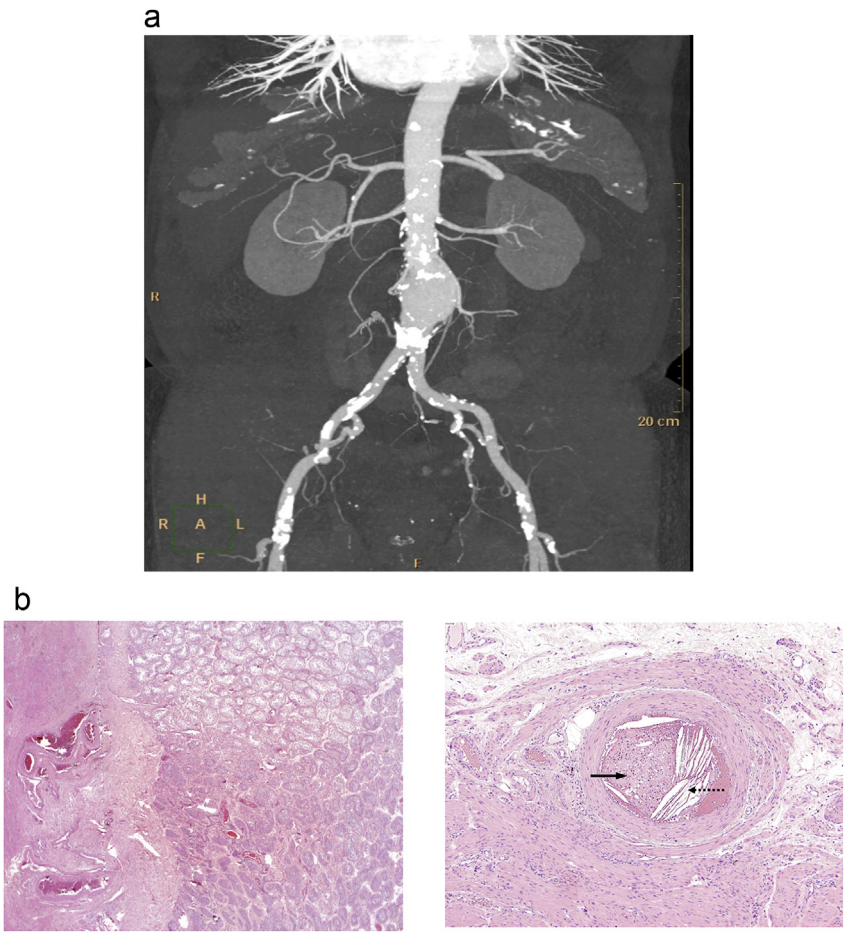

Figure 1. (A) Three dimensional reconstruction of the computed tomography angiogram showing the position and morphology of the infrarenal aortic aneurysm. (B) (Left) Low power view (×20 magnification) showing patchy infarction of the testicular parenchyma. (Right) High power view (×100) showing partially thrombosed artery containing fibrin, inflammatory cells (solid arrow), and cholesterol crystals (dotted arrow).

performed revealing an abnormal lie of the right testicle with decreased blood flow, commented on by the radiologist to be consistent with testicular torsion. Specialist urology review determined the right testicle to be swollen, tender, and high riding with a horizontal lie, in keeping with the radiological findings of suspected testicular torsion. It was explained to the patient that as the symptoms had persisted over 24 h, the testicle was likely to have infarcted. The patient underwent scrotal exploration. Intra-operatively, the testicle was necrotic and hence removed, but no evidence of torsion was found.

Histopathological examination concluded that there was patchy testicular infarction in keeping with the clinical impression of torsion. The patient recovered well post-operatively and was discharged home. The patient re-presented 1 week post-operatively to the emergency department with tenderness and dusky purple discolouration of the left hallux. The patient had a palpable left popliteal pulse with cold toes, but the remainder of the foot was warm. The clinical impression of the patient's presentation was that of probable distal embolisation ("trash") causing microvascular ischaemia, raising suspicion that the right testicular infarction may have occurred via the same mechanism given the lack of intra-operative findings to support testicular torsion.

