Case Report

Conservatively managed spontaneous intraperitoneal bladder perforation in a patient with chronic bladder outflow obstruction

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Abstract

We present the unusual case of a spontaneous intraperitoneal bladder rupture as a first presentation of chronic bladder outflow obstruction secondary to benign prostatic hyperplasia. A contributing factor to diagnostic delay was unfamiliarity with the classical presentation of abdominal pain, abdominal distension and urinary ascites leading to autodialysis represented by an unusually high serum creatinine. A cystogram was performed after a non-contrast computed tomography (CT) scan originally performed to determine the cause of abdominal pain, failed to confirm the diagnosis. The patient's initial acute presentation was successfully managed conservatively with prolonged urinary catheterization.

Key Words: Benign prostatic hyperplasia, Bladder, intraperitoneal, perforation, prostate

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INTRODUCTION

Chronic bladder outflow obstruction can remain asymptomatic for a significant period of time, and may present acutely in numerous ways including as a painless or painful abdominal mass, urinary retention or with complications associated with severe renal impairment. Atraumatic spontaneous bladder rupture is a poorly considered consequence of chronic bladder outflow obstruction which can lead to diagnostic delays as illustrated in our case presentation

CASE REPORT

A 66-year-old man presented acutely to hospital with a week's history of abdominal pain, 'coffee ground' vomiting and progressively worsening malaise with signs of dehydration. His symptoms commenced with a sudden onset of severe abdominal pain followed by the inability to pass urine. There was no history of abdominal trauma. He was a smoker with a previous history of alcoholism, however, had been abstinent now for several years. He had no Urological history.

On examination, he was significantly dehydrated with a moderately distended generally tender abdomen with a moderate degree of peritonism. No other specific signs were elicited.

The patient was resuscitated with crystalloid fluid and bloods were sent for analysis. Noted abnormalities of his blood results included a C-reactive protein of 107, a creatinine of 812 mmol/l and potassium of 5.9 mmol/l. Amylase and hemoglobin were within normal range.

A non-contrast CT (NCCT) scan was arranged in view of the patient's significant renal impairment; images revealed a

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moderate amount of ascites, a thickened but collapsed bladder, and a small cirrhotic liver with no evidence of hydronephrosis or pneumoperitoneum [Figure 1]. He was admitted under the general medical team for further investigations including upper gastrointestinal endoscopy which demonstrated signs of esophagitis, gastritis and a chronic duodenal ulcer in the first part of the duodenum.

His serum creatinine normalized over the course of a few days and a subsequent ultrasound scan that had been arranged to exclude portal venous hypertension, appeared to show that the ascites had resolved. A week after admission, the patient failed his trial without catheter, with a painful 200 mls clear urine residual on re-catheterization and a subsequent rise in creatinine to 200 mmol/l. On direct questioning, the patient admitted that he had suffered from urinary frequency and nocturia with progressively poor flow for the last year. Digital rectal examination revealed an enlarged benign-feeling prostate, hence an alpha-blocker (Tamsulosin 400 mcg) was commenced. A second trial without catheter several days later, resulted in a similar outcome, hence a referral to Urology was made.

His admission NCCT images were reviewed once more with the radiologists, who believed that the integrity of the bladder was sound [Figure 2]. However, due to remaining clinical uncertainties, a retrograde cystogram was performed which clearly demonstrated an intraperitoneal bladder perforation [Figure 3] and this was confirmed on subsequent CT [Figure 4].

The patient had remained clinically well during his hospital admission of nearly two weeks whilst catheterized and his creatinine had normalized. He was discharged home with a urethral catheter and underwent a trial of voiding 12 weeks later, subsequently resulting in failure to void adequately despite continuation of medical therapy and a repeat retrograde cystogram which demonstrated no further leak.

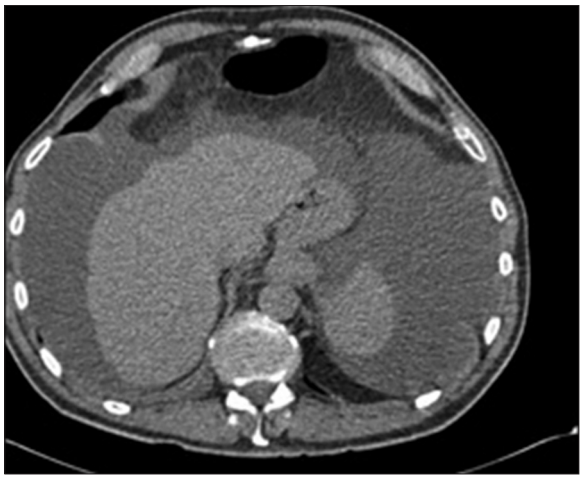

Figure 1: Axial slice of a NCCT illustrating a small cirrhotic liver and a moderate amount of ascites; the latter originally thought to be due to alcoholic liver disease

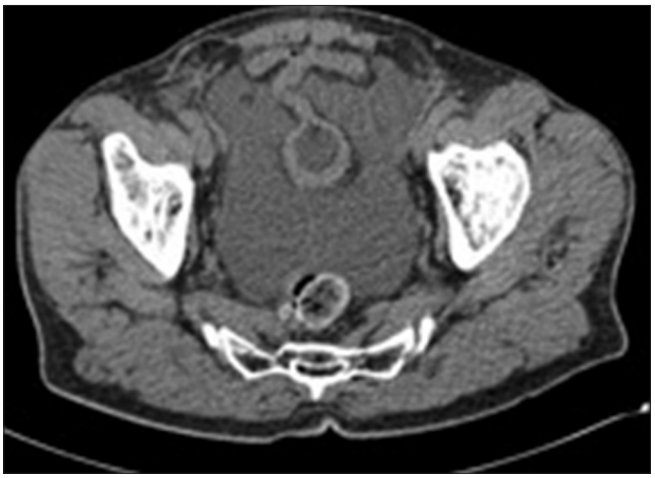

Figure 2: Axial slices of a NCCT showing an abnormally collapsed bladder with a thickened wall

