Jpn J Radiol (2012) 30:284–287
DOI 10.1007/s11604-011-0042-7

CASE REPORT

CT findings of spontaneous intraperitoneal rupture of the urinary bladder: two case reports

Junichi Izumi · Hiroko Hirano · Takeshi Kato · Takuo Ito · Koji Kinoshita · Toshiki Wakabayashi

Received: 28 July 2011 / Accepted: 5 December 2011 / Published online: 7 January 2012

Abstract We report two cases of spontaneous bladder rupture. Preoperative diagnosis was difficult and the correct diagnosis was made at surgery. Reviewing the initial abdominopelvic CT of our second patient, the bladder wall defect and blood attenuation near the bladder were observed. These findings were consistent with the operative findings, and would have led to correct preoperative diagnosis if we had had sufficient knowledge of spontaneous bladder rupture. Under urinary catheterization, ascites and free intraperitoneal air were identified in both patients. These findings were indistinguishable from those for bowel perforation, which was our preoperative diagnosis. Significant changes in ascites volume between pre and post urinary catheterization can be an indication of spontaneous bladder rupture.

Keywords Spontaneous bladder rupture · CT

J. Izumi (✉) · H. Hirano
Department of Radiology, Yokote Municipal Hospital, 5-31 Negishicho, Yokote 013-8602, Japan
e-mail: j_izumi@yokote.mhp.jp

T. Kato
Department of Surgery, Yokote Municipal Hospital, Yokote, Japan

T. Ito
Department of Urology, Yokote Municipal Hospital, Yokote, Japan

K. Kinoshita
Department of Internal Medicine, Yokote Municipal Hospital, Yokote, Japan

T. Wakabayashi
Department of Surgery, Akita City Hospital, Akita, Japan

Introduction

Spontaneous rupture of the urinary bladder is a rare and potentially life-threatening event [1]. Herein, we present two case reports of unsuspected urinary bladder rupture that was diagnosed only through surgery. Preoperative CT images were reviewed and diagnostic findings are discussed.

Case report

Case 1

An 87-year-old woman who had had an indwelling Foley catheter for approximately 7 months because of chronic urinary retention, came to the emergency room with hypoactivity and oliguria. On examination, she was afebrile (36.6°C), with blood pressure of 80/50 mmHg and pulse of 80 beats per minute (bpm); her oxygen saturation was 90% on room air. Laboratory tests revealed a white blood cell count of 13,400/mm^3, serum C-reactive protein (CRP) of 10.9 mg/dL, blood urea nitrogen (BUN) of 30.1 mg/dL, serum creatinine of 0.8 mg/dL, and proteinuria and hematuria. A non-contrast abdominopelvic CT revealed free intraperitoneal air and a small amount of ascites (Fig. 1). On the basis of these CT findings, diagnosis of peritonitis because of gastrointestinal perforation was made.

At laparotomy, the abdominal cavity contained purulent fluid in both the subphrenic space and the pouch of Douglas. A pin-hole perforation was identified in the left side of the urinary bladder, and partial resection of the bladder was performed. On histopathological examination, muscle layer defect with fibrosis and hemosiderin

