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Case Report

Nontraumatic urinary bladder rupture presenting as renal pseudo-failure and ascites

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ABSTRACT

A 24-year-old man, with past medical history significant only for nocturnal enuresis until the age of 12 years, presented to the emergency department with acute abdominal pain after an episode of difficulty with micturition in the middle of the night. On presentation, physical examination was suggestive of ascites and laboratories revealed an elevated serum creatinine of 1.88 mg/dL. He was subsequently found to have a ruptured bladder, without any inciting trauma, which required surgical repair. His only surgical history is an unknown, apparently urologic, surgery when he was 11-12 years old. The patient's unique presentation prompts discussion of bladder rupture and its manifestations, the role of clinical information in informing imaging protocol, and the importance of sagittal images in identifying pathology.

Introduction

There are multiple reports in the medical literature regarding traumatic urinary bladder rupture as well as peripartum urinary retention with subsequent bladder rupture [1–4]. There are also several case reports and literature reviews of nontraumatic bladder rupture, but these cases are often associated with malignancy, radiation therapy, or other causes of obstructive pathology [5–8]. Previous reports of spontaneous bladder rupture without a known inciting event are rare—the most recent being in 2014 [9–14]. No matter the etiology, bladder rupture may present as renal pseudo-failure due to reabsorption of creatinine across the peritoneal membrane. Renal pseudo-failure can be defined as elevated creatinine with an etiology that does not fall under the traditional categories of prerenal, renal, or postrenal kidney injury. In renal pseudo-failure, there is no underperfusion of the kidney, no increased pressure in the glomerulus caused by obstruction, and no intrinsic disease of the kidney. Rather, the kidneys are functioning normally, but there is an increase in measured serum creatinine due to increased production or reabsorption.

Before presenting the case at hand, a brief review of nighttime urine incontinence in the pediatric population is prudent. Nocturnal enuresis can be thought of as primary if

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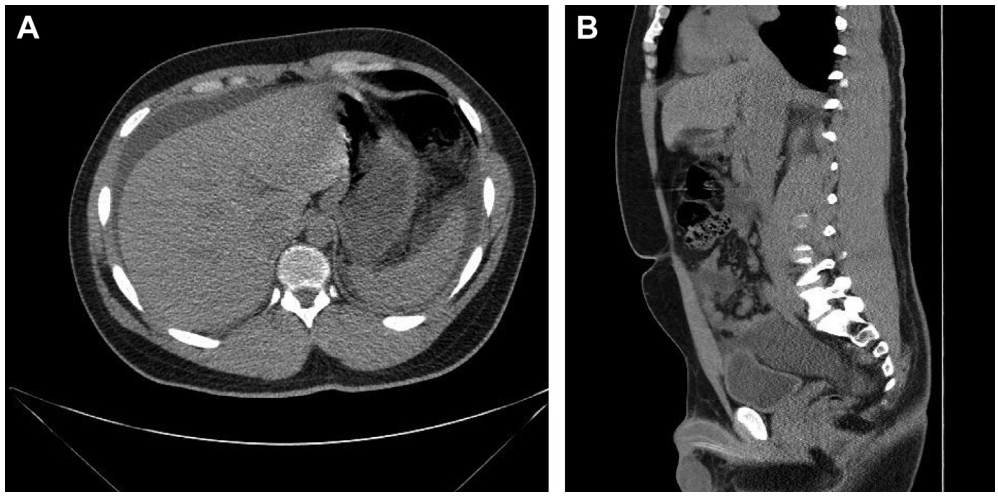

Fig. 1 – (A) Transverse slice of CT abdomen without contrast on day 1 showing free fluid collections around the liver and spleen. (B) Sagittal slice CT abdomen without contrast on day 1 notable for disruption of smooth demarcation of bladder wall in the dome of the bladder sagittal reconstruction showing findings consistent with bladder rupture. CT, computed tomography.

there has never been any period of nighttime continence or secondary if there has been a period of 6 months of dry nights followed by re-emergence of bed-wetting. At the age of 5 years, approximately 1 in 5 children will have primary nighttime incontinence that improves to roughly 1 in 100 children by the age of 17 years. Generally, the process is self-limited and thought to be due to delayed neurological maturation of micturition pathways. However, in patients with secondary enuresis or persistent daytime symptoms, there is increased prevalence of underlying organic or psychological pathology to include parental divorce, detrusor overactivity, antidiuretic hormone hyposecretion, or physiologic obstruction. If the diagnostic workup does not reveal any underlying abnormality, the cornerstone of therapy for enuresis includes educating the family about voiding habits, limiting afternoon and evening fluid intake, and bed-wetting alarms. Desmopressin, with or without oxybutynin, is the most common pharmacologic therapy but is of limited value given the high rate of symptom recurrence after cessation of therapy [15].

Case report

A 24-year-old healthy man, with past medical history significant only for primary nocturnal enuresis until the age of 12 years, presented to the emergency department with diffuse abdominal pain, nausea, vomiting limiting his oral intake, and difficulty with micturition. He reports waking up in the middle of the night before presentation and trying to urinate, after which he experienced sudden, intense abdominal pain that worsened over several hours prompting him to present to the emergency department. On presentation, he was noted to be agitated and was unable to find a comfortable position. Examination revealed suprapubic fullness and tenderness with associated mild abdominal distention and no other abnormalities noted. An indwelling urinary catheter was placed and approximately 1000 mL of nonbloody, translucent, yellow urine rapidly drained. The laboratory values on admission revealed apparent acute kidney injury with a creatinine of 1.88 mg/dL that resolved over the next 24 hours to 0.9 mg/dL with persistent indwelling urinary catheterization and intravenous fluids. A computerized tomography (CT) scan identified large-volume ascites (Fig. 1A). The day after admission, his abdominal pain had spontaneously resolved, laboratory abnormalities normalized, and the patient tolerated oral intake. The patient was discharged with plans for therapeutic paracentesis 2 days later as an outpatient.

The patient underwent paracentesis as scheduled. The procedure yielded 2000 mL of yellow, slightly turbid fluid. Further examination of the fluid revealed 378 WBC/μL and 113 RBC/μL. The serum-ascites albumin gradient value was greater than 3, consistent with a transudative or nonperitoneal cause of ascites. After paracentesis, he had minimal abdominal pain and was sent home.

Three days after paracentesis, he presented to the ED once again with recurrent abdominal pain similar in quality to that which prompted the first hospitalization. A comprehensive metabolic panel and complete blood count revealed a creatinine of 11.44 mg/dL, hemoglobin of 18.1 g/dL, and urinalysis was notable for 500 mg/dL of proteinuria. An indwelling urinary catheter was placed, promptly yielding 1400 mL of pale, yellow urine, and aggressive fluid resuscitation with normal saline at 200 mL/hour was initiated. His creatinine dropped precipitously to 6.43 mg/dL after 10 hours and then to 0.87 mg/dL after 36 hours. Hemoglobin dropped to 13.1 g/dL during this same interval. Twenty-four–hour urine collection demonstrated 212 mg/day of protein. At this point, a postrenal etiology for elevated creatinine was suspected and the patient underwent fluoroscopic cystourethrogram that revealed contrast moving outside the bladder into the peritoneal space (Fig. 2). He subsequently underwent laparotomic surgical repair of the ruptured bladder without complications. The surgeon noted that in addition to a small perforation in the dome of the bladder, which was repaired with 3 layers of