Massive Scrotal Cystocele as a Rare Cause of Obstructive Uropathy

D. Novăcescu[1,2], S. Laţcu[1,2], A. Croitor[1,2], V. Dema[1,2], C. Tarța[3,4], A.A. Cumpănaș[1,2]

[1] Department of Urology, "Pius Brînzeu" Emergency Clinical County Hospital, Timişoara, Romania
[2] Department of Urology, "Victor Babeș" University of Medicine and Pharmacy, Timişoara, Romania
[3] Department of General Surgery, "Pius Brînzeu" Emergency Clinical County Hospital, Timişoara, Romania
[4] Department of General Surgery, "Victor Babeș" University of Medicine and Pharmacy, Timişoara, Romania

ABSTRACT

Introduction and Objectives. Inguinal herniation of lower urinary tract viscera is uncommon. Massive bladder herniation, i.e. scrotal cystocele, involving the terminal ureters bilaterally and causing obstructive uropathy, with chronic and progressive renal function impairment, is particularly rare and scarcely documented. Although pathognomonic clinical elements have been described, most cases are asymptomatic and remain undiagnosed preoperatively. Disease pathogenesis is incompletely understood and therapeutic management may be quite problematic, especially in occult cases and/or neglected, advanced disease clinical phenotypes.

Materials and Methods. The current case report focuses on the diagnostic characterization of scrotal cystocele, by reviewing the clinical assessment and multimodal imaging analysis of a 73-year-old patient, with progressive renal function deterioration, and a neglected, irreducible, massive right inguinal hernia. The morphological impact upon the upper urinary tract, as well as the intimate relationship between the herniated elements involved (i.e. bladder and terminal ureters), are identified and characterized using contrast-enhanced computer tomography (CT). The implications of these anatomical findings, regarding the personalized surgical strategy, are thoroughly discussed.

Results. Surgical exploration of the right inguinal canal revealed a direct, extraperitoneal, voluminous, bladder and terminal ureter herniation, protruding medially and being intimately adherent to the fascial covering of the spermatic cord, while extending within the distended right scrotal cavity. We performed a tactical right orchiectomy, to facilitate a modified hernia repair technique (posterior inguinal canal anatomic repair, with Liechtenstein-like mesh placement), and allow for adequate and durable parietal reconstruction. Notwithstanding minor postoperative complications, the patient made a full recovery, with normalized renal function parameters and no recurrence after 3 years.

Conclusions. Adequate preoperative diagnosis and clinical assessment of scrotal cystoceles will facilitate therapeutic decision-making. Persistent lower urinary tract symptomatology, in patients with inguinal hernia and positive risk factors for scrotal cystocele (obesity, age > 50 years, benign prostatic hyperplasia), should mandate further morphological assessment, beyond the routine abdominal/pelvic ultrasonography, especially in the presence of obstructive uropathy.

Key-words: scrotal cystocele, obstructive uropathy, inguinal hernia modified repair, direct extraperitoneal bladder and bilateral terminal ureter inguinal hernia.

Correspondence to:
Dr. Silviu Laţcu M.D.
"Pius Brînzeu" Emergency Clinical County Hospital, Department of Urology
156 Liviu Rebreanu av., code 300723, Timișoara, Romania
Tel: +40741032154
e-mail: silviu.latcu@umft.ro

INTRODUCTION AND OBJECTIVES

One of the earliest recorded and most common surgical pathologies, inguinal hernia occurs when abdominopelvic visceral elements protrude into the inguinal canal. Essentially, this mechanism of organ displacement is facilitated by either a point of anatomic weakness, i.e. direct herniation - via the posterior wall of the inguinal canal, through the area of least resistance (Hesselbach's triangle), medial to the inferior epigastric vessels; or a proper continuity solution, within the normal fibro-muscular parietal tissue, i.e. indirect herniation - usually congenital, following the path of the descending testicle, via an incomplete closure of the processus vaginalis and/or deep inguinal ring, lateral to the inferior epigastric vessels. Inguinal herniation of pelvic organs can be additionally classified with regard to their relationship with the peritoneum, into: paraperitoneal hernias - the hernia lies along the medial wall of the peritoneal sac; trans/intraperitoneal hernias - the herniated viscera are completely covered by peritoneum; and extraperitoneal hernias – without any relation with the peritoneum [1]. Obviously, these pathogenic distinctions can only be truly assessed intraoperatively.

