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Inflammation and infection

Testicular ischemia secondary to epididymo-orchitis: A case report

Badr Alharbi[a,*], Emad Rajih[b], Adewunmi Adeoye[c], Bashar Abed Allatiefe[d], Mohammad Hasan Abdullah[d]

[a] Urology Department, College of Medicine, Qassim University, Buraidah, Saudi Arabia
[b] Urology Department, College of Medicine, Taibah University, Madinah, Saudi Arabia
[c] Pathology Department, King Fahd Specialist Hospital, Buraidah, Saudi Arabia
[d] Urology Department, King Fahd Specialist Hospital, Buraidah, Saudi Arabia

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ABSTRACT

Testicular infarction is a common urological emergency in clinical practice, it is still underreported when it results from other than spermatic cord torsion. It rarely arises from other pathological processes like vasculitis and infectious disease. We report a case of 18-year-old with epididymo-orchitis complicated by testicular ischemia. This case accentuates the need for a high index of suspicion to rule out concomitant testicular ischemia secondary to epididymo-orchitis that might be salvaged in the future with immediate surgical intervention. Testicular infarction is a devastating complication from epididymo-orchitis that is difficult to predict and distinguish from more common presentations of acute scrotum.

Introduction

Epididymo-orchitis is a common urological disorder in clinical practice and one of important differential diagnoses of acute scrotum. Medical management is the mainstay therapy that include antimicrobial and anti-inflammatory treatment. Unusually, severe and progressive infectious processes involving the epididymis and testicle result in testicular loss. Testicular infarctions have been reported rarely as a catastrophic complication to epididymo-orchitis.[1] We report a case of unresolved and progressive epididymo-orchitis complicated by scrotal abscess and testicular infarction.

Case report

An 18-year-old young man presented to the emergency department with left scrotal pain and swelling for two days. His pain was intermittent initially and increased in severity over time, dull aching in nature, and radiating to his left inguinal area. He denied any chills, rigors, trauma, or similar episodes in the past. He had no lower urinary tract symptoms and unremarkable past medical and surgical history. Physical examination revealed normal vital signs at time of presentation. Genital examination showed left epididymal swelling and tenderness with normal overlying scrotal skin. Investigation revealed negative urine analysis, blood workup showed mild leukocytosis (WBC) of 11×10^9/L. Ultrasound doppler showed enlarged left epididymis with heterogeneous echopattern and increased vascularity suggesting epididymitis and preserved testicular blood flow (Fig. 1-A). Oral ciprofloxacin and doxycycline with non-steroidal anti-inflammatory were commenced and discharged home with out-patient follow-up.

Two days later, he revisited the hospital because of unresolved symptoms and experiencing chills and fever. Physical examination revealed temperature of 37.8 °C and interval increased swelling and tenderness over epididymis extending to ipsilateral testis. WBCs increased to 17.8×10^9/L and repeated sonography showed heterogonous echotexture left testes and epididymis with decreased but present blood flow to the left testes.

Septic screening including urine and blood culture were taken and in-patient parenteral Tazocin 4.5 g three times a day (piperacillin/tazobactam) was started. Hospital course showed no improvement with progressive clinical derangements. The WBC counts reached 33×10^9/L and repeated scrotal doppler ultrasound showed absent blood flow to the left testicle as well as development of scrotal abscess (Fig. 1-B). Operative scrotal exploration revealed black discoloration of the left testes and scrotal abscess without evidence of testicular torsion. The left testis was wrapped in warm sponge followed by drainage of the abscess. The testis showed persistent black discoloration and orchiectomy was performed (Fig. 2). Post-operatively, he improved dramatically and became afebrile. The WBC counts decreased to 9×10^9/L on day three

* Corresponding author. Consultant of Urology, MIS/Andrology and Infertility Head, department of Surgery College of Medicine, Qassim University P.O. Box 6633, Buraidah, 51452, Saudi Arabia.dr_badr@hotmail.com
E-mail address: badralharbi@qumed.edu.sa (B. Alharbi).

