EPIDIDYMO-ORCHITIS LEADING TO GLOBAL TESTICULAR INFARCTION IN A PEDIATRIC PATIENT – A CASE REPORT

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Introduction:

The differential diagnosis for the acute scrotum is broad. The approach often involves ruling out emergent causes of acute scrotal pain such as testicular torsion, which if left untreated, will result in testicular infarction. Epididymo-orchitis (EO) is the most common cause of scrotal pain in an outpatient setting, accounting for 600,000 cases annually in the US.[1] EO usually presents indolently with unilateral scrotal pain, rarely requires surgical intervention, and is typically managed successfully with antibiotics, analgesia, and scrotal support.[2] Complications of EO are uncommon but can include worsening infection or abscess formation, which can be detected with ultrasonography if symptoms fail to resolve. Global testicular infarction as a result of EO, however, is exceedingly rare and has been reported to occur in only 1-2% of cases.[3]

We present a rare case of global testicular infarction secondary to EO in a 17-year-old patient.

Case Report:

A 17-year-old male with a past medical history of congenital deafness, distal hypospadias repair, and recurrent urinary tract infections presented with left scrotal pain. The patient's pain began two days prior after he accidentally struck his groin on a bench and had progressively worsened since. The pain radiated to the abdomen and was associated with mild nausea, dizziness, and subjective fever. The patient initially presented to an outside emergency department where a scrotal ultrasound revealed hyperemia of the left epididymis and spermatic cord and was negative for testicular torsion (Figure 1). Urinalysis was suggestive of infection; complete

blood count was significant for 28,000 white blood cells. He was prescribed doxycycline and discharged from the outside emergency department. Urine culture ultimately grew 20,000 CFU of mixed organisms. Cultures for gonorrhea and chlamydia were negative.

Despite antibiotic treatment, he continued to have scrotal pain and swelling which ultimately led to his presentation at our center two days later. Upon arrival, the patient was febrile and had a white blood cell count of 30,000. Physical exam revealed an edematous, erythematous, and tender left hemiscrotum, and the left testicle could not be distinctly palpated. An urgent scrotal ultrasound showed the left testicle to be three times larger than the right testicle. Left testicular blood flow was absent and the spermatic cord appeared abnormal (Figure 2). A loculated left hydrocele was also noted. The right testicle appeared normal. The patient was taken immediately to the operating room for scrotal exploration. Intraoperatively, there was significant inflammation and thickening of the scrotal wall and dartos. When the left testicle was delivered, it was noted to be gray in color and there was no twist of the spermatic cord. The left tunica vaginalis was incised and there was no bleeding and tissue was clearly necrotic. Left orchiectomy and right orchiopexy were performed. Tissue cultures were obtained from the left testicle and epididymis.

Pathologic examination of the specimen revealed a tan-white and congested tunica albuginea surface and testicular parenchyma with diffuse hemorrhagic and edematous tubules (Figure 3). Sectioning of the spermatic cord revealed a hemorrhagic, lobulated, fatty cut surface interspersed with multiple dilated vascular channels. No clear knot or twist was identified in the spermatic cord. Tissue culture

and smear were positive for *Escherichia coli*. A final diagnosis of infarction from epididymo-orchitis was made.

Discussion:

Global testicular infarction as a complication of EO is a rare event. The pathogenesis of testicular ischemia in the setting of EO is most likely due to severe inflammation involving the spermatic cord and extrinsic compression of testicular vessels by epididymal edema.[4] There have also been reports of venous thrombosis of the epididymis and cord as a result of endothelial damage from bacterial toxins.[5]

Differentiating testicular hypoperfusion secondary to EO from that due to testicular torsion can be difficult, especially if the diagnosis is unclear based on the history and physical exam. However, distinguishing between etiologies is worthwhile as the management options for testicular salvage are different. Some clinicians suggest the measurement of C-reactive protein, which is more likely to be elevated in cases of EO.[3] Further, ultrasound imaging typically reveals different pathologic signs in EO and torsion. In testicular compromise secondary to EO, a juxta-epididymal "string-of-beads" sign can be seen[6], whereas torsion of the spermatic cord gives rise to the classic "whirlpool" sign. On our patient's ultrasound, there were subtle findings of the string-of-beads sign (Figure 2).