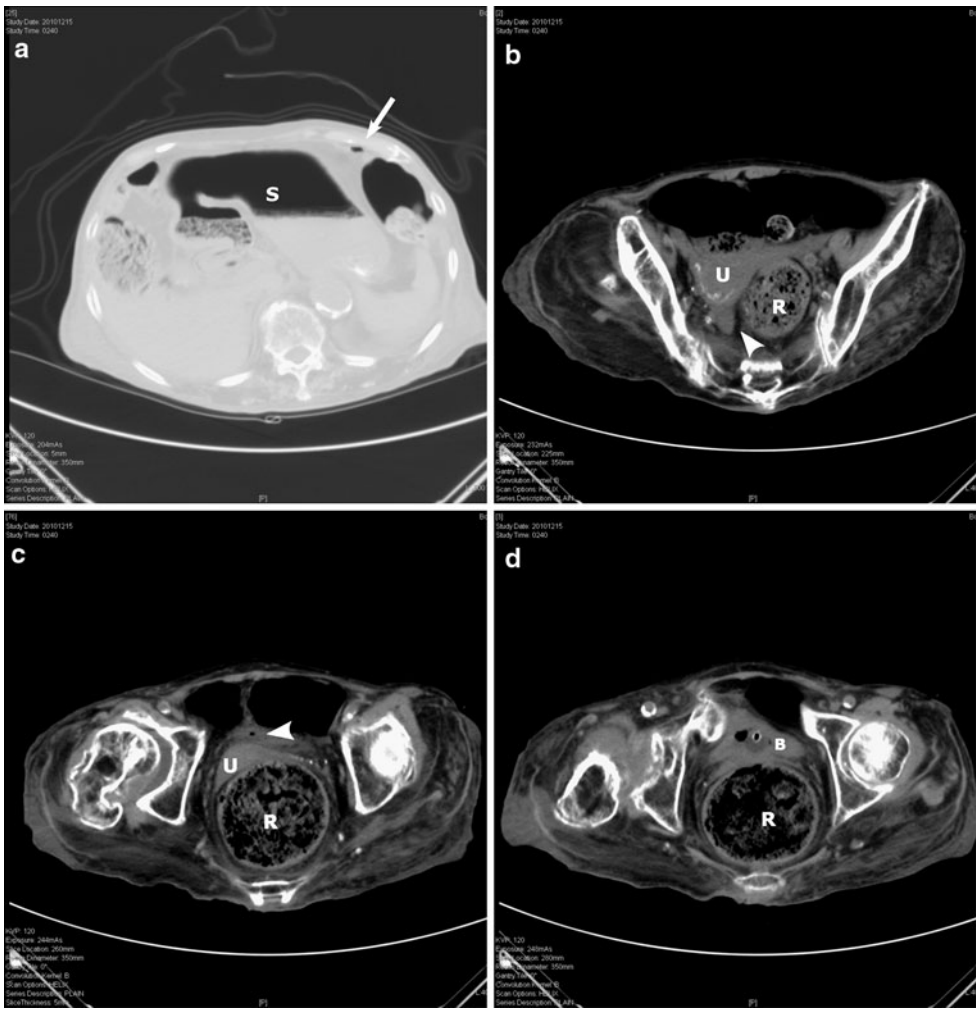

Fig. 1 An 87-year-old woman with spontaneous urinary bladder rupture. **a** Non-contrast CT at the upper abdominal level shows a tiny amount of free intraperitoneal air (*arrow*) with a lung window setting. **b** At the pelvic level, a small amount of ascites (*arrowhead*) is identified in the pouch of Douglas. **c** Free intraperitoneal air (*arrowhead*) is also seen at a level just above the bladder. Although this finding is indicative of bladder wall rupture, the origin of the air was not confirmed preoperatively. **d** A urinary catheter is inserted. Neither blood attenuation nor urinary bladder wall defect is identified. *B* Bladder, *R* rectum, *S* stomach, *U* uterus

deposition was identified in the resected bladder wall. No malignancy was noted. We reviewed the abdominopelvic CT and found free intraperitoneal air near the bladder (Fig. 1c). However, the origin of the air was not determinable. Although her clinical condition gradually improved, she died of complications of pneumonia on the 41st postoperative day.

Case 2

A 56-year-old man, who had a drinking habit, presented to our hospital with clinical symptoms of appetite loss, nausea, vomiting, abdominal pain, hematuria, and oliguria of 2 days' duration. Laboratory data were indicative of renal dysfunction and inflammation, and ultrasonography identified ascites. He was treated with fluid replacement, with diagnoses of acute renal impairment and dehydration.

