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Idiopathic Urinary Bladder Perforation in Early Childhood

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INTRODUCTION

Urinary ascites is a rare condition characterized by abdominal distention and renal impairment on biochemical investigation. Most often it occurs secondary to trauma, abdominal surgery, or after chemotherapy. In the neonate, it can occur either spontaneously or secondary to a urogenital malformation or umbilical catheterization. We describe a unique case of spontaneous urinary bladder perforation presenting with urinary ascites and an acute kidney injury picture in early childhood without underlying urogenital anomaly or trauma.

CASE PRESENTATION

A previously healthy 2.5-year-old boy presented to the emergency department with severe generalized abdominal pain, persistent vomiting, and decreased urine output. His preceding history was relevant for 5 days of dysuria and suprapubic abdominal pain, initially relieved with sitz baths. Physical examination revealed a mildly dehydrated, lethargic, and afebrile child with a heart rate of 128 bpm and blood pressure of 120/60 mm Hg. The abdomen was distended with demonstrable ascites and generalized tenderness without rebound, which was most severe in the suprapubic area. There was no skin rash or ecchymosis. Peripheries were warm and well perfused, with no edema.

Initial laboratory investigations (Table 1) suggested acute kidney injury. Complete blood count, Igs, antinuclear antibody, liver enzymes, and pancreatic enzymes were all normal, as well as complement serology (C3 and C4, 1.40 g/l and 0.3 g/l, respectively).

An abdominal X-ray showed centrally distributed bowel loops compatible with ascites, with no signs of obstruction or perforation, and abdominal ultrasound revealed anechoic ascites extending throughout the peritoneal cavity. The kidneys were of normal size bilaterally, with normal cortical echogenicity and corticomedullary differentiation. There was no dilatation of the collecting system or ureters. The bladder was normal. Color Doppler findings were normal.

The patient was maintained nil *per os*, received a bolus of 20 ml/kg of 0.9% saline solution, and was then fluid restricted to insensible fluid losses and urine output replacement. Potassium-lowering therapy was administered in the form of sodium polystyrene sulfonate (1 g/kg), salbutamol (2-mg nebulizer), and sodium bicarbonate (1 mmol/kg). After remaining anuric for 6 hours, the patient was catheterized with a Foley catheter inserted by the urology fellow without difficulty or trauma to the urethra. Of note, he had been catheterized with a Foley catheter for urine culture collection at the referring hospital without documented difficulty in catheterization. After catheter insertion, the urine was initially clear then became grossly bloody, with intermittent clearing. Urinalysis showed specific gravity 1.025, pH 6.0, protein 1.0 g/l, glucose negative, ketones 1.5 mmol/l, nitrite negative, and microscopic hematuria. A spot protein/creatinine ratio was elevated at 169.8 mg/mmol (normal range is $<$20 mg/mmol; nephrotic-range proteinuria is $>$200 mg/mmol).[1] Urine microscopy revealed numerous uniform red cells, with no red cell casts, compatible with a nonglomerular source.

Following catheterization, urine output as well as biochemical abnormalities (Table 1) improved dramatically, which coupled with results of urine microscopy and raised suspicion of a genitourinary anomaly. A voiding cystourethrogram (VCUG) was obtained and was in keeping with a bladder perforation, with extravasation of contrast from a point near the bladder dome (Figure 1). The Foley catheter was left *in situ* on straight drainage, and following the VCUG

Table 1. Serum biochemical markers at intervals from presentation to resolution

Serum parameter		Normal range	Presentation	Prior to Foley catheterization	After Foley catheterization	Prior to surgical repair	After surgical repair
Creatinine	(μmol/l)	<36	172	211	120	22	30
Urea	(mmol/l)	2.9–7.1	17.4	19.1	13.4	4	1.6
Na	(mmol/l)	135–143	139	140	142	137	138
K	(mmol/l)	3.7–5.0	5.8	5.7	4.1	4.9	4.2
Cl	(mmol/l)	99–111	107	105	109	103	105
Total CO_2	(mmol/l)	22–30	19	16	17	31	24
Albumin	(g/l)	32–56	47	48	38	25	26
Ca	(mmol/l)	2.17–2.65	2.38	2.46	2.29	2.24	2.17
Phosphate	(mmol/l)	1.16–2.10	2.56	2.65	2.03	1.69	1.82
Mg	(mmol/l)	0.70–0.95	1.06	1.09	0.98	0.95	0.79

confirmation of a urinary leak, a peritoneal drain was inserted by interventional radiology and was also left to straight drainage. Peritoneal fluid and serum creatinine were 164 μmol/l and 65 μmol/l, respectively, in keeping with the diagnosis of urinary ascites.

