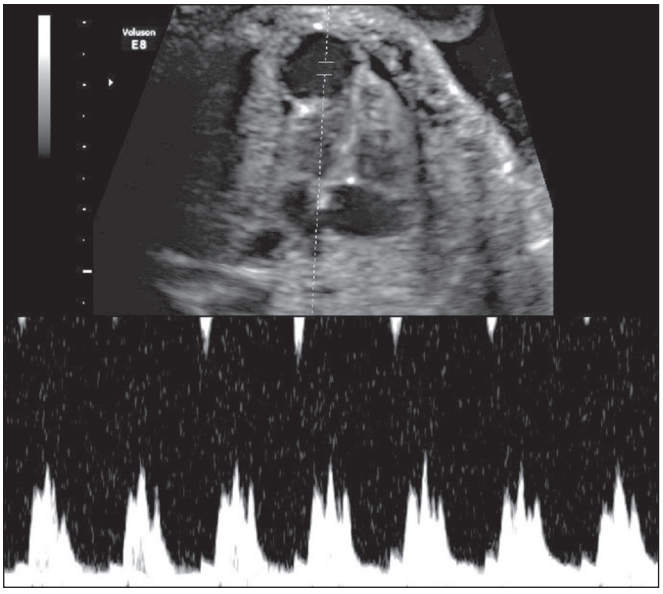

Figure 3. Spectral Doppler ultrasound showing triphasic flow and high pulsatility within the diverticulum.

no associated complications were identified. Cesarean section was performed at 35 weeks of gestation, because of fetal distress. The newborn weighed 2183 g; the 1- and 5-minute Apgar scores were 7 and 10, respectively. Postnatal echocardiography confirmed the LVD in the free wall of the left ventricle. On the 3rd day of life, the newborn underwent surgery to correct the defect, and there were no postoperative complications. The newborn remained in the neonatal intensive care unit for 9 days and was discharged from the hospital on the 18th day of life, with preserved cardiac function and no complications.

An LVD is defined as a protrusion of the free wall of a ventricle. Although it is of unknown etiology, it is probably congenital. The weakness of the myocardial wall during embryogenesis can lead to a focal protrusion of the heart wall[3]. An LVD has a narrow neck through which it communicates with the ventricular cavity; in contrast, a left ventricular aneurysm (LVA) has a wide base for connecting with the ventricular cavity[2]. The wall of an LVA is akinetic, whereas an LVD contracts synchronously with the ventricle[1,2]. An LVD can be accompanied by other congenital and cardiac anomalies such as the pentalogy of Cantrell[3].

The prenatal diagnosis of LVD or LVA can be made by ultrasound, and these anomalies are frequently accompanied by pericardial effusion, which can cause fetal pulmonary hypoplasia and progressive hydrops[4]. LVD is reported to have a more favorable long-term prognosis than does LVA[5]. The prognosis is usually favorable when there is no change in the size of the diverticulum, which was the case in the patient described here. When a fibrous LVD has a thin wall, disruption can occur and is usually fatal, although such a development is rare[6]. Prenatal monitoring, with serial examinations by fetal cardiology and cardiac surgery teams for proper programming of prenatal or postnatal interventions, will therefore be necessary[7].

REFERENCES

1. Bernasconi A, Delezoide AL, Menez F, et al. Prenatal rupture of a left ventricular diverticulum: a case report and review of the literature. Prenat Diagn. 2004;24:504–7.
2. Krasemann T, Gehrmann J, Fenge H, et al. Ventricular aneurysm or diverticulum? Clinical differential diagnosis. Pediatr Cardiol. 2001;22:409–11.
3. Treistman B, Cooley DA, Lufschanowski R, et al. Diverticulum or aneurysm of the left ventricle. Am J Cardiol. 1973;32:119–23.
4. El Kady D, Gerscovich EO, Moon-Grady A, et al. Congenital cardiac left ventricular aneurysm with pericardial effusion: early prenatal diagnosis and intervention. J Ultrasound Med. 2005;24:1011–5.
5. Hunter LE, Pushparajah K, Miller O, et al. Prenatal diagnosis of left ventricular diverticulum and coarctation of the aorta. Ultrasound Obstet Gynecol. 2016;47:236–8.
6. Ohlow MA. Congenital left ventricular aneurysms and diverticula: definition, pathophysiology, clinical relevance and treatment. Cardiology. 2006; 106:63–72.
7. McAuliffe FM, Hornberger LK, Johnson J, et al. Cardiac diverticulum with pericardial effusion: report of two cases treated by in-utero pericardiocentesis and a review of the literature. Ultrasound Obstet Gynecol. 2005;25:401–4.