The next day, he was admitted to our hospital because of deteriorating his renal function. Pulse rate was 76 bpm, blood pressure 163/111 mmHg, and his temperature was 36.7°C. Clinical examination revealed tenderness in the lower abdomen and abdominal distention. Laboratory tests revealed: white blood cell count 101,600/mm^3, hemoglobin 13.5 g/dL, BUN 34.2 mg/dL, serum creatinine 5.2 mg/dL, and CRP 17.6 mg/dL. A non-contrast abdominopelvic CT revealed massive ascites, and dilated small bowel which indicated paralytic ileus because of peritonitis (Fig. 2a–c). An ileus tube insertion and a Foley catheterization were performed, and the urine obtained from the catheter was bloody. Abdominal paracentesis yielded cloudy, red colored peritoneal fluid. Biochemical study of the ascites showed the specific gravity was 1.012, protein was 1.3 g/dL, glucose was 112 mg/dL, and cell count was 5,212/μL. Bacterial culture of the ascites was negative, and no malignant cells were identified in the ascitic fluid. Electrolytes in the ascites were not measured.

On the second day of admission, the total volume of urine reached 3,600 mL, and his renal function improved rapidly. Contrast-enhanced CT was then performed for re-evaluation of intra-abdominal pathology. The CT revealed a significant decrease in ascites volume and free intraperitoneal air (Fig. 2d). Although the rapid change of

