Acute global testicular infarction: A rare and important complication after endovascular abdominal aortic aneurysm repair

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ABSTRACT

Testicular ischemia is one of the most rarely reported complications of endovascular abdominal aortic aneurysm repair (EVAR). Although the pathogenesis remains unclear, thromboembolic events in the setting of testicular artery origin occlusion by the stent graft and poor baseline collateral testicular circulation are presumed causes. A 73-year-old man developed acute right testicular infarction 3 days after EVAR, requiring orchiectomy. This case emphasizes the importance of recognizing and evaluating testicular pain after EVAR and counseling patients on this possible EVAR complication. (J Vasc Surg Cases Innov Tech 2024;10:101522.)

Keywords: EVAR; Testicular ischemia; Complication; Cholesterol emboli; Testicular artery; Endovascular abdominal aortic aneurysm repair; Orchiectomy; Testicular infarction

Testicular ischemia after endovascular abdominal aortic aneurysm repair (EVAR) is emerging as a rare, but serious, complication. To the best of our knowledge, there have been only 12 cases of post-EVAR testicular ischemia reported worldwide,[1-12] and this is the fourth case reported in the United States.[3,4,6,9] This report aims to highlight the importance of early recognition of this complication and contribute to our understanding of its pathophysiology.

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committees and with the Declaration of Helsinki. The patient provided written informed consent for the report of his case details and accompanying imaging studies.

CASE REPORT

A 73-year-old man with a 6.6-cm infrarenal abdominal aortic aneurysm (AAA) presented for elective endovascular abdominal aortic aneurysm repair (EVAR). His relevant medical history included coronary artery disease, diabetes mellitus type 2, congestive heart failure, and cardiac catheterization with a drug-eluting stent placed 1 month before EVAR.

A percutaneous approach from the groin was used for all the steps of the procedure. Because the patient had a patent inferior mesenteric artery (IMA) and patent internal iliac arteries with patent collateral colonic arterial circulation, we elected to perform IMA embolization to prevent a type II endoleak. In patients with a patent IMA, we perform IMA embolization on a case-by-case basis depending on the size of the IMA at the aortic orifice, the patency of the internal iliac arteries, and satisfactory collateral perfusion of the distal colon from the superior mesenteric artery. In this case, before deployment of the stent graft, the origin of the IMA was cannulated and coil embolized with 2.6-cm, 4-3 and 5-3 Tornado embolization coils (Cook Medical Inc). An angiogram after embolization showed excellent placement of the coils, all within the proximal IMA without concern for distal embolization. A Gore Excluder AAA Endoprosthesis (W.L. Gore & Associates) was then deployed. No difficulties were encountered with groin access, IMA cannulation, or stent graft deployment that would require excessive wire or device manipulation. A completion angiogram demonstrated bilateral internal iliac artery perfusion, aneurysm exclusion without endoleaks, and no evidence of embolization.

On postoperative day 3, the patient developed new-onset severe right testicular pain. Examination revealed a grossly normal-appearing scrotum and testis. Testicular ultrasound demonstrated no arterial flow with decreased venous flow and early parenchymal changes, suggesting testicular infarction (Fig 1). Urology deemed testicular torsion unlikely given the patient's physical examination findings and age. Nonoperative management vs scrotal exploration were discussed with the patient. The patient elected to proceed with scrotal exploration.

Intraoperatively, the right testis was grossly ischemic without evidence of testicular torsion. Right orchiectomy was performed. The final pathology examination demonstrated parenchymal infarction with background ischemic and hemorrhagic changes. A review of the patient's preoperative computed tomography angiogram demonstrated opacification of the right testicular artery originating from the aorta despite a moderate amount

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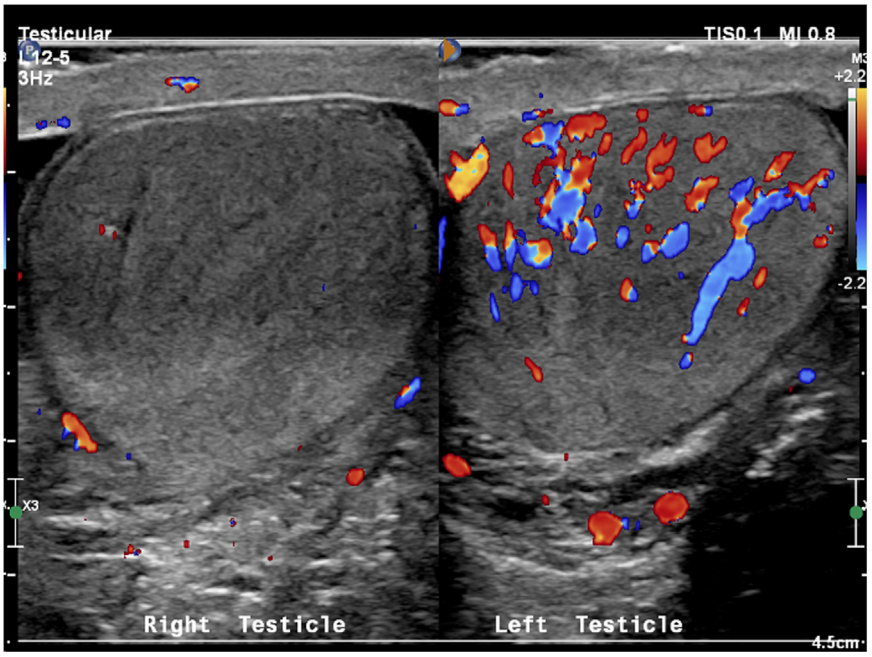