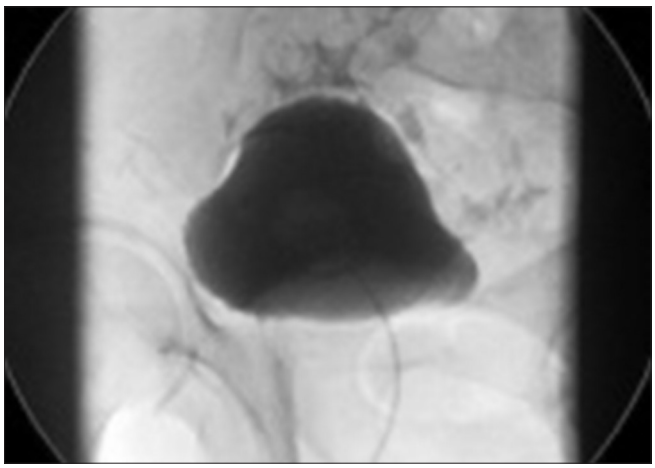

Figure 3: A retrograde cystogram revealing leakage of contrast into the peritoneum confirming an intraperitoneal bladder rupture

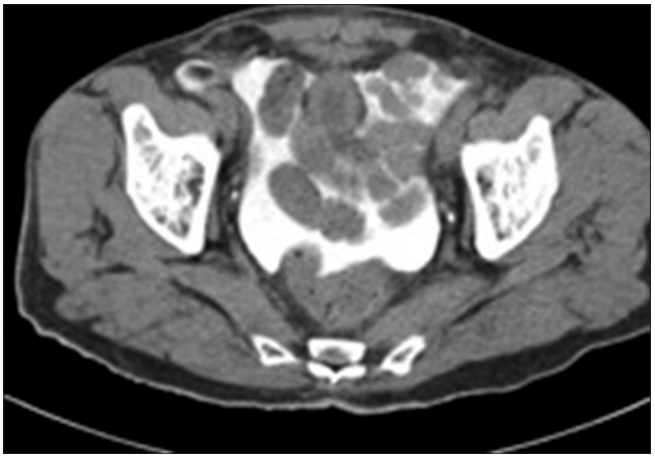

Figure 4: Subsequent CT illustrating the same contrast instilled during the cystogram in Figure 3, which remains intraperitoneal

A flexible cystoscopy performed revealed no significant bladder abnormalities, however, revealed a moderately occlusive prostate. An elective Transurethral Resection of the Prostate (TURP) was arranged with a satisfactory outcome.

DISCUSSION

Compared to traumatic perforation, spontaneous rupture of the urinary bladder is extremely rare, and has been reported to be associated with pre-existing bladder pathology[1-3] which alters the integrity of the detrusor muscle, for example radiotherapy, malignancy or previous surgery.[1-3] Bladder rupture tends to be intraperitoneal rather than extraperitoneal because the dome and posterior walls are less well supported.[4]

There are few cases reported in the literature relating to patients with chronic urinary retention presenting with bladder perforation. The likely etiology in these patients is a perforated bladder diverticulum; an intrinsic weakness in the detrusor secondary to chronic bladder outflow obstruction. In this particular case, the cause was due to benign prostatic hyperplasia.

The delay to diagnosis is multifactorial; direct urethral catheterization may not always provide an accurate residual due to the presence of urinary ascites. Standard CT reveals the presence, but cannot determine the cause of ascites or accurately evaluate the bladder wall. Retrograde cystography or a CT cystogram is the gold standard investigation for diagnosing bladder perforation, involving the use of a sufficient amount (>300 ml) of contrast and intravesical pressure to demonstrate the migration of contrast into the peritoneal cavity.

Serum creatinine will be significantly elevated due to peritoneal autodialysis of urinary ascites, in combination with profound hypovolemia. An ascitic tap would demonstrate significantly elevated creatinine levels compared to that of the serum if urinary ascites were present.

It is important for the clinician to consider bladder rupture as a differential diagnosis in patients presenting with acute abdominal pain with an inability to pass urine, and a low-volume (<500 mls) residual. It is likely that both the filling and micturition phase can cause an increase in intravesical pressure, allowing urine to reflux through the injury into the peritoneal cavity, causing severe pain and consequentially a raised serum creatinine. Another hallmark feature of this diagnosis is the resolution of ascites on imaging after urinary catheterization.[5]

In the vast majority of cases, acute intraperitoneal bladder rupture is managed surgically due to the accumulation of urea within the peritoneal cavity causing peritonitis. However, as illustrated in our case, a delayed presentation can be managed conservatively with prolonged urinary catheterization allowing bladder decompression, if the patient remains clinically stable.

CONCLUSION

This case illustrates an uncommon complication of chronic urinary retention secondary to bladder outflow obstruction. Atraumatic spontaneous bladder rupture can occur in patients with pre-existing bladder pathology or patients with chronic bladder outflow obstruction. It is therefore essential to take note of the pre-morbid history of lower urinary tract symptoms in these patients, which may overcome any potential delays to diagnosis.

Contrary to the usual surgical management for intraperitoneal bladder rupture, we have demonstrated that these cases can be managed conservatively in the acute phase without significant complication. However, we would strongly recommend close follow-up of these patients due to the risk of developing chronic urinary retention with the necessity for further urodynamic studies or bladder outflow surgery.

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