Overall, inguinal hernias account for 75% of abdominal wall hernias, with a lifetime risk of 27% in men, respectively 3% in women, and approximately 20 million inguinal hernia surgical repairs being performed annually worldwide [2]. Conversely, even though inguinal hernias are common, the involvement of lower urinary tract viscera (i.e. bladder diverticulum, healthy bladder wall and/or terminal ureters) is quite rare. In fact, even though bladder involvement occurs most frequently, inguinal bladder hernias (IBHs) only account for 0.5–3% of all lower abdomen hernia cases, within the general population [1,2]. However, IBH incidence rates are greatly increased by certain risk factors, i.e. male gender, age >50, obesity – body mass index (BMI)>30, bladder outlet obstruction (benign prostatic hyperplasia specifically), bladder wall disease (chronic inflammation, malignancy), previous pelvic injuries, abdominal/pelvic surgical history, decreased detrusor tonus and/or pelvic muscular weakness [2,3]. Thus, in obese older males, bladder involvement is reported to occur in ~10% of inguinal hernia cases. Even so, as reported within a recent literature review, since 1951, when Levine firstly defined the notion of IBH, using the term "scrotal cystocele" to describe a large inguino-scrotal bladder protrusion, only 116 cases of IBH have been identified, with less than a third of these cases manifesting scrotal extension [4]. Additionally, advanced massive inguino-scrotal bladder herniation, i.e. a proper scrotal cystocele, has been even more scarcely reported, with less than 30 cases currently published in the medical literature [5]. Moreover, even among the limited individual cases reported, considerable heterogeneity exists, regarding both morphopathological characteristics and, implicitly, clinical manifestations.

Seeing as inguinal hernias may involve different parts of the bladder, i.e. preexisting diverticula, limited or extensive areas of the healthy bladder wall, even almost the entire organ, a wide range of symptomatology has been reported. For the most part, IBHs are usually asymptomatic, reflecting a very limited degree of bladder involvement, but may cause a wide range of nonspecific, mixt, storage and/or voiding, lower urinary tract symptoms, i.e. urinary frequency, urgency, weak urinary stream, dysuria, hematuria, nocturia, post-micturition dribbling, as disease progression occurs. Rarely, in more advanced cases, i.e. scrotal cystoceles, more severe complications may occur, such as urinary tract infections (UTIs), bladder strangulation, infarction, and perforation, ureteral hernia involvement and/or extrinsic compression, with obstructive uropathy and secondary acute renal failure, as well as bowel co-involvement and ischemia [2,5]. Specifically regarding scrotal cystoceles, a rare, yet pathognomonic clinical finding has been reported, known as Mery's sign, meaning a two-step urination process, with an initial, very limited, spontaneous voiding, followed by manual compression of the scrotum, necessary to facilitate additional voiding, resulting in the significant/complete reduction of hernia volume post-micturition [6]. Regardless, with most cases being typically asymptomatic, the diagnosis of IBHs is usually made intraoperatively, with <7% of cases being diagnosed preoperatively, through

endoscopic (cystoscopy) and/or imaging modalities (ultrasound, voiding cystography, CT), and ~16% of cases being diagnosed postoperatively, as a result of significant complications occurring (parietal suppuration or vesico-cutaneous fistula) [7].

In contrast, even though inguino-scrotal hernias may involve the distal ureters bilaterally, this morphopathological configuration is extremely uncommon. Despite being firstly described in the late 18th century, only ~70 cases of ureteral inguinal herniation have been reported so far in medical literature [4]. Most frequently, these cases occur in pediatric patients, in the presence of developmental defects, and less frequently in adults, either in the context of obesity and/or post renal transplantation. In fact, currently, the available literature reports under 10 cases of ureteral inguinal herniation occurring in adults with native kidneys and no previous surgical manipulation of the urinary tract [8]. Thus, apparently there are two main pathogenic mechanisms involved in the occurrence of inguino-scrotal ureteral herniation, namely: either within the natural evolution of a sliding inguino-scrotal bladder hernia, which will eventually traction and/or extrinsically compress the distal ureters, or within the context of renal transplantation, due to the anterior localization of the transplanted ureter, within the prevesical space of Retzius [9]. In contrast to the mostly occult IBHs, ureteral inguinal hernias are usually symptomatic, due to the secondary obstructive uropathy they generate. Thus, further morphological assessment is generally mandated, allowing for the definitive diagnosis of ureteral involvement to be established preoperatively in most cases [8].

In the current paper, we provide a detailed clinical report regarding a neglected, advanced case of scrotal cystocele, with bilateral ureteral involvement and secondary severe bilateral ureterohydronephrosis, leading to a progressive decline of renal function.

MATERIALS AND METHODS

A 73-year-old patient, non-smoker, with class II obesity (BMI=38.1 kg/m2), undergoing medical treatment for multiple cardiovascular and metabolic comorbidities (grade II primary hypertension, chronic coronary syndrome, NYHA II heart failure, first degree atrioventricular block, LOWN 3 ventricular extrasystoles, primary hypercholesterolemia and asymptomatic hyperuricemia), and previously diagnosed with stage G3b chronic kidney disease, is referred to our clinic for urological evaluation by his attending nephrologist, with the clinical suspicion of obstructive uropathy, as a possible explanation for the recent additional decline in renal function.

