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The challenging diagnosis of acute scrotum: remaining difficulties and further insights

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Abstract: The main target during management of a male pediatric patient with clinical signs of acute scrotum is the timely diagnosis, in order not to jeopardize the viability of the affected testicle. Thorough evaluation of the patient’s medical history, symptomatology, clinical and ultrasonographic findings, constitutes the basis of the diagnostic procedure. After comprehensive research of the relevant literature, we highlight the remaining difficulties in the evaluation of the clinical and ultrasonographic findings for the accurate diagnosis of the acute scrotum. In conclusion, it is worth emphasizing on the following: a. the most common diseases that come under the diagnosis of the acute scrotum may present with similar symptoms, b. in neglected cases the diagnostic approach becomes more difficult, constituting the evaluation of the pathognomonic clinical signs challenging, and c. inability to exclude the diagnosis of spermatic cord torsion should be an indication for the surgical exploration of the affected hemiscrotum.

Keywords: acute scrotum, spermatic cord torsion, acute epididymitis, appendiceal torsion.

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Background

The term acute scrotum (AS) encompasses clinical entities characterized by pain, redness and scrotal swelling. In a previous retrospective analysis, Yang *et al.* treated 1,215 male children with AS in a total of 12,804 admissions (9.48% of total admissions), demonstrating that 103 out of the 1,215 (8.5%) children suffered from sper-



matic cord torsion (SCT), whereas 8 out of 103 cases (7.8%) occurred during neonatal period [1]. In the recent years, the incidence of acute epididymitis (AE) has been reported to range from 10–71% of all cases of AS [2]. In a study of 65 cases of AS, it was shown that 42 out of 65 patients (64.6%) suffered from AE [3]. According to another report, 0.8% of children admitted to emergency departments, suffer from AE [4].

Causes

The commonest causes of acute scrotum in childhood are summarized in Table 1.

Table 1. Classification of the most common causes of acute scrotum during childhood.

| | Etiology | Diseases spectrum |
|---|-------------------------------|--|
| 1 | Ischemia | Spermatic cord torsion, appendiceal torsion |
| 2 | Infection | Acute epididymitis, pyocele, extension of an intra-abdominal infection, Fournier gangrene |
| 3 | Aseptic inflammation | Vasculitis (Henoch-Schonlein purpura) |
| 4 | Trauma | Blunt or puncture trauma of the scrotum, rupture of tunica albuginea, testicular contusion or intratesticular hematoma, scrotal hematoma |
| 5 | Processus vaginalis pathology | Incarcerated inguinal hernia, acute or tension hydrocele |
| 6 | Unclassified | Varicocele, primary or secondary scrotal neoplasia |

AS represents a field of frequent medical errors, most commonly associated with the underestimation of AE [5], while SCT usually manifests in an atypical manner, almost in one third of all cases [6].

Table 2 summarizes the essential clinical features of all entities responsible for the development of AS except for SCT, appendiceal torsion (AT) and AE. The last three diseases will be discussed in further detail within the next sections of the present article.

Male children with acute idiopathic scrotal edema are usually asymptomatic, however they may report unspecified discomfort in the scrotum, while itching may precede scrotal edema.

Scrotal edema is usually the first manifestation, develops suddenly, is accompanied by diffuse redness of the area and spreads rapidly within a few hours. Often, the edema starts from one hemiscrotum (left to right ratio = 1) and progressively spreads throughout the scrotum. Redness extends to the groin (67% of cases), to the perineum (42%), to the penis (20%), or to the suprapubic area. Edema and erythema are major manifestations of the disease in all patients, with local pain present in 80% of cases. The duration of the disturbances is 6–72 hours (average 14 hours). Regression of the edema and redness begins soon, while it usually ends within 48 hours, with no residual lesions. In 21% of cases the disease recurs up to three times without complications [7].

Table 2. An in-depth listing of the most common causes of acute scrotum, except for SCT, AT, and AE.