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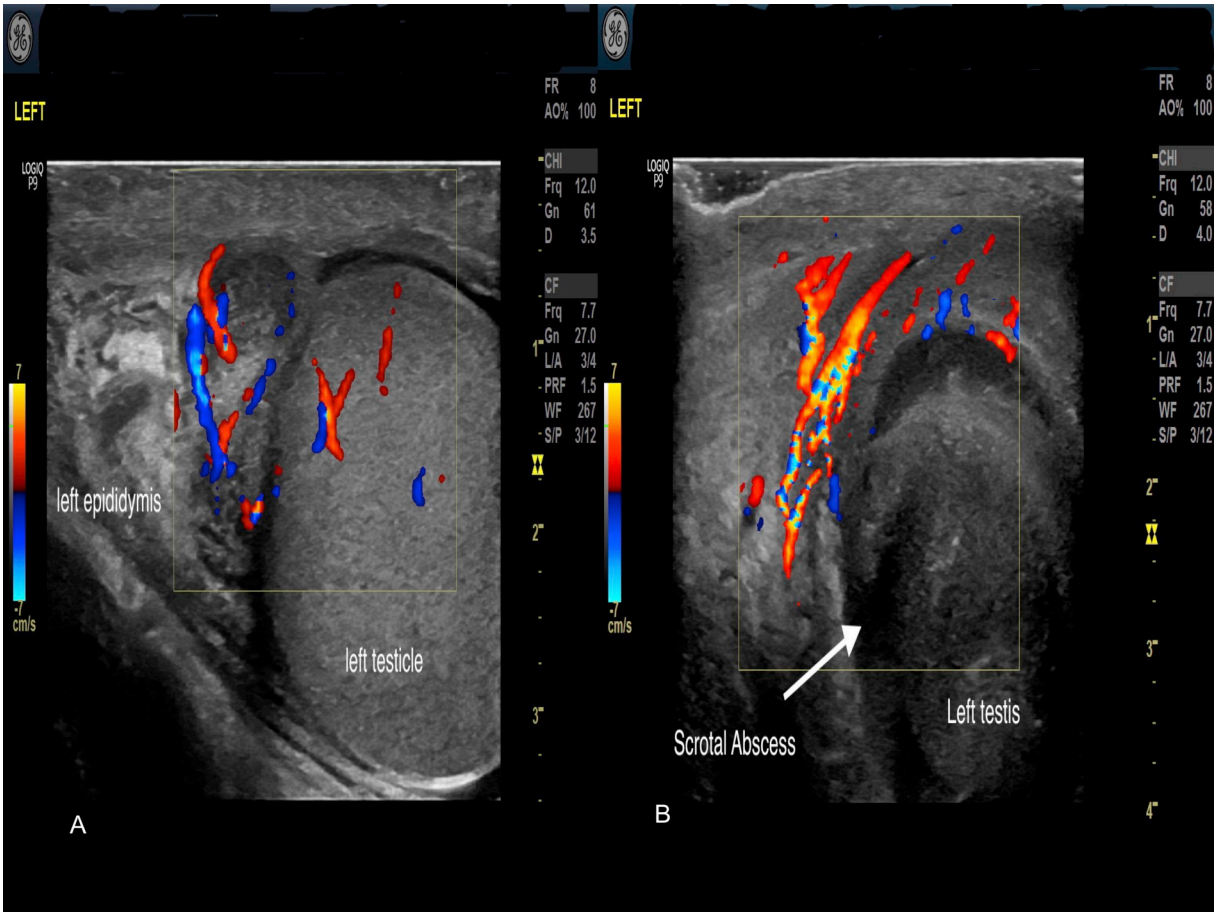

Fig. 1. a. Initial doppler ultrasound showing enlarged left epididymis with heterogeneous echopattern and increased vascularity suggesting epididymitis and intact testicular blood flow. b. Follow-up ultrasound showing absent blood flow to the left testicle as well as development of scrotal abscess.

post-operatively. He was discharged home in post-operative day four with oral ciprofloxacin 500mg PO twice a day for two weeks based on the culture and sensitivity from the pus sent intra-operatively that grew *Escherichia coli*. Histopathology revealed extensive hemorrhagic infarction, heavy infiltration by acute and chronic inflammatory cells, excessive tissue edema, and congested blood vessels. There was no granuloma or tumor found (Fig. 3).

Discussion

In young sexually active men, epididymitis is commonly caused by Chlamydia and Neisseria gonorrhea. Whereas in men with voiding dysfunction, infection process of the genital organ is triggered by *Escherichia coli* and other gram-negative bacteria. Epididymitis could result from noninfectious process secondary to medications, reflux of sterile urine through ejaculatory ducts, or vasculitic disease. Epididymo-orcitis occurs when the infection extends from epididymis and affects the testis.

Segmental and global testicular ischemia is a rare but devastating sequela of epididymo-orchitis that needs urgent medical attention and proper management. Testicular ischemia occurs when blood flow to the testis is impaired. Different theoretical aspects have been proposed in the literature. Once the acute inflammatory processes commence within the testis, recruitment of inflammatory cells with production of abundant exudates and tissue edema occur that might compress the blood vessels within the epididymis and testis and result in compartment effect within the testis. Furthermore, extra luminal compression of the testicular microvasculature and the increased in venous pressure can result in compartment syndrome as well. Vascular thrombosis causing