Anamnesis further revealed a long standing history of umbilical and bilateral inguinal hernia. The inguinal hernias were initially diagnosed ~20 years ago, yet they remained untreated due to the patient's fears regarding the required surgical interventions. On the right side, the inguinal hernia reportedly increased in size considerably within this timeframe, becoming quite bothersome. Moreover, the patient reported a history of recurrent UTIs, with persistent, mixt, storage and voiding lower urinary tract symptoms: urinary frequency, urgency, weak urinary stream, dysuria, nocturia and post-micturition dribbling, uninfluenced by curative antibiotic treatment. In recent years, the patient reports developing a curious two-step urination process, with an initial, limited spontaneous voiding, followed by manual compression of the scrotum, leading to additional voiding and a significant reduction of the right inguinal hernia volume post-micturition (positive Mery's sign).

Physical examination of the pelvic area confirmed the presence of the aforementioned bilateral inguinal hernias. A massive right scrotal bulge (35x30x20 cm) was identified, irreducible and incoercible, with right-sided inguinal and scrotal spontaneous discomfort, mild pain upon palpation and trophic modifications of scrotal skin. The ipsilateral testicle and spermatic cord were apparently inseparable from the scrotal mass. The patient was apyretic and showed no signs of peritoneal irritation, with normal bowel movements and no nausea, vomiting, diarrhea or constipation). Digital rectal exam revealed an enlarged prostate, with no signs of malignancy.

Abdominal ultrasound revealed bilateral renomegaly and renal ptosis, with severe grad IV bilateral ureterohydronephrosis, a bilaterally decreased renal parenchymal index and greatly distended hydroureters, traceable until the level of the iliac vessels. The bladder had a small size overall and was filled with ~ 300 ml of urine, while the prostate could not be visualized. A linear probe was used to examine the groin/scrotum and a fluid-filled lesion was identified within the right scrotum, demonstrating a beaked appearance in its cranial portion, which could be traced entering the right inguinal canal. A Foley catheter was placed and evacuated ~400 ml of clear urine, with no change in appearance or size of the scrotal fluid-filled lesion, yet allowing for the visualization of an enlarged prostate (45 cm3). The right testicle was displaced infero-laterally, hypotrofic and showed reactive fluid accumulation (hydrocele).

The clinical findings documented upon initial, ambulatory, assessment were suggestive of a massive right inguinal bladder hernia, complicated with obstructive uropathy through ureteral involvement. The patient was admitted in the Urology Department for further investigations. Upon admission, vital signs were within normal limits. Urine cultures were sterile. Biologically, the only relevant modifications were nitrogen retention (serum creatinine=2 mg%, serum urea=102 mg%) and hyperuricemia (9.7 mg%). A contrast-enhanced CT of the abdomen and pelvis, with excretory exposure, was performed in order to confirm the diagnosis.

