A Case Series of Spontaneous Rupture of the Urinary Bladder

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Key Words

Intraperitoneal bladder rupture • Bladder diverticulum • Spontaneous bladder rupture • Interposition omental flap • Extraperitoneal bladder rupture

Abstract

We report 2 cases of spontaneous bladder rupture related to chronic outlet obstruction and urinary retention. In both cases, focal perforation was identified within diverticula. Bladder rupture in the absence of trauma is a rare and serious event with a mortality rate approaching 50%. These injuries are often initially misdiagnosed and it is our goal to provide insight to the presentation, management and treatment of this rare event.

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Introduction

Spontaneous urinary bladder perforation is a rare and life-threatening condition making up less than 1% of all bladder injuries [1–9]. It is often initially misdiagnosed, even with the aid of CT imaging. There are several factors that can predispose patients to an event like this, but

most commonly it includes a chronic increase in bladder wall pressure and chronic inflammatory states. We believe this is the etiology in these 2 patients as well.

Case Report

The first patient is a 72-year-old African American male who arrived at the emergency room for a change in mental status. He complained of a 2-month history of progressively worsening abdominal pain associated with nausea and vomiting and exacerbated by coughing. His past medical history included hypertension, a previous cerebral vascular accident, and hyperlipidemia. He has no previous surgical history. Among antihypertensives, his medications also included oxybutynin. The patient had never seen an urologist before and was placed on this by his primary care physician for urgency and frequency after his cerebrovascular accident.

On presentation he was afebrile, normotensive and mild tachycardic at a heart rate of 115 bpm. He was peritonitic and general surgery was consulted. His labs showed a slightly elevated WBC count of $12.6 \times 10^3/\text{ml}$, a BUN of 108 mg/dl, creatinine of 12.9 mg/dl, and lactate of 57 mg/dl. His baseline creatinine was 0.8 mg/dl. A Foley catheter was placed and urinalysis was remarkable for microscopic hematuria with 93 RBC/HPF and 78 WBC/HPF.

A noncontrast CT scan revealed dilated loops of bowel with mesenteric edema and free fluid in the abdomen and pelvis. The bladder wall was thickened and the prostate gland unremarkable. He was brought to the operating room for an exploratory laparotomy with general surgery under the working diagnosis of mesenteric ischemia. Intraoperatively, the bladder was dusky and had focal areas of necrotic tissue overlying obvious diverticula. Urology was consulted and cystoscopy revealed an obstructing prostate,

Fig. 1. a Necrotic serosa on the patient's right side of the bladder dome. There is an accompanying bladder diverticulum on the left lateral wall of the bladder. **b** Quarter-sized hole on posterior aspect of bladder.

severe trabeculations and multiple diverticula. The serosa was extremely thin and friable and almost entirely detached from the surface of the bladder with a subserosal pocket filled with murky, purulent fluid. After debridement of the serosal layer a quarter-sized perforation was identified on the posterior bladder wall (fig. 1). The defect was closed in a 2-layer fashion and over which an omental flap was placed. The repair was tested to be watertight. An extensive washout was performed and JP drain was placed and a 16F Foley catheter for maximal decompression.

Postoperatively, the patient progressed well and was weaned off vasopressors within 24 hours. Interestingly, his intraoperative cultures resulted as negative, but he was started on antibiotics prior to the cultures being sent. His labs normalized; most notably his creatinine trended down to 0.8 mg/dl. When the patient was stable and able to provide more history, we discovered his baseline lower urinary tract symptoms included urgency, frequency, intermittent urge incontinence, weak stream and nocturia 3–4 times a night. His primary care physician had started him on oxybutinin after his cerebrovascular accident for the urgency and frequency. He was discharged on post-operative day 11 to a skilled nursing facility with the Foley catheter in place. A cystogram obtained 2 weeks after discharge was negative for a leak.