The testis was extensively further sampled and the original pathology blocks were re-examined, and within the testicular parenchyma several small thrombosed arteries containing cholesterol crystals were identified, concluding that the parenchymal infarction was secondary to cholesterol emboli (Fig. 1B).

DISCUSSION

Surgical repair of abdominal aneurysms can be done via an open or endovascular approach. Three major randomised trials have demonstrated significantly lower operative mortality of EVAR compared with open repair, and as a result it is the primary treatment option for patients with AAAs if they are morphologically suitable.[1]

According to the current literature, general ischaemic complications occur in 3—10% of patients undergoing EVAR. The incidence of embolic ischaemia is approximately 2%, including pelvic and limb ischaemia.[6] End organ embolic injury is prevented in open surgery by cross-clamps, whereas flow is maintained during EVAR while intraluminal manipulation of wires, catheters, and devices is occurring, and

plaque and thrombus can be dislodged. Thompson et al. reported that particulate embolisation to the lower limbs occurred more frequently in patients undergoing EVAR.[7] This was detected by insonation of the superficial femoral artery with a Doppler probe.[7] Cholesterol crystal embolisation due to athero-emboli is associated with significantly high rates of morbidity and mortality.[6]

Dadian et al. describe microembolisation as a major cause of post-operative colonic ischaemia in patients undergoing EVAR, with high mortality associated with widespread embolisation.[8] Of the patients with colon ischaemia four (50%) had direct evidence of microembolisation, three of whom died from widespread embolisation and two required colectomy. Toya et al. describe nine patients with embolic complications (2%) of whom four developed ischaemic colitis (0.91%) in their group of 438 patients.[6]

Lower extremity ischaemia after EVAR, or "trash foot", is a well documented complication, occurring in 1—5% of patients. The pathophysiology of this is thought to be dislodgment of atherosclerotic plaque causing cholesterol embolisation, which obstructs the distal vascular bed resulting in ischaemia. Trash foot can be treated medically with vasodilators, statins, and anticoagulation, but toe amputation may be required.[6]

Spinal ischaemia from embolisation is a rare but devastating complication of EVAR with persistent paraplegia. An incidence of 0.2—0.3% is documented in the literature, with cases diagnosed on magnetic resonance imaging.[9]

Renal embolic injury after EVAR has been documented in several studies, with an incidence of 5—6% after elective repair.[10] However, renal cortical defects after EVAR do not necessarily result in impaired renal function.[10]

To date there have been only four documented cases of testicular ischaemia/infarction post-EVAR. It is interesting to note that an extensive review of the available literature revealed no documented cases of testicular ischaemia or infarction related to open repair of abdominal aneurysms, suggesting that collateral circulation develops as thrombus in the aneurysm sac occludes inflow over time. In a case report by McKenna et al.,[2] a 67 year old man presented 6 weeks post-EVAR with left sided scrotal pain, with ultrasound confirming absence of arterial perfusion to the testis. A subsequent emergency orchidectomy was performed for ischaemic infarction. Histopathology reported that left testicular necrosis was secondary to thrombus in the testicular artery.[2]

Hall et al. report a similar case with delayed presentation one week post-EVAR with testicular pain, and ultrasound confirmed testicular ischaemia.[3] However, in this case the patient was managed conservatively, and the testicle was noted to be infarcted at the 6 month follow-up, with the patient asymptomatic. The aetiology of ischaemia was hypothesised to be due to occlusion of the left gonadal artery during deployment of the graft, with inadequate collateral flow to the testis, delayed occlusion of collaterals, and progressive thrombus in the aneurysmal sac.

Finnerty et al. provide an alternative case of right testicular ischaemia following EVAR in a patient who presented 2 days post-EVAR with right flank, lower abdominal, and testicular pain.[4] However, this patient was managed conservatively with serial examinations, and was discharged 3 days post-admission with stable blood flow to the testis.