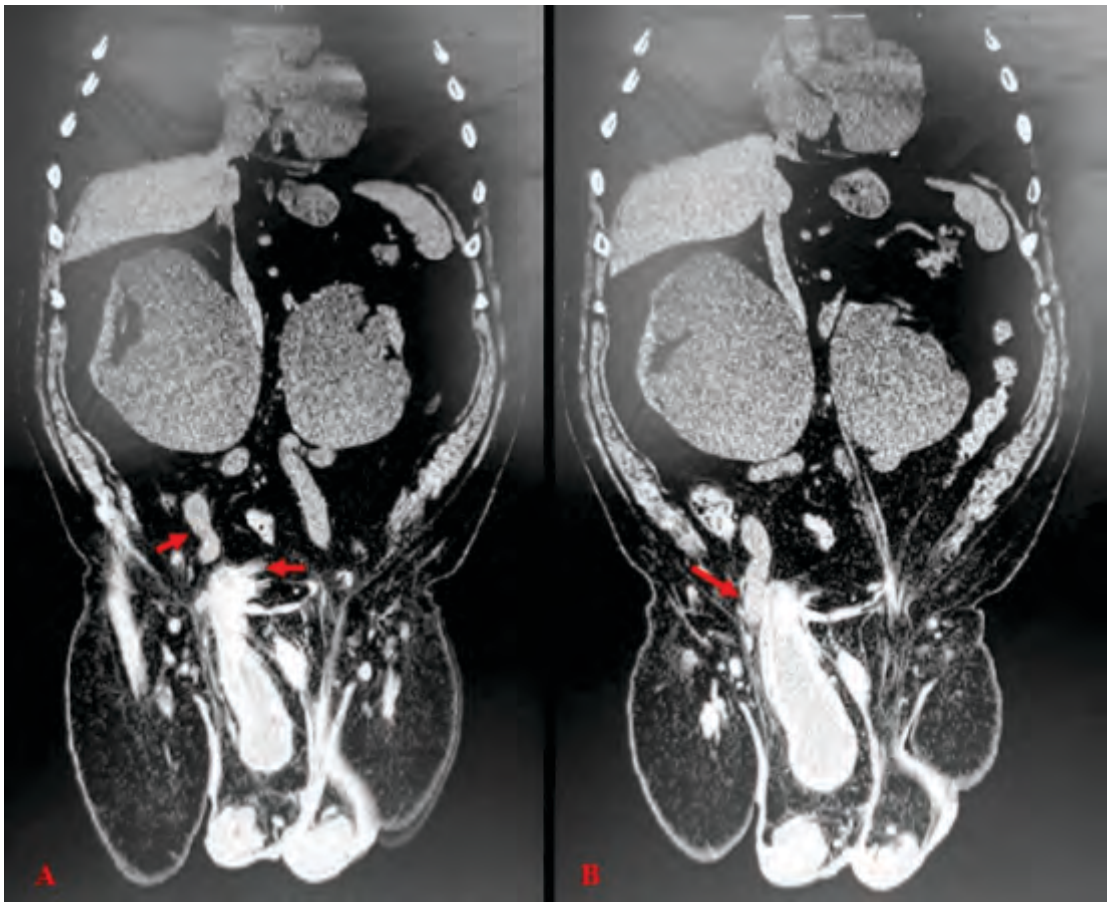


Image 1. Contrast enhanced CT of abdomen and pelvis, coronal view. A: Bilateral ureteral implantation in herniated bladder trigone; B: Involvement of terminal right ureter within inguinal herniation

The CT scan corroborated the ultrasound findings, showing a heavy distended upper urinary tract, with absent excretion at 15 and 30 minutes (Img. 1, Img. 2A). The hydroureters were visualized as they descended towards the ectopic bladder trigone (Img. 1A), converging towards the right inguinal area (Img. 2B). The distal aspect of the right ureter was found to additionally protrude outward and inferiorly, alongside most of the bladder, to form a large inguinoscrotal herniation on the ipsilateral side (Img. 1B). Moreover, in the pelvic area, this large right inguinal hernia containing the terminal right ureter and trigonal bladder wall could be view in comparison to the much milder left intestinal inguinal herniation (Img. 2C). Finally, within the lower right scrotum, a consistent portion of bladder wall was visible (Img. 2D).