| Disease | Main Symptoms | Clinical signs |
|--|--|--|
| 1. Fournier gangrene | Sepsis with rapid deterioration, pain in the scrotum and perineum | Intense sensitivity and crepitation in the palpation of the affected area, swelling with skin pigmentation. Rapid deterioration with progression to septic shock |
| 2. Trauma | Report of the injury and its mechanism (crushing or puncturing trauma) | Obvious entrance gate or ecchymosis, swelling and sensitivity of affected testicle, hematocele, inability to determine testicular integrity |
| 3. Strangulated inguinal hernia | Pain, flatulence, vomiting, inability to excrete gases and feces | Swelling of groin that can extend to the ipsilateral scrotum, fixed to the posterior wall of the inguinal canal, which can be repositioned. Symptoms of intestinal obstruction. |
| 4. Hydrocele | Positive history of fluttering scrotum swelling, usually without discomfort. | It is usually possible to palpate the ipsilateral testicle. Transillumination of the scrotum plays a crucial role |
| 5. Tumor | Painless swelling of the scrotum gradually increasing. Bleeding or rupture: pain, sharp increase in swelling. In leukemic infiltration, symptoms of hematological disorders. | Palpation of a hard, compact and painless swelling usually. |
| 6. Varicocele | Mild and intermittent discomfort is found in the left — usually — hemiscrotum, exacerbated by increased intra-abdominal pressure. | Swelling — noticeable with inspection or palpation — of the pampiniform plexus either by Valsalva manipulation (stage II) or automatically (stage III). Signs of onset testicular “atrophy” ipsilateral. |
| 7. Henoch-Schonlein purpura | It affects the scrotum in 10% of cases. In boys aged less than 10 years (average 3–7 years): macular rash, colicky abdominal pain, arthralgia, nausea, vomiting. | Macular rash, arthritis, signs of digestive, urinary and central nervous system involvement. |

Clinical spectrum

SCT usually manifests with acute pain, which is essentially related to the prompt admission of the pediatric patient to the Emergency Department [8, 9]. However, both AT and AE can also manifest with acute pain.

Cass *et al.* found that 51% of patients with AE had sudden onset of pain [8]. An essential observation was that 16% of cases of SCT presented gradually [8]. In a study

by Waldert *et al.*, acute pain was reported in 96% of all cases of SCT (60/62 patients), 72% of all cases of AT (121/168 patients) and 50% of all cases of AE (12/24 patients) [10]. In their studies, Yang and Murphy reported that 5–6% of male patients with SCT underwent operative scrotal exploration without complaining of scrotal pain [1, 11]. The latter can be interpreted in the context of neglected cases, where necrosis of the twisted testicle had already occurred, leading to alleviation or complete remission of the scrotal pain.

The manifestation of SCT with abdominal pain is a common cause of a misdiagnosis: 5 to 12.5% of all cases of SCT manifest either with abdominal pain or abdominal pain preceding scrotal pain [8, 12]. It should be highlighted that the reflection of pain in the hypogastric region may accompany SCT in 7–28%, AT in 7–9% and AE in 7–21% of all cases [13, 14].

Nausea and vomiting are also accompanying symptoms of SCT [15, 16]. Jefferson *et al.* in their study including 83 boys with SCT, demonstrated a strong positive predictive value of nausea (96%) and vomiting (98%) as accompanying symptoms [17]. The latter finding is doubted by other authors, as shown in Table 3 [10, 14, 18].

Table 3. Relative frequency of nausea and vomiting in the main causes of AS.

| Author | % presence in SCT | % presence in AT | % presence in AE |
|----------------------------|-------------------|------------------|------------------|
| Lyronis <i>et al.</i> [18] | 62.8 | Not reported | 12.9 |
| Waldert <i>et al.</i> [10] | 32 | 3 | 12.5 |
| Mushtaq <i>et al.</i> [14] | 33 | Not reported | 14 |

Urinary symptoms are most commonly reported in patients with AE. However, the presence of urinary symptoms, along with documented pyuria and positive urine culture occur in 3.9–4.1% of all cases of AE [19, 20]. Cass *et al.* demonstrated that 7% of patients with SCT as well as 7% of patients with AE had concomitant urinary tract disorders [8].

Transient testicular torsion (TTT) occurs when the spermatic cord is automatically dislodged after twisting, with subsequent restoration of blood flow to the affected testis. TTT manifests with acute scrotal pain that automatically recedes within minutes. In 25% of cases it is accompanied by nausea and vomiting. A transient swelling of the affected hemiscrotum is frequently observed. TTT episodes may be repeated 1–30 times (average 4–5 times) prior to surgical exploration. In 30–61% of boys with established SCT, preceding episodes compatible with TTT are reported [21].

Physical examination

Cremasteric reflex is defined as an immediate contraction of the cremasteric muscle after a stimulus that pulls up the testis ipsilaterally, in the upper and inner third of the femoral region, femoral-inguinal fold or scrotum. It is a superficial cutaneous reflex produced by the T₁₂-L₂ myelotomy. Normally it is produced in 48% of newborns, 45% of infants and toddlers, and in 70% of older boys [22, 23]. Caldamon *et al.* documented the absence of cremasteric reflex in 100% of cases of SCT as well as in 33% of patients with AT and 25% of those with AE [24]. In contrast, Beni-Israel *et al.* found that 5 out of 17 patients with SCT (27%) had a normal cremasteric reflex [15]. Converging findings were highlighted by other authors [10, 11, 25–28].