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Incidentally detected massive scrotal cystocele

Dear Editor,

A 65-year-old male patient was referred to our institution for investigation of a 10-year history of epigastric pain. His pain had been progressively worsening during the past months, intensified after the consumption of solid foods. The only notable aspect of his medical history was arterial hypertension. He complained of nocturia, awakening to void about six times per night, but did not report dysuria, hematuria, scrotal swelling, or other urinary tract symptoms. Physical examination revealed epigastric tenderness and hepatosplenomegaly. Upper gastrointestinal endoscopy showed an ulcerated lesion on the greater curvature of the stomach. In the analysis of the biopsy sample, the lesion was classified as non-Hodgkin lymphoma. A computed tomography (CT) scan of the abdomen and pelvis, performed for staging, revealed an unsuspected massive inguinoscrotal hernia of the urinary bladder, a condition known as scrotal cystocele (Figures 1 and 2). The CT scan also showed moderate right-sided uronephrosis, which was attributed to extrinsic compression of the right ureter. The results of the urinalysis were unremarkable.

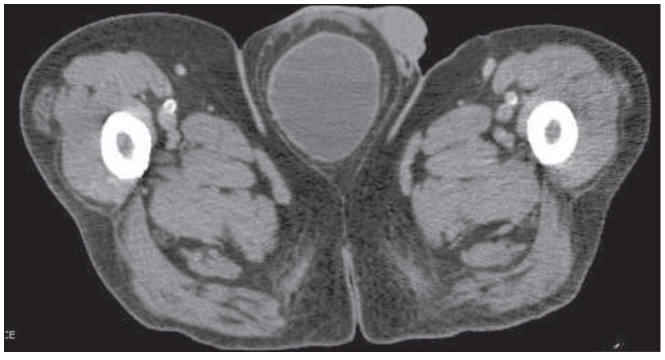

Figure 1. Axial CT scan of the pelvis showing the urinary bladder herniated into the scrotum.

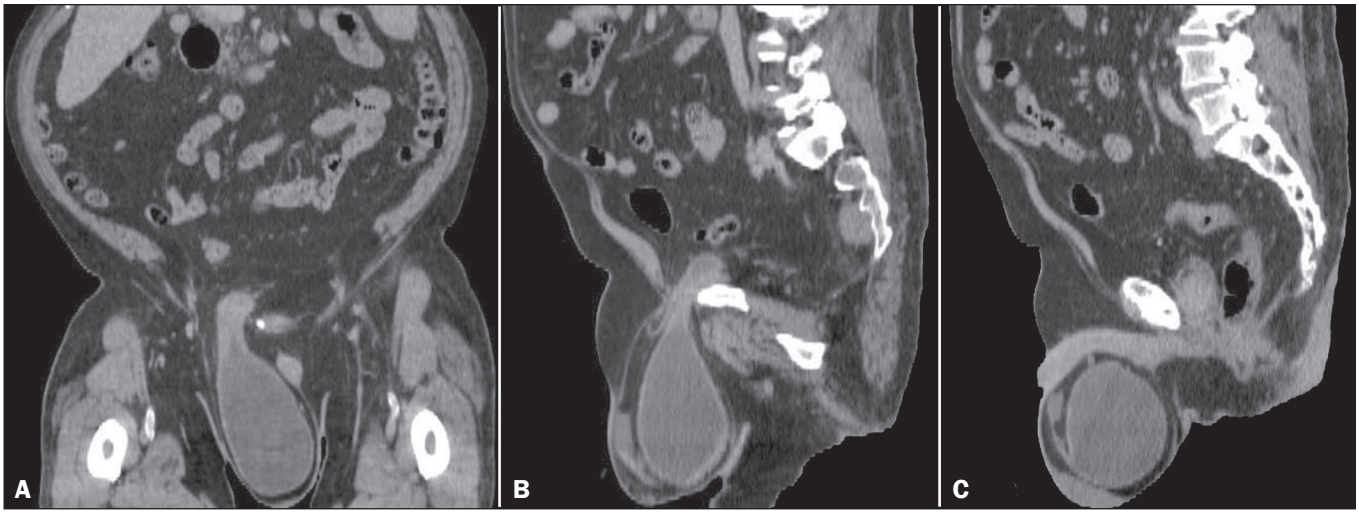

Figure 2. CT images of the abdomen and pelvis, in a coronal view **(A)** and in sagittal views **(B,C)**, showing a massive right-sided scrotal cystocele.