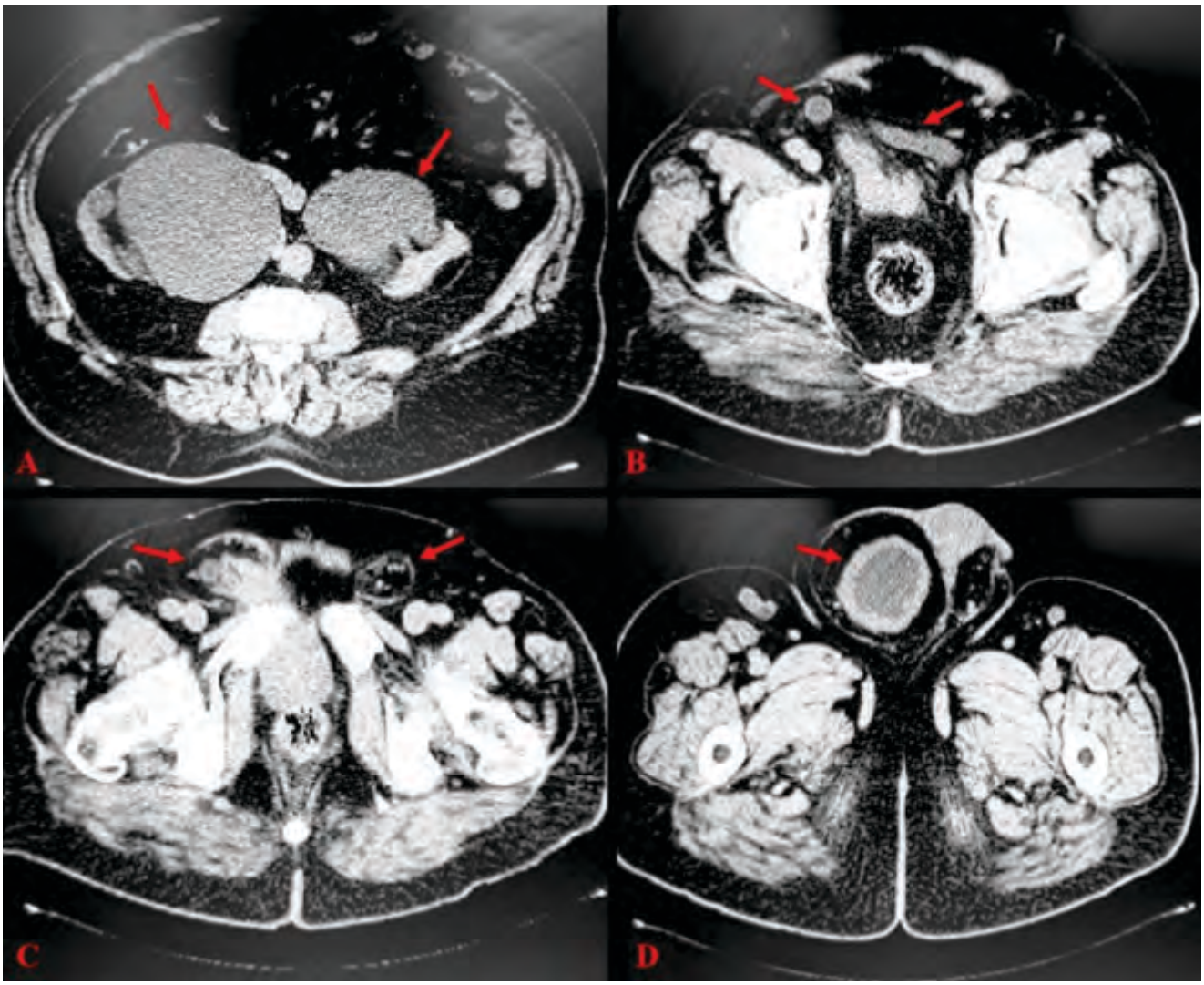


Image 2. Contrast enhanced CT of abdomen and pelvis, axial view. A: Severe bilateral ureterohydronephrosis, with areas of parenchymal index reduction and no urinary passage of contrast at 15 min; B: Pelvic portion of hydroureters converging towards the right inguinal canal, alongside the bladder; C: Comparative view of large right inguinal hernia containing the terminal right ureter and trigonal bladder wall versus left intestinal herniation; D: Extensive scrotal displacement of bladder wall.

Thus, we established the definitive diagnosis of scrotal cystocele with bilateral ureteral involvement and secondary obstructive uropathy. We extensively reviewed the anatomy in preparation for curative surgical treatment (right inguinal repair). The need to preoperatively drain the upper urinary tract, through nephrostomy, was taken into consideration and abandoned due to the chronic nature of the upper urinary tract modifications and the lack of any foreseeable additional benefits.

RESULTS

Once the definitive diagnosis of advanced scrotal cystocele was established, in order to prepare for recommended curative surgical treatment, the patient underwent a thorough preoperative work-up, i.e. cardio-pulmonary assessment and evaluation of routine biological parameters. The patient was deemed clinically fit for surgery. Additionally, microbiological sterility of the urinary tract was considered a mandatory prerequisite for surgical treatment and was confirmed by preoperative urine culture. Thus, for bacterial prophylaxis, a single dose of 3rd generation Cephalosporin was administered intraoperatively.

Under spinal anesthesia, the patient was placed in a standard supine position, scrubbed with betadine and draped. A Foley catheter was placed in a completely sterile manner and access to the distal end of the catheter was ensured. Beginning 2 cm medially to the pubic tubercle, an oblique, 5 cm right inguinal incision was performed, parallel and 1-2 cm cephalad to the projection area of inguinal canal, i.e. an imaginary line uniting the antero-superior iliac spine and the pubic tubercle. The underlying subcutaneous tissue was dissected to reveal the external oblique fascia/anterior wall of the inguinal canal, which is further incised to allow for the surgical exploration of the inguinal canal and the dissection and isolation of its visceral contents.

Initially, the anterior/inferior aspect of the fascial covering of the spermatic cord, i.e. the external spermatic, cremasteric and internal spermatic fasciae, was identified and dissected (Img. 3A). Following this superficial sharp fascial dissection, blunt digital dissection was used posteriorly, in an attempt to isolate the spermatic cord circumferentially. However, this attempt was unsuccessful, due to the lack of an identifiable posterior-medial dissection plain between the fascial coverings of the spermatic cord and the hernia contents, seemingly completely enveloped by these fasciae as well. Thus, the possibility of an indirect bladder herniation, occurring via a defect at the level of the deep inguinal ring, following the inguino-scrotal trajectory of testicular descent, and being completely enveloped within the cremasteric fascia, alongside the vascular elements of spermatic cord, became apparent and plausible. Therefore, the incision was extended and gentle manual traction allowed for the complete exteriorization of the spermatic cord and testicle, together with the herniated bladder, within the operative field.

Curiously, upon meticulous inspection, it soon became apparent that, in fact, the spermatic cord fascial coverings only extended towards the hernia components, without enveloping them completely. Due to chronic compression and fibrotic remodeling, the posterior/medial cremasteric fascia became extremely adherent and thus intimately bound to the anterior, inferior and lateral aspects of the hernia sac, i.e. a fibrotic pseudo-membrane, throughout the inguinoscrotal trajectory of both the spermatic cord (located laterally) and the hernia contents (located medially). Thus, to properly isolate the spermatic cord and continue the exploration of hernia contents, the cremasteric fascia had to be incised (Img. 3B).

