



Case Report

Testicular Infarction in the Presence of Epididymitis: An Anatomico-Clinical Continuum?

Calcagno C*, Introini C and Calcagno S

Abstract

Epididymitis is an inflammation limited to the epididymis, common cause of acute scrotal pain and usually responsive to antibiotics, non-steroidal and anti-inflammatory drugs. Nevertheless complications such as orchitis and abscess may occur; a testicular infarction is less frequent. We are going to discuss two cases of segmental testicular infarction both associated with epididymitis. The first one developed in a massive hemorrhagic infarction after performing a not responsive homolateral epididymitis to conservative therapy and subsequent orchiectomy was performed. The second one was treated with successful medical therapy and complete restoration of testis. We reviewed the pertinent literature in terms of differential diagnosis, etiology with particular interest in physiopathological mechanisms of testicular infarction giving an original etiologic hypothesis.

Keywords

Massive and segmental testicular infarction; Epididymitis; Segmental testicular; Angio-architecture

Introduction

Epididymitis is the commonest cause of an acute scrotum in post-pubertal men [1]. Although responsive to medical treatment with antibiotics and non-steroidal anti-inflammatory drugs, epididymitis may have in some cases a prolonged course and run with complications such as orchitis, abscess, late testicular atrophy and testicular infarction both segmental and massive [2,3].

We discuss two cases of segmental testicular infarction both associated with epididymitis. The first one developed in a massive hemorrhagic infarction after unsuccessful medical therapy and subsequent orchiectomy. The second one was treated with conservative therapy and completed healing of the testis. We reviewed the literature with particular regard to predisposing factors and mechanisms of testicular infarction associated with epididymitis proposing an original hypothesis about clinical relationship between segmental and massive testicular infarction.

Case Reports

Case 1

A 40-year-old white man in healthy conditions arrived at the

*Corresponding author: Calcagno C, MD, Division of Urology, Evangelic International Hospital, Genova, Italy, E-mail: ste.calcagno@alice.it

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Emergency Room (ER) complaining about a 2-day persistent and worsening left scrotal pain. He had a past history of hepatitis C but no other systemic disease. The patient stated that he had not unprotected sexual intercourse the previous six months prior. Three months before he was treated at ER of another hospital with no specified antibiotic for a prostatitis and contextual left epididymitis with partial resolution but not complete disappearance of scrotal pain in the following weeks. The patient denied recent scrotal trauma, urethral discharge, micturitional disorders and fever. On presentation he appeared in healthy conditions and afebrile. After clinical examination all systems were normal, with the exception of the genitourinary tract. Rectal examination revealed an enlarged and mildly painful prostate; the left side of the scrotum was swelling with erythema of the overlying skin and severe tenderness to palpation. Left testis was moderately enlarged, low-lying and vertically placed; left epididymis was enlarged in all its length and extremely painful; there was no sign of torsion of spermatic cord. Blood cell count, urine culture and serum biochemistry were normal. Tumor markers (alpha-fetoprotein and human chorionic gonadotropin) were negative. An ultrasound examination revealed a hypoechoic wedge-shaped mass with base in the very external part of the medial zone of the left testis; the rest of the left testis and the right one had a normal echogenic appearance. Color Doppler sonography detected an increased blood flow in the left epididymis and a reduced blood flow in the hypoechoic wedge-shaped zone. These findings were believed to be consistent with left acute epididymitis and segmental testicular infarction.

The patient was prescribed to take antibiotics (quinolone and nitroimidazole) and non-steroidal anti-inflammatory drugs and was instructed to elevate the scrotum and use local cool compresses. After two days of apparent clinical and subjective improvement, the patient referred an unbearable pain in the left scrotum. An ultrasound color Doppler examination revealed a complete infarction of the left testis with absent blood flow in the gland. The patient was subjected to a left scrotal exploration which revealed complete necrotic testis and epididymis; so orchiectomy was tested. The microscopic examination of the specimen showed a complete hemorrhagic infarction of the testis with areas of coagulative necrosis and infarction with diffuse necrosis and important inflammatory reaction in the epididymis. The epididymis showed a very extensive infiltrate of plasma cells, lymphocytes and histiocytes with rare and perivascular neutrophils and a wide ranging of venous occlusions. No normal tubules or spermatozoa were identified in any specimen. The postoperative period was uneventful.