Although serum biochemistry returned to normal, significant output from the abdominal drain persisted for 1 week, suggesting an ongoing urine leak. The patient therefore underwent cystoscopy, which showed a normal anterior and posterior urethra, with both ureteric orifices in the normal orthotopic position. A large defect with some adjacent inflammatory reaction was seen along the right posterior wall of the bladder (Figure 2), confirming the diagnosis of bladder rupture. The 3-cm defect was repaired surgically, with cessation of the abdominal drainage. No biopsy of the bladder wall was performed at the time of the repair. After 3 months, a follow-up VCUG was performed and was normal (Figure 3).

DISCUSSION

Spontaneous urinary bladder perforation with urinary ascites in children is rare and, to our knowledge, has not been reported in a toddler. The reported causes of bladder perforation are listed in Table 2.

Despite vigorous investigation of our patient, no clear etiology was discovered. The active process of toilet training and a catheterization attempt at the referring hospital are possible risk factors, although the latter is less likely, given that there were no documented difficulties during the catheterization and there was no unusual pain or bleeding per the urethral meatus after the catheterization in the referring

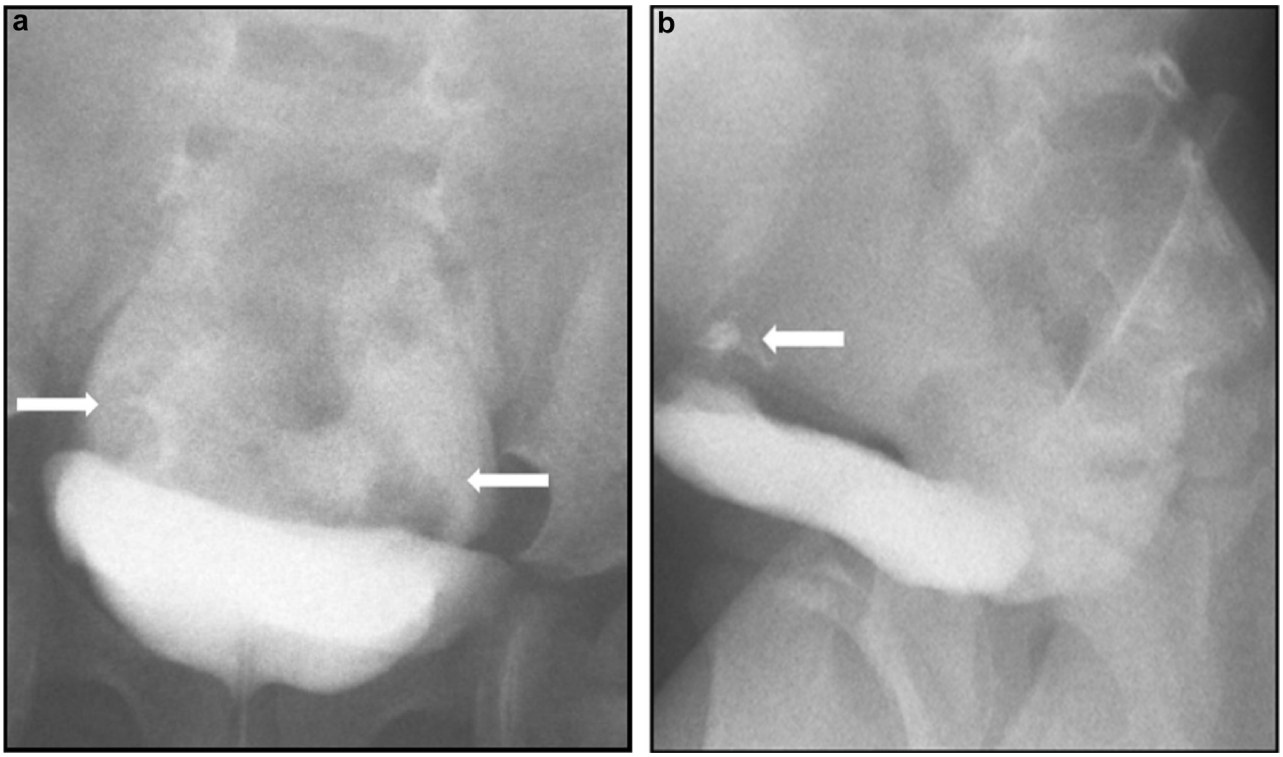

Figure 1. Voiding cystourethrogram (VCUG) images showing extravasation of the contrast from the urinary bladder (arrows). (a) Anterior–posterior view; (b) lateral view.