Fig 1. Testicular ultrasound scan showing minimal to no color flow in the right testis, a hyperemic right scrotal wall and peritesticular tissues, and a normal ultrasound appearance of the left testis.

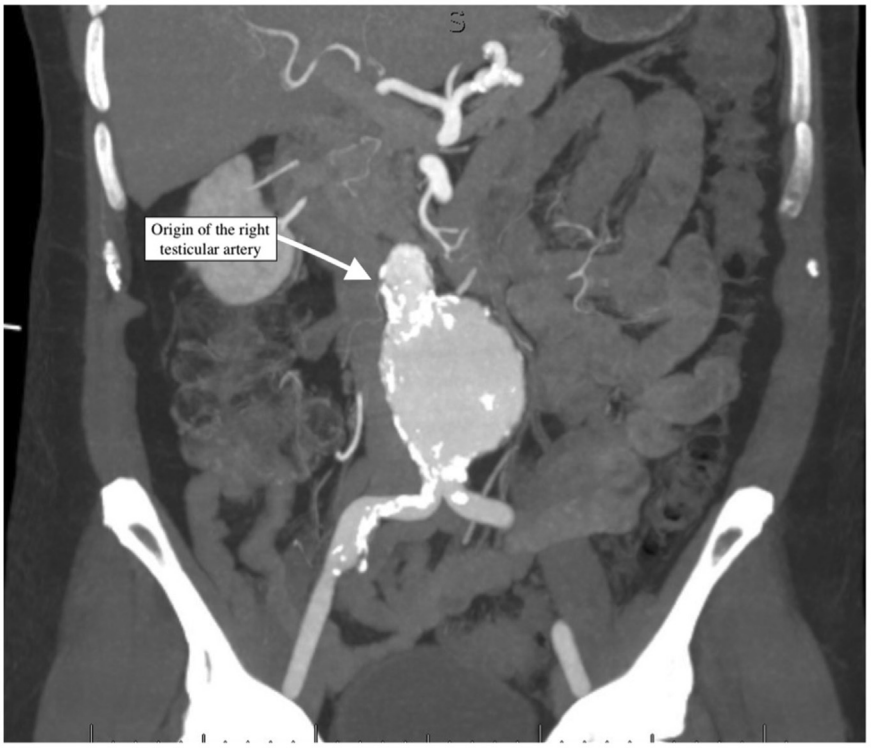

Fig 2. Preoperative computed tomography angiogram, coronal view, showing the right testicular artery originating from the anterolateral aspect of the abdominal aorta.

of aortic wall calcification at its takeoff (Fig 2). A collateral supply from the right deferential and cremasteric arteries to the right testis was not visualized on the preoperative imaging studies.

DISCUSSION

Based on a review of the available case reports, post-EVAR testicular ischemia is characterized by acute inguinoscrotal pain immediately postoperatively to ≤6 weeks

after EVAR. Testicular ultrasound findings include absent or decreased Doppler signals within the testicular parenchyma. It is important to differentiate post-EVAR testicular ischemia from testicular torsion because the management differs. Although testicular torsion always requires operative intervention, post-EVAR testicular ischemia might not necessitate surgery. In the absence of global testicular infarction, conservative medical treatment with nonsteroidal anti-inflammatory drugs has been reported to be effective.[1-4]

Global testicular infarction is rare due to the collateral blood supply. The blood flow to each testis is supplied by three arteries: the testicular, deferential, and cremasteric. The testicular arteries arise directly from the infrarenal aorta and serve as the primary blood supply to the testes, supplying 80% of the testicular volume.[2] The cremasteric artery, a branch of the inferior epigastric artery, provides only minimal blood flow to the testicular parenchyma. The deferential artery is a branch of the inferior vesicle artery that originates from the internal iliac artery. It primarily supplies the epididymis and vas deferens but also anastomoses with branches of the testicular artery within the testis, supplying nearly 20% of the testicular volume.[2] A study using an injection-corrosion casting technique to establish a three-dimensional topography of the testis showed that all examined testes had testicular, deferential, and cremasteric arteries with clear anastomotic communications between the three.[13] Given this collateral network, occlusion of the testicular artery by the stent graft during EVAR should not typically lead to symptomatic testicular ischemia.