Further blunt fascial dissection allowed for the abolishment of the fibrotic pseudo-membranous hernia sac, but also, implicitly, the fascial covering of the spermatic cord. A common mass of fatty tissue subsequently emerged, containing both the abundant herniated perivesical fat, but also the constitutive spermatic cord fat, the last protective covering of the testicular vessels. Palpation allowed for the identification of the testicular artery, and meticulous sharp dissection was used to isolate the spermatic cord from the hernia content (Img. 3C). This process proved difficult to finalize due to widespread fibrotic adhesions. Unavoidably, a few minor vascular cord elements were encountered

and ligated. The right testicle was also isolated, but demonstrated a hypotrophic appearance, with multiple degenerative fatty inclusions and a reactive hydrocele of significant volume (Img. 3C/D).

Subsequently, deeper dissection of the hernia contents allowed for the identification of the parietal defect, situated medially to the inferior epigastric vessels, and of a tight, fibrotic and hernia neck (Img. 3D), thus anatomically confirming the direct nature of the inguinal bladder herniation. Moreover, the herniated contents seemingly had no relationship to the peritoneum, i.e. extraperitoneal bladder herniation. Dissection of the hernia neck was cumbersome and led to additional proximal spermatic cord vascular lesions, yet finally allowed for the reduction of the hernia contents, which were repositioned within the pelvis, in their physiological anatomic layout (Img. 4A).