The second case is of a 65-year-old male who presented with 3 weeks of worsening abdominal pain, lethargy, nausea and vomiting, decreased oral intake and 1 week of decreased urine output. His past medical history included hypertension, hyperlipidemia, diabetes mellitus, and benign prostate hyperplasia last seen by

a urologist 6 months prior at which time he was placed on Si-lodosin for his lower urinary tract symptoms. However, he had not followed-up for reevaluation. On exam, he was afebrile, but tachycardic and hypotensive. He had moderate suprapubic tenderness, but was not peritonitic. His labs showed an elevated WBC of $36.9 \times 10^3/\text{ml}$, a BUN of 68 mg/dl and creatinine of 7.3 mg/dl. His baseline creatinine was 1.0 mg/dl 6 months prior. A CT scan obtained revealed a $5.4 \times 4.0 \times 5.1$ cm phlegmonous area superior to the bladder (fig. 2). General surgery was consulted and ultimately recommended a urology consult. Urology obtained a CT cystogram which revealed an intraperitoneal bladder rupture and he was taken to the operating room for exploratory laparotomy and cystorrhaphy. The delay from presentation to operating room was roughly 36 hours.

Intraoperatively, there was purulent intraperitoneal fluid and multiple adhesions between the bladder and overlying peritoneum. The bladder was necrotic and when distended multiple points of leakage were identified from diverticula on the posterior bladder wall. The bladder was bisected posteriorly and the necrotic tissue overlying the diverticula was debrided and the bladder was then closed in 2 layers for a watertight closure. The tissue overall was very friable and an omental flap were used for reinforcement of the repair. A JP drain and an 18F Foley catheter were placed.

His hospital course thereafter was complicated and protracted. He was taken back to the operating room several times for washouts and a redo repair of the bladder after the suture line had broken down. His sepsis was overwhelming and his family eventually withdrew care and he died on postoperative day 17.

Fig. 2. A coronal cut of CT demonstrating a phlegmonous area superior to the bladder.

Discussion

In this case series we describe 2 patients who presented with progressively worsening abdominal pain, sepsis and acute renal failure and were found to have spontaneous perforations of bladder diverticula. We suspect perforation resulted from chronic outlet obstruction and over distension which was complicated by an acute on chronic cystitis with severe inflammation within diverticula. Additionally, in the first case there the oxybutynin may have worsened the retention while his obstructive symptoms went untreated.

Signs and symptoms of spontaneous bladder rupture can be non-specific and are commonly misleading, often delaying diagnosis and treatment. Most patients present

with an acute abdomen and complain of lower abdominal pain, dysuria, difficulty voiding, hematuria and sometimes inability to void [9]. Patients can also present with acute renal failure and sepsis as did in these patients. These patients are often initially misdiagnosed; according to Mallick et al. [9] an accurate initial diagnosis of urinary bladder rupture was made in 2 out of 15 case reports between 1967 and 2007. The correct diagnosis is usually made at time of discovery in the operating room with general surgery as happened in our first case.

Our 2 patients both had evidence of chronic outlet obstruction in the form of severe bladder wall trabeculations and multiple diverticula. We believe that chronic urinary stasis and inflammation in the diverticula combined with high pressure voiding contributed to spontaneous rupture. Bladder diverticula are herniations of the bladder urothelium through the muscularis propria of the wall and represent a significant point of weakness in the bladder [10]. They are essentially non-functioning outpouchings lined with only a thin layer of serosa and adventitia and this inherent weakness places patients who have poorly controlled outlet obstruction at risk for perforation.

Conclusion

Spontaneous bladder perforation is a rare cause of an acute abdomen, but must be kept on the differential diagnosis in a patient with risk factors for dysfunctional voiding. One should suspect it in a patient presenting with an acute abdomen, acute renal failure, hematuria, urinary retention, oliguria or anuria in patient with risk factors for dysfunctional voiding. We hope this case report successfully heightens awareness of spontaneous bladder ruptures so that they can be detected early and managed appropriately to give these patients the best possible outcomes.

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