Case 2

A 57-year-old white man presented to our hospital complaining of a five-day persistent left scrotal pain. After close clinical tests, the left testicle appeared smaller, low-lying and vertically placed; the left epididymis was enlarged and extremely painful; no sign of torsion of spermatic cord; rectal examination revealed a mildly enlarged and tender prostate. In his past there was a history of left undescended testis resolved spontaneously at the age of five. Blood cell count, urine culture and serum biochemistry were normal. Tumor markers (alpha-fetoprotein and human chorionic gonadotropin) were negative. Ultrasound color Doppler examination showed a hypoechoic wedge-

shaped zone in the equatorial part of left testis with absence color Doppler signal and a diffuse unhomogeneity of left epididymis. The MRI examination confirmed a wedge-shaped in the left testis, marked perilesional edge and low contrast enhancement after injection of contrast medium (Figure 1).

The clinical and instrumental results were consistent with acute prostatitis, contextual left epididymitis and left segmental testicular infarction. The patient was on netilmicin 300 1gr/die, non-steroidal anti-inflammatory drugs and scrotal suspensory support. Clinical and ultrasound examination in a strict follow-up was assessed and at three months later there was a complete restoration of the testis and a complete regression of epididymitis and prostatitis.

Discussion

Coping with any painful testicular mass the clinical priority is differential diagnosis. This latter includes spermatic cord torsion, acute orchiepididymitis, tumor and testicular infarction. Spermatic cord torsion is essentially a clinical diagnosis based on anamnesis of acute onset of scrotal pain, clinical examination, and color Doppler ultrasound findings. Prompt identification of spermatic cord torsion is of course very important for the patient but it is also important from a theoretical point of view because if we can't recognize a funicular torsion, we will not never know if in case of late testicular atrophy we cope with a overlooked torsion or a testicular atrophy subsequent massive testicular infarction associated with epididymitis [1]. The same concerns, of course with different findings, acute orchiepididymitis: gradual onset, possible presence of fever, associated micturitional symptoms coming from an infective prostatic origin, clinical objective findings. Nevertheless, in case of doubtful diagnosis, surgical scrotal exploration is mandatory. Testicular tumors rarely present with pain, nonetheless it is correct from a clinical point of view to consider neoplastic every testicular swelling until there is clear evidence to the contrary [4]. Any hypoechoic testicular mass must be considered neoplastic until proved otherwise. The negativity of neoplastic markers, the color Doppler ultrasound and MRI findings are now useful in detecting segmental testicular infarction and it can usually avoid surgical exploration [5]. Otherwise, when diagnosis is not certain, surgery with possible frozen section and sparing surgery is mandatory; in absence of sure histological response the choice must be orchietomy. It is compulsory to rule out every suspect of testicular neoplasm. Negativity of neoplastic tumor markers and pattern of

hypo vascular lesion at ultrasound color-Doppler examination consent in fact a wait and see policy and a conservative management without forgetting that also in presence of a hypo-a vascular pattern 86% of testicular neoplasms of less than 1,6 cm can show a similar pattern [6]. Finally, global testicular infarction is not infrequent and it usually results from torsion of the spermatic cord, severe scrotal trauma, incarcerated inguinal hernia, subsequent to an inguinal hernia repair and it usually represents a post- surgical diagnosis. On the contrary, segmental infarction of the testis is much more rare (less than 100 cases in literature), and it is often diagnosed only in a post-surgical stage [7] although there's been an impressive improvement in pre-surgical diagnosis.

In fact ultrasound examination, at dawn in 60's, has revolutionized clinical approach to scrotal pathology and some cases of segmental testicular infarction have been discovered in consistent series [8] besides single case reports. . In particular, contrast-enhanced ultrasound can increase confidence in the diagnosis of segmental testicular lesion compared with reliance on gray-scale and color-Doppler findings. Presence of perilesional rim enhancement is evident within 2-17 days after the onset of symptoms [9]. Concerning the specific relationship between segmental testicular infarction and epididymitis, it was rarely assessed by ultrasound examination in the past. For instance, in 1966 Mitemeyer et al. [10] reported no cases of testicular infarction in a large series of acute epididymitis and Bilagi et al., [11] on the contrary; in 2007 in a six year experience (1999-2005) reported 14 cases of acute epididymitis out of 24 cases of segmental testicular infarction. Vasculitis needs a special reference. With this term we refer to anatomic alterations characterized by necrosis and inflammation of the vascular wall. The clinical incidence of testicular alterations in Panarterite Nodosa is at 2-18% [12], at 10-15 % in Henoch-Schonlein Purpura and at 9% in Wegener's Granulomatosis [13]. Gonadal vasculitis affects testis, epididymis, spermatic cord and scrotal skin but only rarely alteration of testis and epididymis represent the first symptom of the disease. In the presence of histological evidence of testicular necrotizing arteritis, exudation of polymorphonuclear leukocytes and extra vascular granulomas all clinical, hematologic and biochemical examination should be performed to rule out systemic disease.