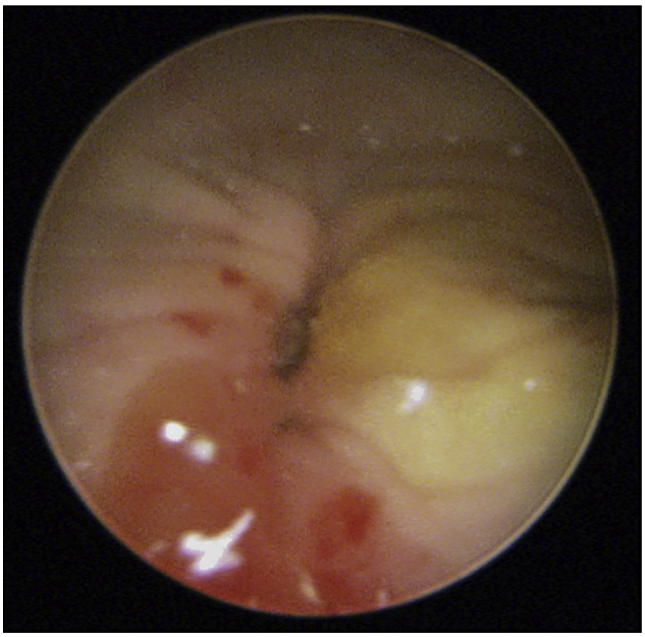

Figure 2. Cytoscopy showing i.p. bladder perforation.

hospital. The cause of the urinary bladder rupture in our patient is unknown; child abuse was also excluded, given no signs and symptoms suggestive of that. In our patient, no biopsy was performed on the margin of the defect; therfore, an inflammatory lesion cannot be ruled out.

Table 2. Causes of urinary bladder perforation

In neonates[3]	In children[7,8]	In adults[9,10]
• Umbilical artery catheterization[4] • Ruptured urachus • Prematurity/hypoxia[5,6]	• Blunt trauma to the abdomen • Enterocystoplasty • Indwelling catheters • Neurogenic bladder • Inflammation of the bladder wall • Obstruction at the bladder neck/urethra • Drugs, e.g., epirubicin chemotherapy	• Binge alcohol drinking • Pelvic irradiation • Obstruction at the bladder neck/urethra • Idiopathic

Although the urinary bladder is an extraperitoneal organ, i.p. perforation is more common in the adult population.[2] Given that a greater proportion of the bladder body and dome is still i.p. in children, i.p. perforation is even more common. In adults, abdominal tenderness and rigidity are the major presenting complaints,[2] but in children these may be easily masked until ascites develops.

To alleviate biochemical abnormalities, immediate intervention by urinary catheter insertion even in the absence of signs of bladder outlet obstruction is warranted, and may be solely adequate if the defect is extraperitoneal.

In neonates, a conservative approach is possible, although it should be used with care, as it may become