The pathophysiology of testicular ischemia after EVAR remains poorly understood and is suspected to be multifactorial. One proposed etiology is cholesterol embolization into the distal testicular arterial network as a result of cholesterol plaque dislodgement during wire and catheter maneuvering and stent graft deployment. Histopathology in prior case reports noted the presence of cholesterol emboli within the testicular parenchyma after orchiectomy.[5,6] Specifically, in the case report by Pathmarajah et al,[5] the patient developed symptoms immediately after EVAR and underwent orchiectomy with pathology findings of thrombosed arteries containing cholesterol crystals. We can speculate that embolic etiology of testicular ischemia would result in a more acute onset of symptoms such as reported by Pathmarajah et al[5] and seen in our patient.

Delayed testicular ischemia after EVAR presented in several prior case reports could be attributed to the eventual thrombosis of the aneurysmal sac with resultant testicular artery thrombosis secondary to a low flow state. Thrombosis could also be delayed by the presence of a negligible type II endoleak from patent lumbar arteries or IMA, which would provide some flow to the testicular artery. As a result, delayed testicular ischemia might not be recognized as a post-EVAR complication and, thus, could be underreported. In our case, because the IMA was embolized during EVAR, there was no residual type II endoleak that could have contributed to perfusion of the testis while collateral circulation develops, potentially resulting in acute testicular infarction.

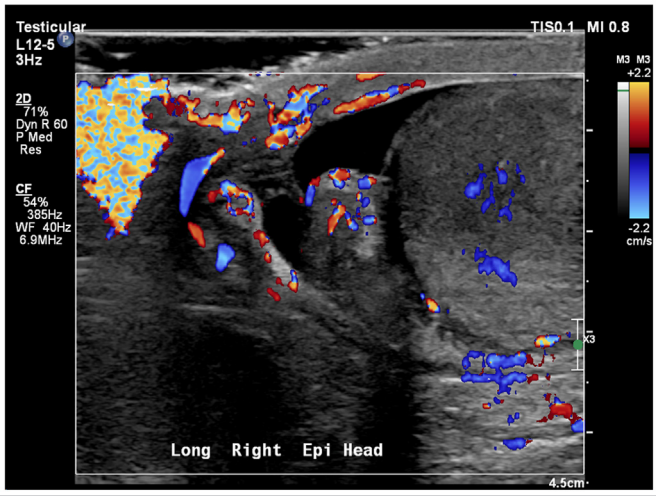

Fig 3. Doppler ultrasound showing perfusion of the epididymal head.

Another factor that could contribute to testicular ischemia after EVAR is occlusion or embolization of the internal iliac artery. McKenna et al[7] reported a case of testicular infarction 6 weeks after EVAR with ipsilateral internal iliac artery embolization. Histopathology demonstrated thrombus in the testicular artery and parenchymal ischemic necrosis.[7] Because the internal iliac artery gives rise to the deferential artery, occlusion of the internal iliac artery, coupled with the testicular artery occlusion by the endograft, would critically decrease testicular blood flow.

In our patient, the onset of postoperative testicular ischemia was relatively acute. As is customary with the EVAR procedure, our patient's bilateral testicular arteries were occluded by the stent graft, leaving testicular perfusion to rely on collateral blood flow from the deferential and cremasteric arteries. Although there was no arterial flow within the right testicular parenchyma on Doppler ultrasound after the onset of symptoms, the perfusion of the right epididymal head continued to be adequate (Fig 3). This suggests that the deferential artery, which supplies most of the blood flow to the epididymis, was patent. However, there was no evidence of collateral vascularization from the deferential and cremasteric arteries, leading us to conclude this represented an embolization event. Inadvertent embolization of the testicular artery by coils during coil embolization of the cannulated IMA would be unlikely given that the right testicular artery was arising from the anterolateral aspect of the

abdominal aorta (Fig 2) and not IMA. To date, we could identify only one case of a testicular artery origin from the IMA.[14]

Furthermore, completion angiogram demonstrated all coils within the proximal superior mesenteric artery. However, this additional step of IMA embolization with extra wire manipulation could have contributed to dislodgement of substantial atherosclerotic plaque at the origin of the testicular artery (Fig 2).

With the growing number of reports of post-EVAR testicular ischemia, this complication should be included in the preoperative risks discussion. Due to the generally brief hospital course associated with EVAR, the incidence of testicular ischemia and infarction after EVAR is likely underreported. Interestingly, no cases of testicular ischemia have been reported after open surgical AAA repair, underscoring the preference of open surgical repair for the medically fit and, especially, young patients.

CONCLUSIONS

With the advent of EVAR as a first-line treatment for patients with anatomically suitable AAA, it is important to recognize testicular ischemia as one of its rarely reported complications. The pathophysiology, incidence, and prevention of post-EVAR testicular ischemia remain areas for ongoing investigation. Due to the low incidence rate of testicular ischemia after EVAR, it is difficult to establish an effective method to predict and prevent this complication. We hope that reporting post-EVAR testicular ischemia cases will help establish the framework for disease pattern recognition and improve our understanding of its pathophysiology.

DISCLOSURES

None.

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