Identifying which patients with EO will progress to testicular infarction presents an additional challenge, as strong evidence to support clinical features that predict this outcome is lacking. Potential warning signs of ischemia include failure of EO to resolve with appropriate antibiotics, worsening pain despite initial improvement, spermatic cord tenderness and thickening, and recurrent EO.[7] It

has previously been established that complications of EO occur more frequently in cases of infection with urinary tract pathogens, as opposed to infection with sexually transmitted organisms.[4] This association with urinary/coliform pathogens appears to be consistent for cases of testicular infarction in EO as well, and many cases reported in the literature had urine cultures positive for E. coli.[2,3,7-10] History of trauma has not been previously reported to be associated with this complication, and it is unclear whether the scrotal trauma that occurred in our patient's case expedited the infectious or ischemic process, or was simply coincidental.

Ultrasonography remains the most useful imaging modality for diagnosing testicular ischemia. In EO, Doppler ultrasound usually shows hyperemia or vascular congestion consistent with an inflammatory process. In contrast, absent Doppler flow combined with areas of hypoechoic tissue within the testicle is highly suggestive of infarction in the setting of EO.[9] If standard ultrasound is inconclusive, microbubble contrast-enhanced ultrasonography can more definitively determine the presence of testicular hypoperfusion.[10] If able to be performed rapidly, scrotal MRI is another option that can more obviously demonstrate lack of perfusion with a specificity approaching 100%.[11]

Once testicular infarction is suspected, management usually involves urgent scrotal exploration and orchiectomy. Some authors have suggested potential testicle-sparing options that may be theoretically pursued in cases of impending infarction. These options include medical management with anticoagulation, antiplatelets or thrombolytic drugs. If there is hypoperfusion to the testicle on ultrasound, surgical exploration with longitudinal incision in the epididymal and external spermatic cord fascia has been shown to restore blood

flow to the testicle in cases of severe EO [8] These options may be of particular use for patients with a solitary testis or sub-optimal fertility**.**

Conclusion:

We present a case of global testicular infarction as a result of EO, a rare complication for which there is little clinical guidance due to its infrequency. Differentiating between testicular ischemia secondary to torsion versus complicated EO can be challenging. Early recognition of impending infarction in EO requires a high degree of clinical suspicion and may provide an opportunity for testicle-sparing interventions. Contrast-enhanced ultrasonography is a useful modality for identification of testicular hypoperfusion, especially if Doppler ultrasonography is equivocal.

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Figures:

Figure 1:

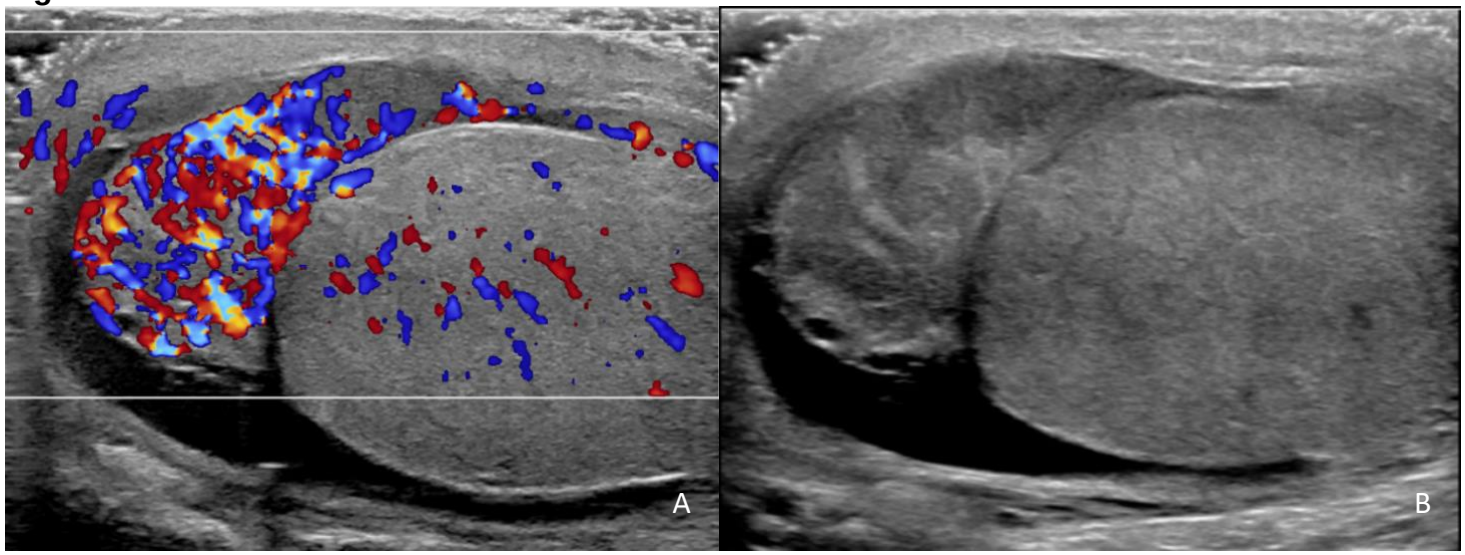

Fig. 1. (A) Doppler ultrasound from initial presentation to outside center showing increased cord and epididymal blood flow. Normal blood flow seen within testicle. (B) Grey scale ultrasound of the testicle showing enlarged epididymis and heterogeneity of the epididymis and testicle.

Figure 2.

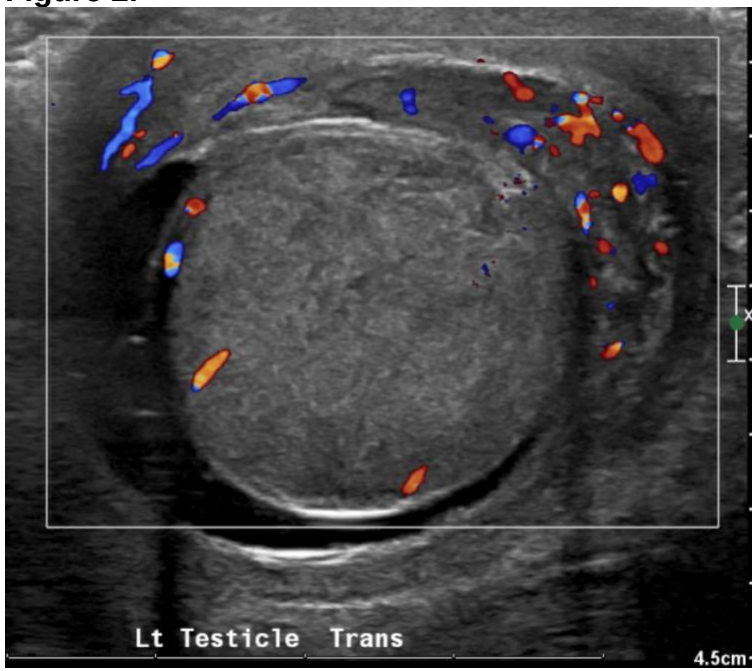

Fig. 2. Color Doppler ultrasound from our center, two days after initial presentation, showing a non-homogeneous, avascular left testicle and possible twisted cord with hypervascularity. Subtle findings of the juxta-epididymal “string-of-beads” sign are present.

Figure 3.