Milburn et al. present the only other documented case of testicular infarction following EVAR secondary to cholesterol emboli.[5] However, in this case the patient was asymptomatic and underwent urgent orchidectomy of the left testicle after noting an incidental left testicular mass suggestive of malignancy while undergoing investigation for a contralateral epididymal cyst. Histological features were in keeping with subacute segmental testicular infarction with central necrosis surrounded by viable peripheral testicular tissue.

The present case is the first reported in the literature of acute global infarction of the testis secondary to cholesterol emboli following EVAR. In the other documented cases, patients presented with delayed symptoms of pain with onset ranging from 2 days to 6 weeks post-procedure. Notably, reports by McKenna et al. and Milburn et al. attributed coil embolisation of the internal iliac as a potential cause of thromboembolic testicular ischaemia.[2,5] In this case, the internal iliac artery was not coiled or lost (Fig. 2), and it is believed the cholesterol emboli originated from the irregular and flared atherosclerotic plaque at the neck of the aneurysm, which was dislodged on graft deployment and embolised via the gonadal artery (Fig. 3A). The thoracic aorta was relatively disease free (Fig. 3B).

Outcomes after EVAR for AAAs with neck thrombus were evaluated by Shintani et al.,[10] who concluded this group had higher rates of renal and lower limb thromboembolic complications. The present patient not only had plaque in the neck of the aneurysm, but also had a right gonadal

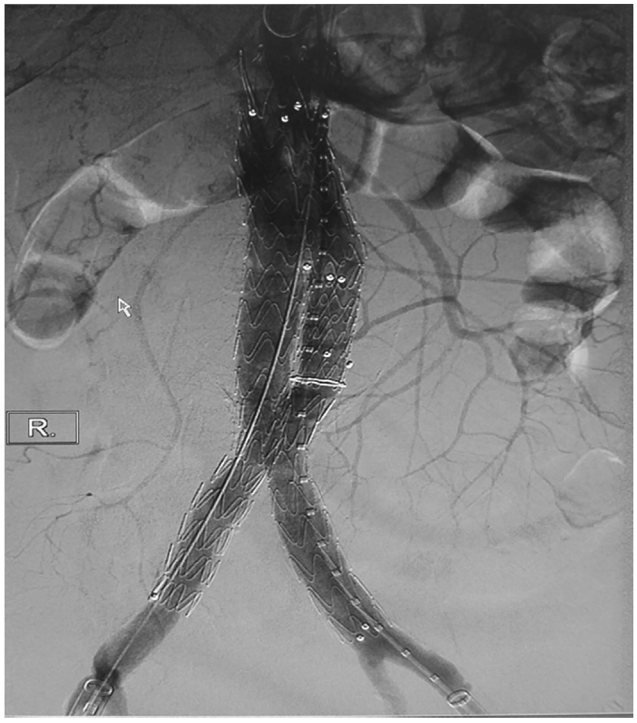

Figure 2. Patent internal iliac arteries post-endovascular aneurysm repair.