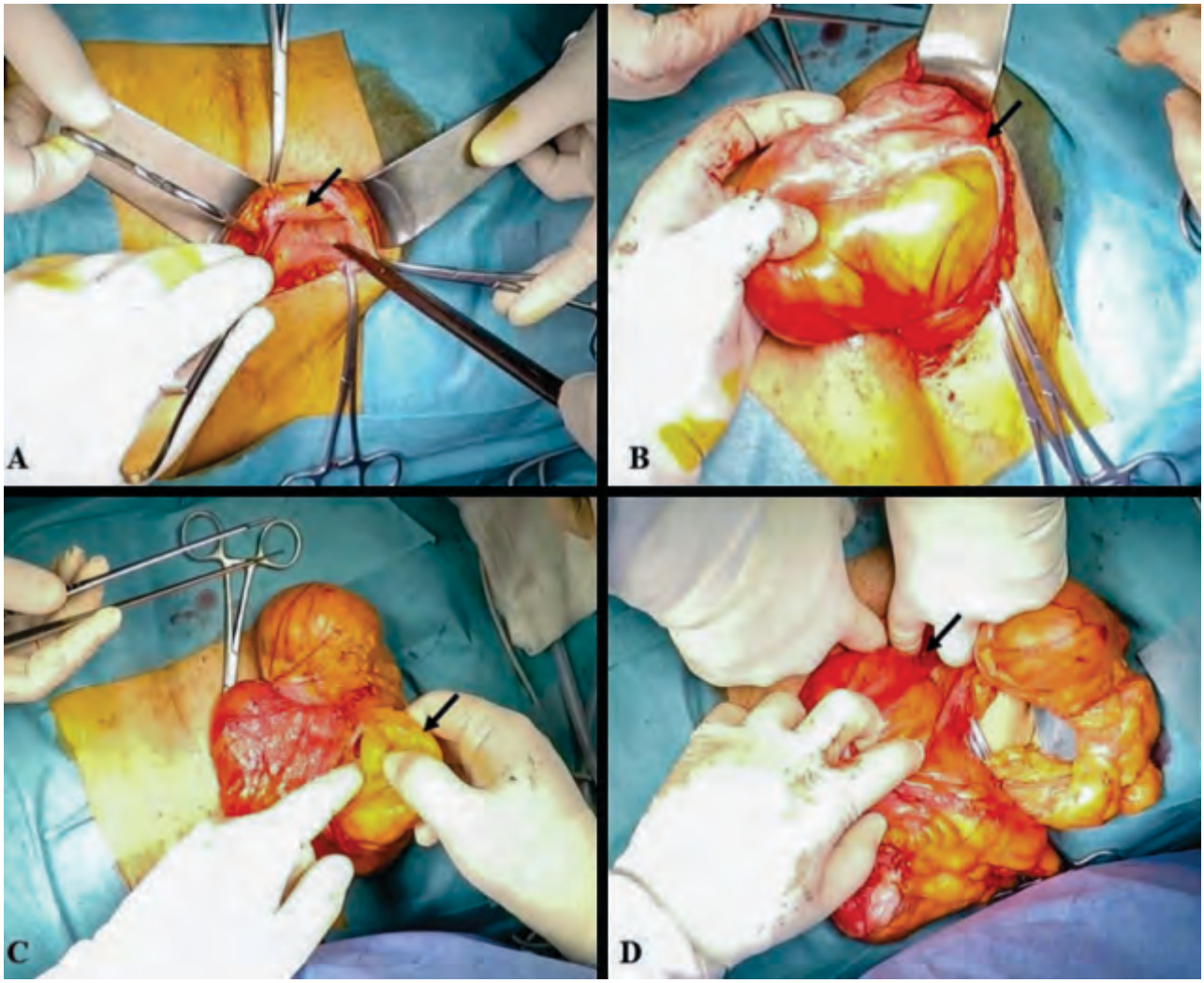


Image 3. Intraoperative images. A: Initial dissection of spermatic cord fascial covering; B: Exposure of hernia contents by cremasteric fascia incision; C: Identification of spermatic cord vascular elements; D: Dissection of parietal defect and hernia neck.

Taking into account the large parietal defect requiring surgical repair, as well as the aforementioned hypotrophic / degenerative right testicle morphological traits, and the multiple possibly ischemia inducing vascular spermatic cord lesions, the surgical team decided to perform a tactical right inguinal orchiectomy, by ligating and dividing the spermatic cord proximally, at the level of the deep inguinal ring, to ensure adequate parietal reconstruction and facilitate durable therapeutic results.

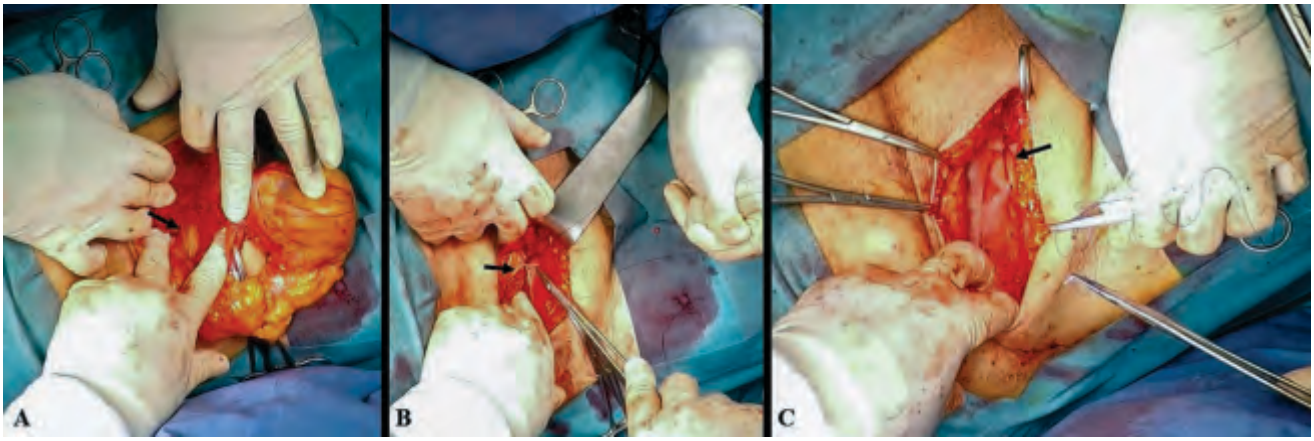


Image 4. Intraoperative images. A: Reduction of direct inguinal hernia contents and identification of inguinal ligament; B: Anatomic posterior inguinal wall repair by approximation of the conjunct tendon/internal oblique fascia to the inguinal ligament; C: Lichtenstein-like mesh implantation, between the internal and external oblique muscles.

A modified hernia repair technique was utilized, which combined a posterior inguinal canal anatomic repair, i.e. the approximation of the conjunct tendon/internal oblique fascia to the inguinal ligament using a single continuous Surjet suture (Img. 4B), with a Liechtenstein-like polypropylene mesh implantation, between the internal and external oblique muscles/fasciae (Img. 4C). Lastly, a scrotal drainage tube was placed through counter-incision and the inguinal incision was closed using discontinuous sutures.

Postoperatively, the patient's evolution was favorable under supportive care. A single prophylactic dose of anticoagulant (low molecular weight heparin) was administered postoperatively (after 8 hours). The Foley catheter remained in place for the remainder of the hospital stay and, remarkably, within the first 24 hours postoperatively, the patient had a total diuresis of 6.5 liters. The renal function normalized within 24 hours postoperatively as well, with a marked decrease in creatinine and urea levels, and implicitly, an improved glomerular filtration rate. The scrotal drain was removed on the second day postoperatively and the patient was discharged a day later, with the Foley catheter in place and the recommendation to maintain set catheter for an additional week. The pre-discharge ultrasound showed a significant decrease in the severity of the ureterohidonephrosis bilaterally. Chronic α-blocker therapy was recommended, with the indication to follow up with a serum PSA, after Foley removal, in order to establish a baseline and additionally commence 5α-reductase inhibitors.