After work-up of the non-Hodgkin lymphoma, a hernia reduction followed by inguinal herniorrhaphy was planned in order to prevent long-term complications of bladder herniation.

Although the urinary bladder is involved in up to 4% of inguinal hernias, massive scrotal cystocele is quite uncommon[1]. Advanced age, obesity, and male gender are recognized risk factors for bladder herniation[2]. Bladder herniation is usually asymptomatic, although some patients complain of voiding-related scrotal swelling, two-stage micturition (a first spontaneous voiding followed by a second requiring manual compression of the inguinoscrotal region), urinary tract infections, or irritative lower urinary tract symptoms (LUTS) such as urgency, frequency, and nocturia secondary to bladder outlet obstruction or infection[1–3]. Possible complications of untreated scrotal cystocele include hydronephrosis, renal failure, cystolithiasis, vesicoureteral reflux, bladder necrosis, and bladder perforation[2,4,5].

The preoperative diagnosis of scrotal cystocele is important to prevent iatrogenic injury of the herniated bladder during repair surgery[3]. The condition should be suspected in all patients presenting with inguinal hernias and concomitant renal failure or LUTS, especially if a painless unilateral scrotal swelling is detected[4,5]. However, as illustrated by our case, the absence of clinically detectable scrotal swelling should not exclude the hypothesis of bladder herniation, nor should it preclude further investigation. The imaging diagnosis can be established by CT, ultrasound, cystography, or intravenous pyelography[2]. Because CT provides a clear anatomical outline of the herniated contents and allows prompt identification of complications, thereby enabling appropriate surgical planning, it is an especially valuable tool in the work-up of scrotal cystocele[6].

Hernia repair has shown to be effective in improving LUTS and reducing complications in patients with significant bladder herniation; therefore, standard treatment of scrotal cystocele consists of reduction or resection followed by herniorrhaphy[1]. Acute bladder infarction or urinary obstruction can require urgent laparotomy with resection of the affected portion of the bladder[3]. In the elective setting, partial bladder resection is often restricted to patients presenting with bladder necrosis, a tumor in the herniated bladder, or a bladder diverticulum[2]. However, timely preoperative diagnosis of scrotal cystocele remains the single most important determinant of a successful surgical outcome, making proper clinical and imaging assessments invaluable.

REFERENCES

1. Bjurlin MA, Delaurentis DA, Jordan MD, et al. Clinical and radiographic findings of a sliding inguinoscrotal hernia containing the urinary bladder. Hernia. 2010;14:635–8.
2. Kim KH, Kim MU, Jeong WJ, et al. Incidentally detected inguinoscrotal bladder hernia. Korean J Urol. 2011;52:71–3.
3. Bisharat M, O'Donnell ME, Thompson T, et al. Complications of inguinoscrotal bladder hernias: a case series. Hernia. 2009;13:81–4.
4. Casas JD, Mariscal A, Barluenga E. Scrotal cystocele: US and CT findings in two cases. Comput Med Imaging Graph. 1998;22:53–6.
5. Regensburg RG, Klinkhamer S, van Adrichem NPH, et al. Micturation related swelling of the scrotum. Hernia. 2012;16:355–7.
6. Catalano O. Computed tomography findings in scrotal cystocele. Eur J Radiol. 1995;21:126–7.

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Hipertrofia lipomatosa do septo interatrial

Lipomatous hypertrophy of the interatrial septum

Sr. Editor,

Paciente do sexo feminino, 74 anos de idade, realizando rastreio para neoplasia devido a emagrecimento nos últimos seis meses, sem outras queixas associadas. Portadora de hipertensão arterial e diabetes mellitus, com bom controle medicamentoso. Durante a investigação foi demonstrado, na tomografia computadorizada (TC) de tórax, espessamento do septo interatrial (SIA) medindo 2,4 cm, devido a componente com densidade de gordura, poupando a fossa oval (Figuras 1A, 1B e 1C). A avaliação complementar por ecocardiograma transesofágico corroborou os achados prévios (Figura 1D). Com base em tais dados, foi confirmado o diagnóstico de hipertrofia lipomatosa do septo interatrial (HLSIA).