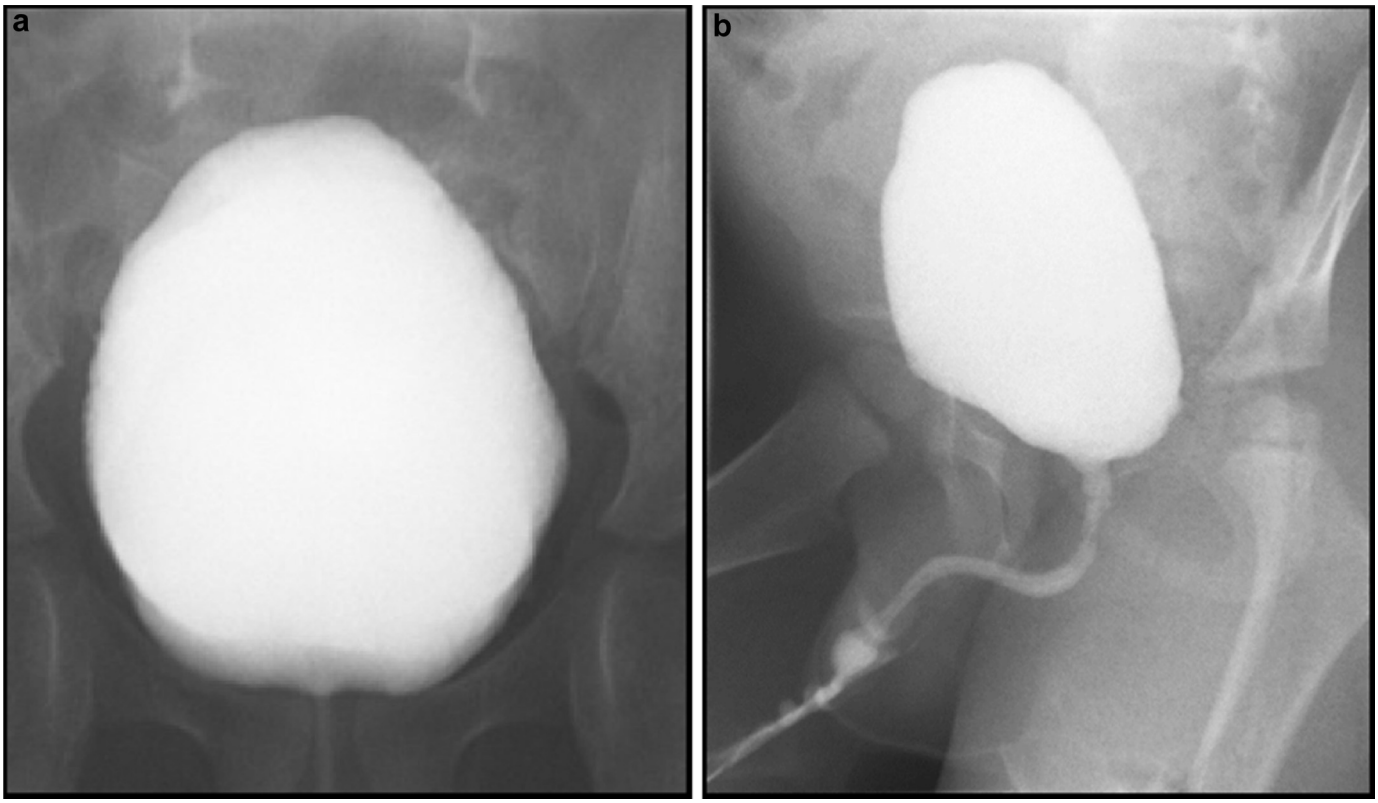

Figure 3. Follow-up cystourethrogram (VCUG) showing normal filling and resolution of extravasation. (a) Anterior–posterior view; (b) lateral view.

Table 3. Important steps in the management of idiopathic bladder perforation

1. Careful history and physical examination
2. Exclude possible causes of bladder rupture (listed in Table 2)
3. Early surgical consultation/VCUG
4. External and internal drainage
5. Surgical repair with or without bladder wall biopsy
6. Follow-up VCUG after surgical repair

VCUG, voiding cystourethrogram.

complicated by sepsis and in rare cases can cause death.[3]

In our patient, conservative management corrected the initial symptoms and biochemical abnormalities, but the defect failed to close on its own and required surgical correction. As such, in cases of spontaneous i.p. bladder rupture, we suggest early consideration for surgical consultation and repair when combined external and intravesicle drainage fails to achieve spontaneous closure. The important steps in the management of idiopathic bladder perforation are listed in Table 3.

In summary, we present a rare case of spontaneous i.p. bladder rupture in a toddler presenting as AKI. A careful history and physical examination, as well as a high index of suspicion, are required to prevent delayed diagnosis. Early surgical repair combined with biopsy of the margin of the defect is suggested in this age group, to prevent unnecessary intervention and complications as well as to diagnose inflammatory lesions as a possible risk factor for the bladder wall perforation.

DISCLOSURE

All the authors declared no competing interests.

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