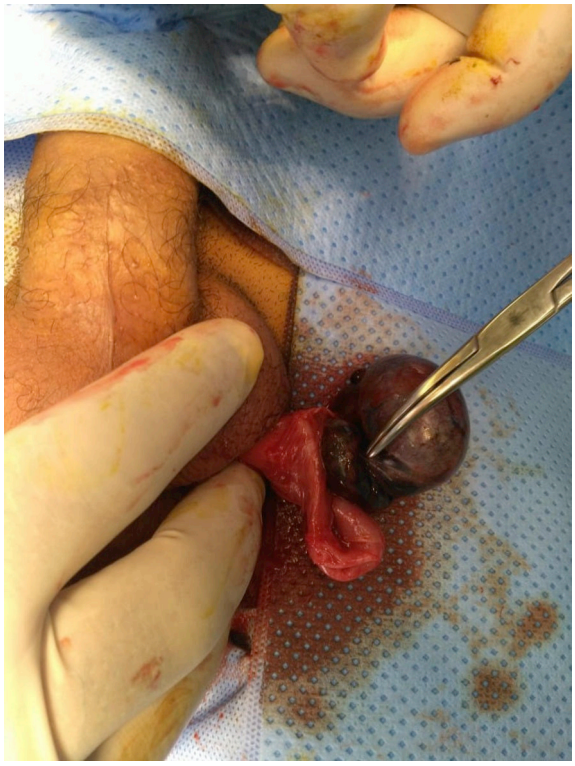

Fig. 2. Intra-operative photograph of scrotal exploration following drainage of the abscess and appearance of black ischemic left testicle.

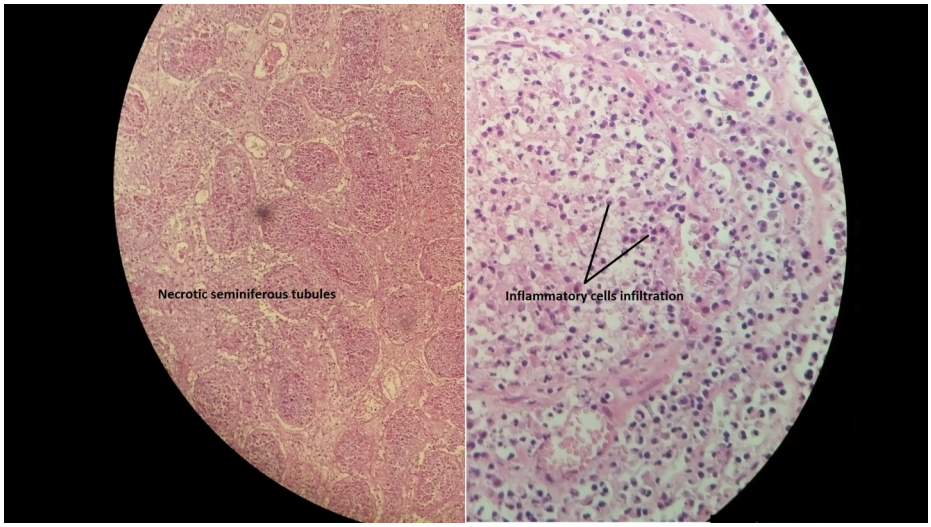

Fig. 3. Histopathology slide with Hematoxylin and Eosin stained sections showing extensive seminiferous tubules necrosis and heavy infiltration by inflammatory cells.

endothelial dysfunction and damage has also been reported to increase the pressure within the testis and further interruption of the circulation. Testicular torsion, inguinal hernia causing compression on spermatic cord, varicocele hypertension and intra-testicular arteriovenous fistula have been described as etiological factors that can lead to increase in venous resistance and may contribute to development of the compartment syndrome.[2]

Theoretically if the compartment syndrome is the main cause of testicular ischemia and the diagnosis picked up early before irreversible damage to the testicles occur, then incision of the tunica albuginea might decrease the compartment effect within the testis. Following capsulotomy of the tunica albuginea, grafting with tunica vaginalis to cover the defect has been reported to normalize the compartment pressure.[3] However, this intervention was invented by Figueroa and his group in patients whose presented with testicular torsion and managed surgically with de-torsion and capsulotomy to improve testicular blood flow. But the application of this surgical approach was still not tested in case of infectious process.

Various forms of vasculitis affect multiple organs and present with systemic manifestations, but occasionally involve single organ. Epididymal and testicular involvement can be a part of systemic disease or rarely reported as isolated organ affected by vasculitis.[4] Although clinical presentations can not be distinguished from other causes of acute scrotum, there has been no specific features to diagnose vasculitis of testis based on scrotal ultrasound. Variable pictures were reported in literature ranging from normal or heterogeneous testicles with diverse blood flow on color doppler to intra-testicular mass like lesions and testicular infarction. The exact pathophysiology of vasculitis induced testicular ischemia is not fully understood. Thrombosis causing obstruction of the blood vessels and thus impairment of the blood flow to the testicle will result in testicular pain, segmental or global testicular ischemia mimicking testicular torsion.[5]

Conclusion

Testicular ischemia is a rare consequence in patients with epididmo-orchitis. In the setting of no clinical improvement after initiation of antibiotic, close observation with broad spectrum antibiotic coverage and serial sonographic examination might be necessary to realize such complication. Although anecdotal reports have been issued, the mechanisms of testicular ischemia were not identified; and adjunctive medical therapy could be identified in the future to avoid such complications.

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Author contribution statement

Design, Interpretation, Data collection and writing: Dr. Badr Alharbi, MD.

Writing and literature review: Dr. Emad Rajih.

Data collection: Dr. Adewunmi Adeoye, Bashar Abed Allatiefe, Mohammad Hasan Abdullah.

Conflict of interest statement

Authors declare no conflict of interest.

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