Testicular ischemia following mesh hernia repair and acute prostatitis

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ABSTRACT

We present a case of a man admitted to our Hospital for right acute scrotum that six months before had undergone a right hernioplasty with mesh implantation. Clinical history and testicular color Doppler sonography (CDS) patterns suggested an orchiepididymitis following acute prostatitis. After 48h the clinical picture worsened and testicular CDS showed a decreased telediastolic velocity that suggested testicular ischemia. The patient underwent surgical exploration: spermatic cord appeared stretched by an inflammatory tissue in absence of torsion and releasing of spermatic cord was performed.

In patients with genitourinary infection who previously underwent inguinal mesh implantation, testicular CDS follow-up is mandatory.

Key words: Acute scrotum, inguinal hernia repair, prostatitis, testicular ischemia

INTRODUCTION

The acute scrotum constitutes the most common urological emergency and color Doppler sonography (CDS), along with the physical exam, represents the imaging modality more frequently employed in the clinical assessment of acute scrotum.[1]

The two most important entities that must be ruled out in every case of acute scrotal pain are torsion of spermatic cord and orchiepididymitis, while other causes occur more rarely. In case of inguinal hernia repair using mesh techniques the spermatic cord is potentially affected by chronic inflammatory tissue remodeling that may impair testicular perfusion inducing acute scrotum.[2]

A rare case of testicular ischemia following mesh hernia repair and acute prostatitis that presented as acute scrotum is reported herein.

CASE REPORT

A 23-year-old man was admitted for right orchialgia arising 24h before admission; although he referred urinary symptoms (stranguria, pollakiuria and gross hematuria) and fever of five days' duration, no therapy was previously administered. Six months before, the patient underwent a mesh implantation to treat a recurrent right inguinal hernia. At admission, history, genitourinary exam, blood and urine test and transrectal CDS suggested an acute prostatitis; testicular CDS revealed normal signal in correspondence of the testis and epididymis. The patient was hospitalized and submitted to antibiotic therapy. After 48h the clinical picture worsened: testicular pain and fever increased, CDS showed a decreased TDV (telediastolic velocity) on intratesticular artery [Figure 1] and a high SPV (systolic peak velocity) in correspondence of the spermatic cord near the external abdominal ring [Figure 2a]. This sonographic pattern highly suggested a testicular ischemia[3] and the patient underwent surgical exploration: spermatic

Figure 1: Absence of TDV on intratesticular artery
cords appeared congested, increased in size, compressed and
stretched by an inflammatory and scar tissue in the absence
of any spermatic cord torsion. Resection of tunica vaginalis
and releasing of spermatic cord was performed. After
surgery the patient became asymptomatic, CDS returned
to a normal pattern [Figure 2b] and antibiotic therapy was
administered for two weeks. Histological examination of the
tissue surrounding the spermatic cord showed a no specific
inflammatory pattern.

**DISCUSSION**

Spermatic cord torsion, orchiepididymitis and trauma
constitute the main causes of acute scrotum, although the
differential diagnosis refers essentially to the torsion of
spermatic cord vs. inflammatory lesions. A careful physical
exam and anamnesis combined with CDS parameters often
address toward the diagnosis of testicular ischemia that, more
rarely, can be secondary to severe epididymitis, inguinal
hernia repair,[4] spontaneous trombosis of funicular vessels,[5]
xanthogranulomatous funiculitis[6] or filarial.[7] Despite the
high diagnostic accuracy of CDS, all authors agree that in the
presence of clinical suspicion of testicular torsion, surgical
exploration is mandatory;[3] false-negative results with CDS
are attributed to an incomplete torsion of the spermatic cord
when the systolic value is still recorded while the diastolic
one is absent or reduced and to the reactive hyperaemia of
the tunica vaginalis which is wrongly interpreted as blood
flow into the capsular arteries.

The use of mesh during hernia repair is associated with
a relative reduction in the risk of hernia recurrence of
around 30-50%; however, there is no apparent difference in
recurrence between laparoscopic and open mesh methods
of hernia repair.[8] Experimental[9] and clinical studies[10]
demonstrated that mesh implantation may induce foreign-
body response that led to the encasement of spermatic cord
by scar tissue. In most cases, testicular perfusion is not
compromised, but a concomitant inflammatory process (i.e.,
funiculitis secondary to prostatitis) may worsen the blood
supply leading to acute scrotum. Peiper et al. performed an
experimental study in 15 adult male pigs that underwent
transinguinal preperitoneal implantation of polypropylene
mesh and shouldice repair on the contralateral side. The
authors reported that mesh repair led to a decrease of
arterial perfusion, testicular temperature and seminiferous
tubules.[2] Dilek et al.[11] reported no statistically significant
differences between the preoperative and postoperative
CDS parameters (VPS, TDV and resistive index), while
Ersin et al.[12] showed that testicular blood flow is influenced
during laparoscopic inguinal hernia surgery. Mincheff et
al.[13] reported a rare case of upper pole testicular infarction
after laparoscopic total extraperitoneal repair of indirect
inguinal hernia.

In our case report testicular ischemia was secondary to
spermatic cord compression by an inflammatory and scar
tissue in absence of torsion and only after releasing of
spermatic cord the patient became asymptomatic.

In conclusion, in patients with genitourinary infection
who previously had an inguinal mesh implantation, clinical
observation and CDS follow-up is mandatory to treat in time
this unusual complication.[14]

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