In the contrary, Van Glabeke *et al.* demonstrated that 10 out of 25 patients with SCT (40% of the total) had a normal cremasteric reflex [26]. Srinivasan *et al.* found that the combination of an absence of ipsilateral cremasteric reflex, nausea, vomiting and a change in the appearance of the affected hemiscrotum features a strong diagnostic value for the documentation of SCT [29].

After a closer look at the aforementioned, one can deduce that the absence of the cremasteric reflex represents a pathognomonic finding of SCT. However, the presence of a normal cremasteric reflex in a child with AS may not be a strong indicator for the exclusion of SCT diagnosis.

Evaluating testicular axis change — from perpendicular to transverse — is not simple in a boy with neglected AS. However, this clinical finding is not generally pathognomonic, as it is observed in only 36.4–46% of patients with SCT [9].

The high position of the affected testicle, despite the fact that in classic literature is considered a pathognomonic clinical sign of SCT, is not accepted by many authors (Table 4).

Table 4. Relative frequency of high position of twisted testicle in SCT cases.

| Author | High position of twisted testicle (%) |
|--------------------------------|---------------------------------------|
| Beni-Israel <i>et al.</i> [15] | 33 |
| Murphy <i>et al.</i> [11] | 50 |
| Eaton <i>et al.</i> [30] | 55 |

In a study of 338 boys with AS, of which 51 had SCT, Barbosa *et al.* suggested the following scoring system [31]: testicular swelling (2 points), testicular hardness (2 points), absence of cremasteric reflex (1 point), nausea or vomiting (1 point) and high position of the affected testicle (1 point). Patients were classified into 3 groups according to the achieved score: low risk for SCT group (0–2 points), intermediate risk group (2–5 points) and high-risk group (6–7 points). They concluded that utilization

of this risk score could establish SCT diagnosis in 80% of the cases, with a high sensitivity, intermediate specificity and high negative and positive predictive values.

Crucial clinical signs for AE diagnosis are the painful swelling of the affected epididymis — compared to the dimensions of the unilateral — normal — and the positive Prehn's sign (lifting of the affected hemiscrotum relieves the pain). After comprehensive research of the relevant literature, we did not identify any study assessing the usefulness of Prehn's sign in the diagnosis of AE.

Pathognomonic clinical findings in AT are the localized sensitivity of the upper pole of the testicle and the identification of the blue dot sign during the transillumination of the affected hemiscrotum.

However, blue dot sign is found in 14–22% of all cases of AT [32]. Due to indefinable and mild pain, male patients usually arrive late at the Emergency Department (in most cases, 12–24 hours after the onset of the disease process), resulting in a complicated clinical presentation, with swelling of the affected hemiscrotum (82.6%), redness (62.1%) and diffuse sensitivity (94.6%) representing the major clinical findings (neglected acute scrotum).

Essentially, the physician is usually unable to assess thoroughly the intrascrotal structures, due to the extent of the inflammation process. All three discussed clinical entities may be responsible for this inflammatory process (Table 5).

Table 5. Relative frequency of inflammatory signs in most frequent causes of acute scrotum.

| Author | % Redness-swelling in SCT | % Redness-swelling in appendiceal torsion | % Redness-swelling in AE | % hardness of testicle in SCT | % hardness of testicle in appendiceal torsion | % hardness of testicle in AE |
|----------------------------|---------------------------|---|--------------------------|-------------------------------|---|------------------------------|
| Cass <i>et al.</i> [8] | 18 | | 19 | 68 | | 20 |
| Waldert <i>et al.</i> [10] | 75 | 35 | 92 | | | |
| Makela <i>et al.</i> [13] | 44 | 39 | 88 | | | |
| Mushtaq <i>et al.</i> [14] | 75 | | 97 | | | |

It is not possible to evaluate the localized sensitivity in the upper pole of the testicle or the enlargement of the epididymis in the neglected AS. This finding is not in favor of AT or AE, as it can be seen in 7.3–18% of all SCT cases [26, 27]. The latter finding might perplex AE diagnosis.

In addition, epididymal swelling is observed in 43% of patients with SCT and 77% of patients with AT [10, 27]. Nussbaum *et al.* documented presence of epididymis swelling — based on ultrasonographic findings — in 94% of patients with SCT [33].