Segmental testicular infarction is usually considered to be an idiopathic disorder commonly presenting painful scrotal swelling. The predisposing factors to segmental infarction include sickle cell anemia [14], hypersensitivity angiitis [15], trauma [16], after orchiectomy and varicocelectomy [17], acute epididymitis [18]. Focusing on this last condition, subject of this report, a very important clinical issue is to recognize clinical situations in which acute epididymitis turns in a chronic one. About this matter it is worthwhile to remember the definition of chronic epididymitis: "a 3-month or longer history of symptoms of discomfort/pain in the scrotum/testicle or epididymis that are localized to one or both epididymis on clinical examination" [19]. Chronic epididymitis must be suspected in patients who are not responsive to an appropriate antibiotic treatment, testicular pain after improvement with medical therapy, patients with recurrent epididymitis who have acute epididymitis, persistent tenderness and palpable thickening of the spermatic cord during treatment of epididymitis [20]. At this point the fundamental clinical question is: why and when can epididymitis cause complete or segmental testicular infarction and which are the fundamental physiopathological mechanisms leading to segmental or massive testicular infarction? Jordan [21] has supposed, beyond every



Figure 1: Ultrasound color MNR examination.

predisposing factor to testicular ischemia, the possibility to have segmental watershed areas in some males as possible explanation for this rare finding. The theory of reduction in blood flow, due to venous thrombosis, to certain areas of testicular tissue which function as end organ associated with segmental watershed areas supposed by Jordan seems to explain the pathogenesis of segmental testicular infarction showing the morphology of focal infarcts comparable in other organs like kidney or spleen. The presence of segmental angioarchitecture of the testicular lobule in man has been confirmed by Ergun et al [22]. These Authors, using serial sections from paraffine and Epon embedded testicular tissue and computer-aided 3-D reconstruction have demonstrated the relationship between the angioarchitecture of the human testis and its inner subdivision into testicular lobes. Connection between testicular artery and pampiniform plexus through a thick capillary vessel net [23] may lie at the bottom of testicular infarction both hemorrhagic and ischemic. About the mechanisms leading to testicular ischemia associated with epididymitis three main factors have been postulated:

(a) Edema of the epididymis compressing adjacent testicular veins and edema of globus major compressing testicular head. (b) Funiculitis which may cause edema resulting in irreversible lymphatic, venous and arterial obstruction of testicular vasculature [24]. (c) Venous thrombosis provoked by bacterial toxins [25]. (d) Vascular compression by edema at external inguinal ring [26]. Venous thrombosis seems to be very important in the pathogenesis of testicular infarction. In a study of eighteen cases of infected infarcts of the testis Hourihane showed that totally occluded veins in the epididymis and spermatic cord were present in 17 cases. These three predisposing factors, probably working together in case of acute epididymitis, seem to be at the core of reduction of testicular flow and subsequent infarction.

Conclusion

The severity of infarction seems to be linked with a degree of testicular arterial constriction within the spermatic cord. The finding of reversal of diastolic blood flow on Doppler sonography in epididymitis as sign of impending infarction appears to confirm the involvement of testicular artery. This is the reason of the relative sparing of epididymis from infarction due to a different arterial supply by the cremasteric and deferential artery. Unresponsive treatment of epididymitis can lead to a prolonged reduction of testicular vasculature and subsequent testicular infarction. Delay in diagnosing infarction, incomplete treatment of epididymitis, duration and severity of epididymitis lead to a prolonged reduction of testicular vasculature provoking from segmental to a complete testicular infarction with arterial involvement in this last case.

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Author Affiliation

Division of Urology, Evangelic International Hospital, Genova, Italy

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