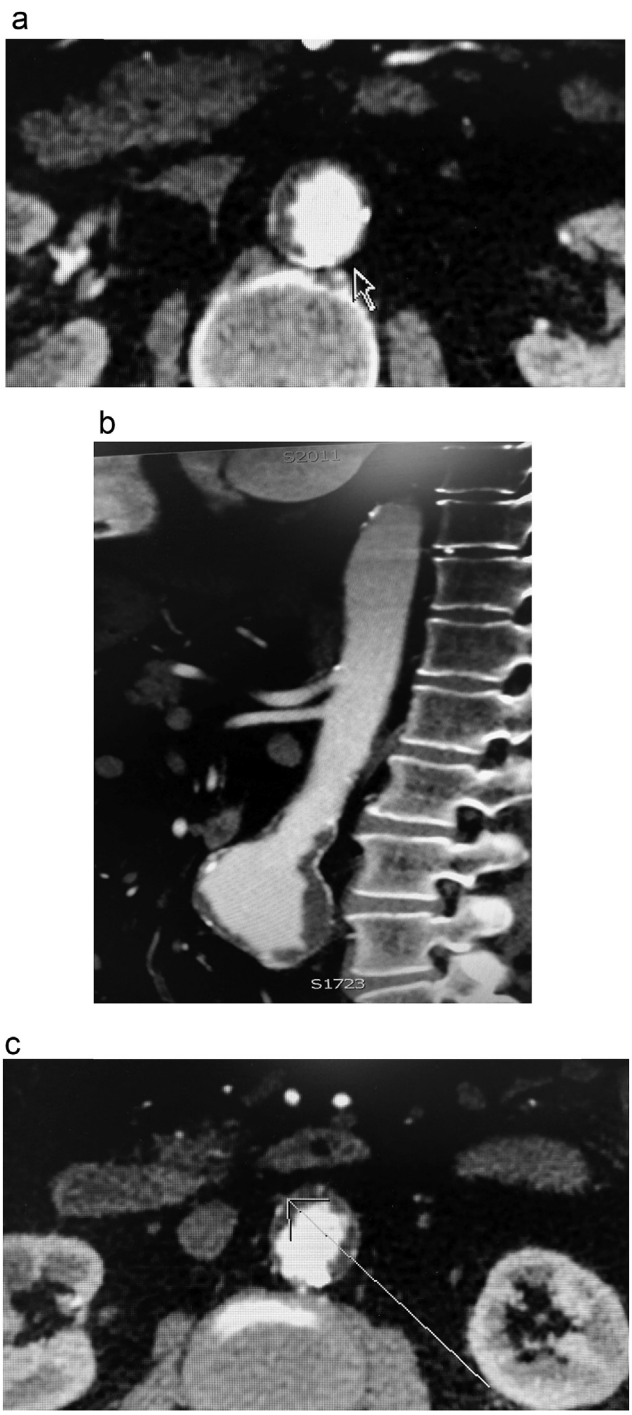

Figure 3. (A) Contrast enhanced computed tomography (CT) angiogram showing disease in the aortic aneurysm neck proximal to the origin of the gonadal artery. (B) Contrast enhanced CT angiogram showing thoracic aorta relatively free of disease. (C) This contrast enhanced CT angiogram shows the right gonadal artery arising from the thrombus filled aneurysm sac (shown by arrow).

artery arising directly from the thrombus-filled aneurysm sac (Fig. 3C). Furthermore, flow in the right gonadal artery was found to be poor on the pre-operative computed tomography scan, making the testis more vulnerable to ischaemic insult. Posterior plaque was also noted at the level of the renal artery (Fig. 3A); however, serial creatinine measurements in the patient did not reveal any renal dysfunction. Raised eosinophil count can be a marker of cholesterol embolisation and is observed in 70—80% of patients. No raised eosinophil count was present in the current patient.[6]

In patients with acute testicular pain following EVAR, distinguishing between testicular torsion and embolic ischaemia is important in the management. Torsion always requires surgical intervention to prevent infarction, whereas embolisation would not be improved by surgery; in fact, surgery may be unnecessary unless infarction is complete. Also, as seen in previous cases, embolisation may not lead to infarction of the entire testis and some function may be retained.[4]

CONCLUSION

EVAR is currently the most commonly used approach for repair of AAAs. Testicular infarction post-EVAR is a rare occurrence. The first reported acute case of global testicular infarction secondary to cholesterol emboli post-EVAR is presented. It is important to note that clinical and radiological findings can mimic torsion. In patients complaining of groin/scrotal pain post-EVAR, it is worth considering testicular ischaemia whether secondary to cholesterol embolisation or gonadal occlusion as this affects management and outcome.

CONFLICT OF INTERESTS

None.

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