The diagnosis of TTT is based on detailed medical history (episode of acute scrotal pain accompanied with nausea and vomiting, which recedes within a few minutes)

and meticulous physical examination. Palpation of the affected hemiscrotum reveals the presence of a mild sensitivity in 56% of all cases. The affected testicle might be slightly enlarged, with normal turgidity, while the cremasteric reflex is normally produced. Clinical evaluation should focus on the exclusion of specific indications of AE (swelling and tenderness of the epididymis, positive Prehn's sigh) or AT (sensitivity to the upper pole of the testicle, blue dot sign).

Another pathognomonic finding is the transverse position of the affected testicle [32, 34].

After multiple episodes of TTT, the physician might confirm indirect indications of ischemic trauma, such as smaller size and hardness during palpation of the affected testicle, or even testicular atrophy in more severe cases.

Physical examination of a boy with acute idiopathic scrotal edema reveals the presence of scrotal edema and redness, and possibly the disappearance of its wrinkles due to dartos contraction. Examination of the intra-scrotal structures is usually normal. However, when scrotal edema is severe, clinical examination may become difficult. The scrotal palpation reveals the presence of mild tenderness and a slight increase in temperature. Finally, enlarged groin lymph nodes can also be found.

Imaging findings

The classic imaging modality for the emergent diagnostic approach of AS is the Doppler ultrasound. The absence of blood flow within the affected testis is a strong indication of its torsion. In addition, maintenance of its normal architecture indicates that it is possible to survive after detorsion.

However, absence of blood flow in the affected testicle is not a safe diagnostic criterion in cases of incomplete or partial SCT or TTT. Waldert *et al.* reported that in 3.22% of SCT cases testicular blood supply was not affected, leading to a misdiagnosis [10]. This was attributed to the fact that SCT was partial with venous congestion of the affected testis, without complete interruption of arterial blood supply.

Many authors believe that the presence of normal arterial blood supply should not exclude SCT diagnosis [35–39]. The presence of normal arterial blood supply in SCT was reported by Kalfa *et al.* to reach up to 24% [39]. It was therefore deduced that the impact of SCT on microcirculation depends on both the number of rotations and the degree of the spermatic cord rotation.

In a previous study by Tadtayev and Mazaris assessing the correlation of Doppler ultrasound findings with operative findings in 117 patients with AS, sensitivity and specificity of this imaging method were 100% and 71.43%, respectively [40].

Diagnostic accuracy measures of Doppler ultrasound in AS across various studies are: specificity ranges from 75.2%–100%, sensitivity ranges from 86% to 100%, positive predictive value ranges from 80.4%–96.9% and lastly, negative predictive value ranges from 89% to 100% [35–39].

The safest imaging indication of SCT is the identification of the spermatic cord rotations (like a snail shell-shaped mass) with length ranging from 7 to 33 mm, by performing high-resolution ultrasound scanning with frequencies of 10–12 MHz. Regarding sensitivity, high-resolution ultrasound features 96% sensitivity versus 76% of Doppler ultrasound [39].

In neglected AS there is often a difficulty in distinguishing primary AE from secondary inflammation due to AT. A twisted appendix with non-reversible ischemia may not be depicted with ultrasonography, when it is concealed by the inflamed and swollen epididymal head. The latter may lead to unnecessary operative exploration.

In the case of acute idiopathic edema, the presence of homogeneous, typically, thickened scrotal wall ranging from 3.4 to 13.4 mm (average 7.7 mm) is revealed during ultrasound examination — in transverse sections. The second pathognomonic finding is the increased vascularization of the scrotum. The scrotum receives arterial blood supply from the anterior and posterior scrotal arteries. The anterior scrotal artery arises from the external pudendal artery, while the posterior is derived from the internal pudendal artery. Transverse imaging of these vascular branches and scrotal hyperemia leads to the formation of the Fountain's sign [41]. Other findings include the emergence of both a growing reactive hydrocele (in 20% of cases) and swollen and hyperemic groin lymph nodes [42].

In episodes of unexplained scrotal pain, which may be due to episodes of incomplete torsion, if the patient undergoes a Doppler ultrasound after the episode, increased vasculature of the affected testicle may be observed. Pathognomonic ultrasonographic finding is the transverse position of the affected testis, which substantially contributes to the documentation of bell clapper deformity.

Concluding remarks

- Most of the clinical entities encompassed under the diagnosis of AS may present in a similar way.
- In neglected cases of AS, diagnostic procedure becomes difficult. Classic pathognomonic findings may not be present.
- Failure to exclude SCT diagnosis should constitute an indication of emergent surgical exploration.

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