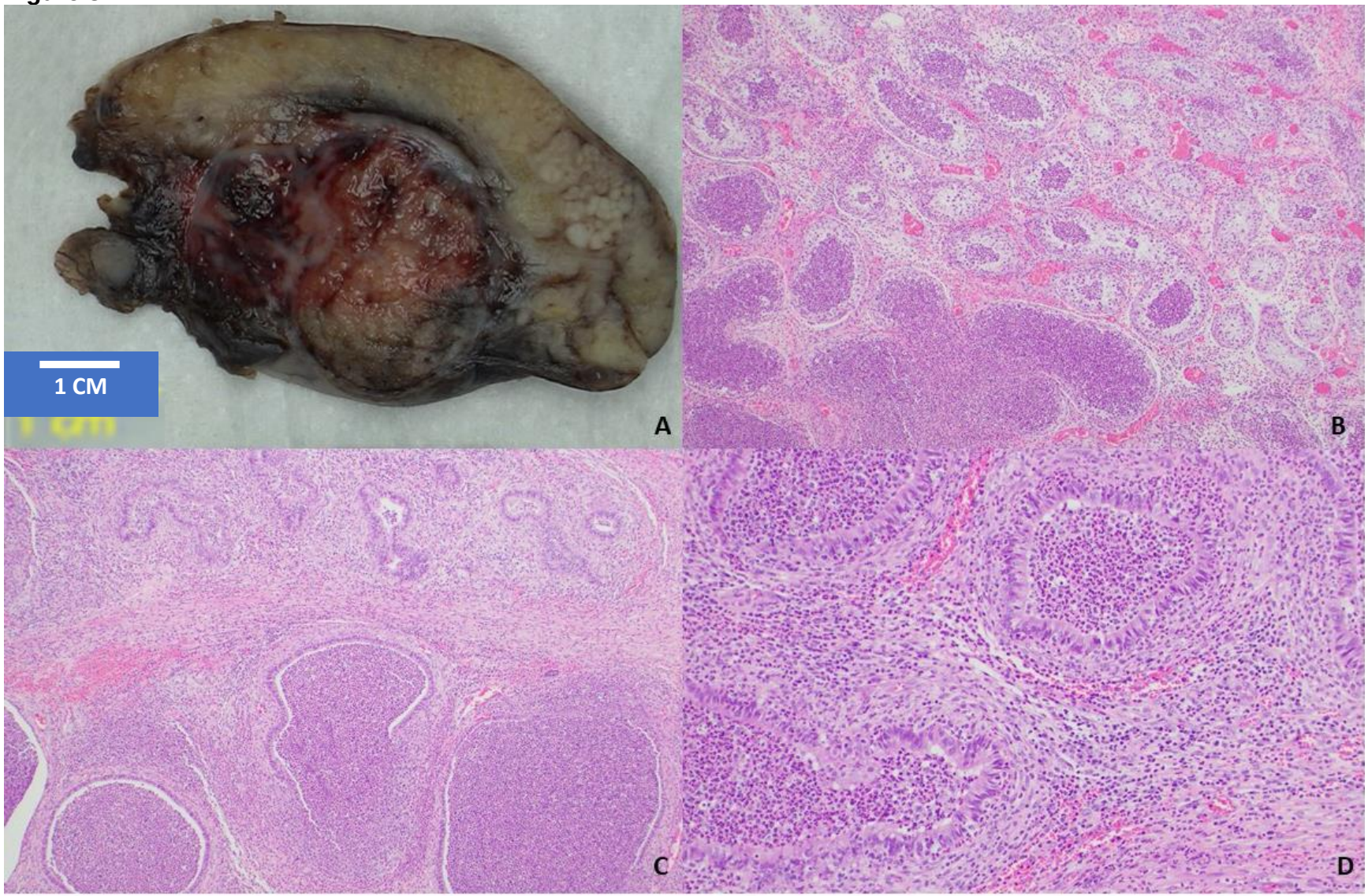

Fig. 3. Pathologic examination of the specimen revealed grossly hemorrhagic and necrotic testicular parenchyma and dull-tan cut surface of the testicular adnexa (A). Microscopic examination revealed marked acute suppurative

inflammation in the testicular parenchyma with abscess formation and necrosis in and around seminiferous tubules (B, 40X; C, 100X). Marked acute and chronic inflammation with mild to moderate fibrosis were identified in the epididymis and paratesticular soft tissue (D; 200X).