Trauma & Reconstruction

Spontaneous Extraperitoneal Bladder Rupture Because of Chronic Appendicitis

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Abstract

A 62-year-old man presented to the emergency department with an episode of syncope after 2-3 weeks of diffuse abdominal pain, now complaining of a severe increase in pain concurrent with >24 hours of no urine output. His workup showed an idiopathic extraperitoneal rupture of the bladder on computed tomography, which was handled conservatively with Foley insertion. Repeated follow-up and imaging showed no resolution or etiology over 2 months. The patient underwent exploratory laparotomy that showed an elongated appendix with a chronic tip appendicitis that had induced bladder rupture by chronic inflammatory changes. After repair, the patient had no further complaints.

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Introduction

This case reports an acute spontaneous bladder perforation secondary to a chronic inflammatory reaction, likely caused by an adjacent tip appendicitis that was previously undiagnosed. Bladder rupture is a relatively rare urologic complaint that is generally associated with trauma with or without concurrent retention and rarely associated with intrinsic processes. Approximately 82% of all bladder ruptures occur secondary to trauma, with the remaining portion nearly entirely categorized as idiopathic and a rate of confirmed spontaneous bladder rupture of <1%. This case will show a unique etiology for spontaneous bladder rupture, by way of a chronic inflammatory process with possible vesicoenteric fistula.

Case presentation

A 62-year-old white man presented to the emergency department after 1 episode of syncope along with a sharp increase in abdominal pain for 3 days, after 2-3 weeks of vague abdominal pain. The patient additionally complained of diarrhea, subjective fevers, and 24 hours of no urine output. Past medical history included coronary artery disease, atrial fibrillation, hypertension, hyperlipidemia, and benign prostatic hyperplasia with recent benign biopsy. The patient has a child who has severe Crohn's disease, but no other pertinent family history. Physical examination showed a diffusely tender and distended abdomen without any positive diagnostic signs. A Foley was placed to manage the retention in the emergency department. Noncontrast computed tomography (CT) imaging study performed in the emergency department showed a fluid collection superior to the bladder with no gastrointestinal abnormalities, and a repeat study with 450 mL of dilute cystogram in the bladder confirmed a contained extraluminal collection of contrast superior to the bladder identified as an extraperitoneal bladder rupture (Fig. 1). No contrast was seen intraperitoneally. The patient was positive for sepsis with episodes of hypotension, had rapid ventricular response atrial fibrillation at presentation, and cultures would later show corynebacterium in the blood. With this presentation, the patient was not a surgical candidate, and conservative management of the idiopathic bladder rupture by Foley maintenance and antibiotics was planned.

Ten days after admission, the patient received a voiding cystourethrogram with fluoroscopic imaging (Fig. 2). The study showed a large contained region of extravasation through a persistent superior bladder defect. It was decided to continue Foley maintenance and repeat the studies to evaluate and further elucidate a cause. Subsequently, a CT study demonstrated an increase in size of the collection with no other abnormalities. Twelve days after admission, the patient’s medical conditions except for the idiopathic bladder rupture had resolved, and he was discharged. The patient returned to our facility 4 weeks later for a CT cystogram, which showed continued persistence of extraperitoneal bladder rupture with a contained superior collection. At this point, surgical options were considered and discussed with the patient.

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Ten days after the latter CT cystogram, the patient returned to the hospital emergency department for 2-3 days of severe abdominal pain, diarrhea, and episodes of delirium with disorientation. Patient found to be septic again, with acute kidney injury. A barium enema was performed on hospital day 1, but there were no abnormal findings. On hospital day 7, the patient was deemed stable enough to undergo surgery, and exploratory laparotomy to evaluate the bladder was performed. Three hundred sixty-degree cystoscopy failed to reveal any lesions, and the laparotomy was started with the bladder fully distended. On exposing the posterior aspect of the bladder, a knuckle of intestinal tissue was found adherent to the bladder at the area of the suspected bladder rupture. Blunt and sharp dissection revealed a ruptured appendix adherent to the dome of the bladder within a larger phlegmon containing urine. General surgery was consulted intraoperatively and performed an appendectomy (Fig. 3). Frozen specimens of the bladder tissue and the appendix revealed no malignant processes, but showed chronic histiocytic inflammatory changes at the appendiceal tip and the bladder wall. The abscess was washed out, no further leakage confirmed by methylene blue injection, and the bladder closed with a suprapubic tube inserted.
The patient was discharged from the hospital 3 days later. At 2-month follow-up, the patient had no further complaints, had resolution of the extraperitoneal collection, and underwent an elective transurethral resection to manage his benign prostatic hyperplasia. Final diagnosis was an extraperitoneal bladder rupture after an episode of retention caused by chronic irritation from a missed tip appendicitis adjacent to the bladder.

Discussion

Spontaneous extraperitoneal bladder rupture is a rare occurrence, which requires a more complete workup than the significantly more common traumatic extraperitoneal bladder rupture. In spontaneous bladder rupture, as in our case, there can be a persistent cause of the rupture that must be addressed before rupture resolution and the common conservative 7-to-10-day approach will not prove effective in many of these cases. Published data on the subject has shown acute appendicitis as etiology for concurrent ruptures but our subject presented with a chronic appendicitis of approximately 3 weeks duration, without many of the diagnostic signs of appendicitis, and with a strong suspicion of the bladder rupturing weeks after the appendicitis. Additionally, owing to the inflammation being confined to the tip, multiple imaging studies noted the appendix to be normal. There are at least 2 reports of an anatomic variant of the gastrointestinal system leading to a vesicoenteric fistula but our case did not develop from an anatomic variant and the formation of a true fistula is questionable given no contrast extravasated to the colon or proximal appendix beyond the phlegmon. It is believed that this might represent the first reported case of a neighboring chronic irritant causing a delayed spontaneous bladder rupture. The bladder biopsy showed chronic histiocytic inflammatory changes throughout the wall, which lead to a local weakening in the area touching the phlegmon, and it is presumed that the inciting event for the rupture was acute retention also caused by the adjacent infectious process. This case stands as example of chronic inflammatory disease, including disease of adjacent nonurologic tissue, as a primary etiology of bladder rupture.

Conclusion

Spontaneous bladder rupture is possible in the setting of an adjacent chronic inflammatory process. Such a situation should be in the differential diagnosis of any nonhealing unexplained bladder rupture.

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