Unfortunately, within a week post-discharge, the patient had to readmitted for an infected right scrotal lymphocele. Antibiotic therapy (Levofloxacin) and drainage of the scrotal collection allow for a prompt and complete recovery. Upon discharge, the surgical incision had completely healed and the ureterohidronephrosis had been completely resolved bilaterally. Spontaneous micturition had reinstated after Foley removal, with minimal lingering voiding discomfort, possibly due to the persistent hour-glass bladder shape, visible on ultrasound (provided sufficient bladder

distension). Even so, post-voiding residuum was minimal (~50 ml). Later on, micturition completely normalized and the bladder regained its physiological shape. The 3 year follow-up evaluation showed no signs of right inguinal hernia recurrence.

DISCUSSIONS

Our current paper reports on the clinical phenotype of an exceptionally rare clinical entity, namely the scrotal cystocele with bilateral ureteral involvement. Even when compared to the extremely limited references available in current medical literature, our case stands out as it present multiple anatomical particularities therapeutic challenges.

Firstly, from an anatomic perspective, it has been established that IBHs may occur either as direct inguinal visceral protrusions, medially to the epigastric vessels, or indirect protrusions, via the internal deep inguinal ring, yet it seems that direct IBHs occur most frequently. Even so, inguinoscrotal bladder herniation, particularly when associated with ureteral herniation, is without a doubt an exceedingly rare clinical entity [3-5, 10]. Specifically regarding ureteral inguinal hernias, the main anatomic variant is indirect, without a proper peritoneal sac, i.e. paraperitoneal or extraperitoneal. The paraperitoneal variant is much more common and accounts for 80% of uretero-inguinal hernias. In this case, the ureter will be intimately adherent to the posterior peritoneum and will herniated alongside the peritoneal sac into the inguinal canal, being often accompanied by the herniation of other digestive organs – colon most frequently [11,12]. Conversely, in the much rarer extraperitoneal variant (20% inguinal ureteral hernias), there is no discernable relationship whatsoever between the peritoneum and the herniated ureter [13]. Even though initially, extraperitoneal ureteral herniation was thought to cause due to a congenital embryonic defect generating a fusion between the ureter and genito-inguinal ligaments [11], multiple reports have since demonstrated that these uretero-inguinal hernias may also occur in the absence of any congenital anomalies or previous hernia repairs [12,14].

Additionally, obesity and collagen synthesis deficiencies have been incriminated as possible risk factors for uretero-inguinal hernias [15]. The case provided herein represent a direct, extraperitoneal, massive inguinal bladder herniation with bilateral ureteral co-involvement, with the right distal ureter being clearly visible on imaging as entering the inguinal herniation.

Secondly, from an epidemiological perspective, our data supports the notion that obesity, advanced age and bladder outlet obstruction (especially untreated benign prostatic hyperplasia) represent important risk factors for the occurrence of IBHs. Conversely, we must state that there are very few cases documented in medical literature of uretero-inguinal herniation occurring in adults with native kidneys and no previous surgical manipulation of the urinary tract [8]. Thus, our report brings an important element of novelty to existing data.

Lastly, it is important to note that, to this day, most IBHs are diagnosed intraoperatively, while only < 7% preoperatively and, confoundingly, ~16% postoperatively, due to significant complications (iatrogenic bladder injury leading to urinary fistula formation) [7]. Surprisingly, even in preoperatively confirmed cases of IBH, accidental bladder injury, is still currently reported to occur in ~12% of hernia repairs [16]. Therefore, it is imperative that clinicians adjust current preoperative assessment protocols, in order to facilitate a more rigorous definitive diagnosis of inguinal hernia contents and, hopefully, reduce surgical complications [16,17].

CONCLUSIONS

All in all, as a general rule, it is becoming increasingly apparent that, in certain high risk populations (obesity, age > 50 years, benign prostatic hyperplasia), in the presence of an inguinal hernia, an increased level of clinical suspicion regarding occult bladder or ureteral involvement should be maintained. Ideally, in these populations, the contents of the inguinal hernia should be assessed preoperatively using CT and irrefutably identified. Adequate preoperative diagnosis and clinical assessment of scrotal cystoceles will facilitate therapeutic decision-

making. Persistent lower urinary tract symptomatology, in patients with inguinal hernia and positive risk factors for scrotal cystocele, should mandate further morphological assessment, beyond the routine abdominal/pelvic ultrasonography, especially in the presence of obstructive uropathy. This attitude may facilitate the reduction of intraoperative complications, as it will allow for a better understanding of IBH morphopatology and facilitate the development of a personalized modified surgical approach.

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