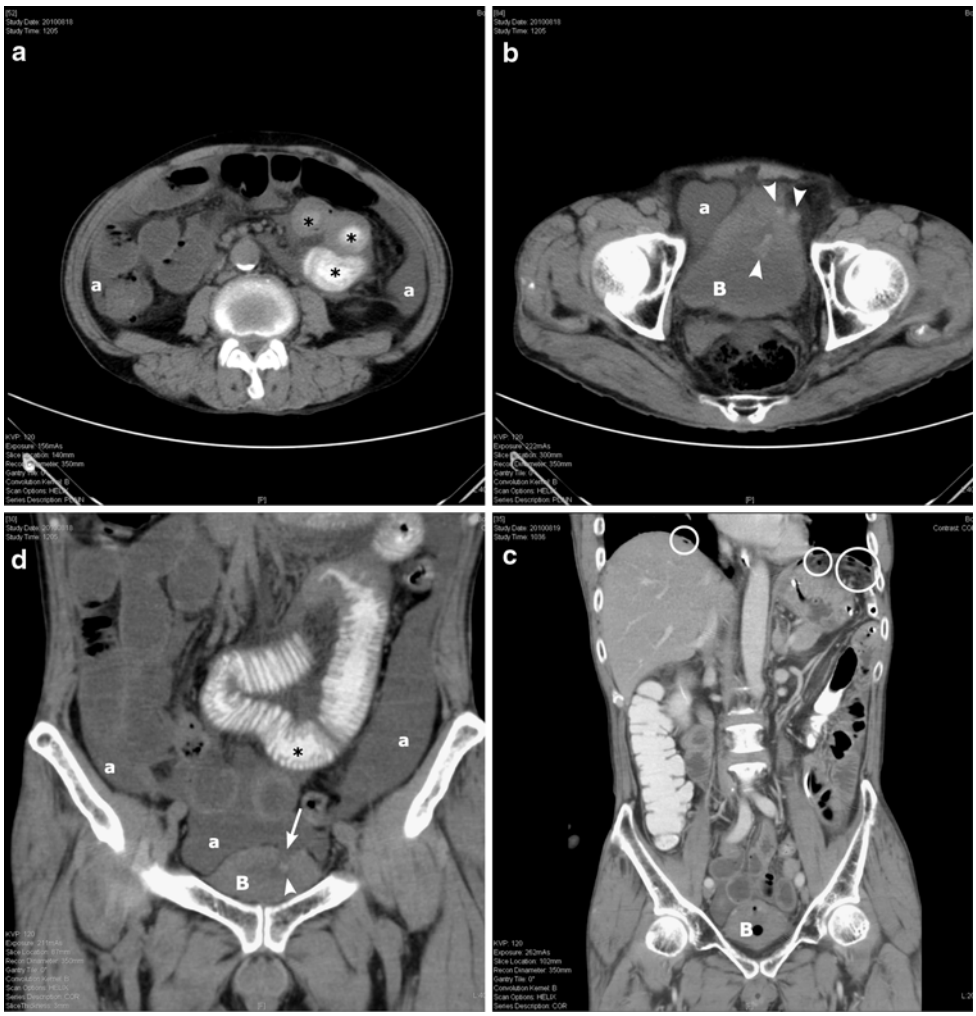

Fig. 2 A 56-year-old man with spontaneous urinary bladder rupture. **a** Non-contrast abdominopelvic CT reveals ascites (*a*) and paralytic ileus. Orally ingested gastrografin is observed (*asterisks*). **b** At the urinary bladder (*B*) level, small high-attenuation foci (*arrowheads*) indicative of fresh blood clots are identified intra and extra-bladder. **c** Reconstructed coronal image shows the urinary bladder defect (*arrow*) and the small clot in the bladder (*arrowhead*). Ascites and paralytic ileus are also observed. **d** After urinary catheterization, contrast-enhanced CT performed on the 2nd hospital day with a coronal reconstruction shows a significant decrease of ascites and free intraperitoneal air (*circles*)

ascites volume was not understood, a provisional diagnosis of bowel perforation was made.

Laparoscopic examination revealed a medium amount of hemorrhagic ascites and a 2 in long bladder wall laceration. The bladder lesion was resected partially and sutured at laparotomy. Histopathological evaluation revealed no evidence of malignancy in the resected bladder wall. We performed a retrospective review of the initial non-contrast-enhanced CT images, and discontinuity of the bladder wall and relatively high attenuation near the bladder abnormality were found (Fig. 2b, c). These findings were compatible with the operative findings. The patient's condition improved after surgery, and he was discharged on the 25th postoperative day.

Discussion

Spontaneous rupture of the urinary bladder is rare. The causes of rupture of the urinary bladder include irradiation therapy for the pelvis, urinary bladder tumor, bladder stones, in postpartum female, bladder infections (tuberculosis, syphilis), bladder outflow obstruction (prostate hypertrophy, urothelial stricture), binge alcohol intake, neurogenic bladder, and urethral or suprapubic catheterization. In the literature, two different types of urinary bladder rupture have been documented, intraperitoneal and extraperitoneal [2]. In most cases, nontraumatic bladder rupture is intraperitoneal. Evans et al. [3] demonstrated through cadaveric study that the peritoneal portion of the urinary bladder, which is unsupported and expansile, is the least resistant to an increase of intravesical pressure.

Common symptoms are severe abdominal pain, nausea and vomiting, rigidity and tenderness, and difficulty or inability to void. There might be macroscopic hematuria. Results from biochemical analyses of ascites may be useful indicators of the presence of urine in the peritoneal cavity [4]. Laboratory examination constantly shows spontaneous intraperitoneal bladder rupture as oliguric acute renal failure, as shown in our second patient. Significant reabsorption of urea and creatinine through the peritoneum leads to significant elevation of blood urea and creatinine

levels [5]. Because transurethral catheterization can drain much urine (urinary ascites) and normalize serum creatinine level [6], urinary catheter insertion was an appropriate procedure for our second patient. In most reported cases, surgical intervention was performed for spontaneous bladder rupture. However, conservative management with urinary catheter has been applied in some reports [7].

According to a study by Shin et al. [8], in patients with traumatic bladder rupture, a relatively highly attenuating and heterogeneous fluid near the bladder (sentinel clot sign) is a useful finding indicative of urinary bladder rupture. In our second patient, this sentinel clot sign was observed after retrospective review of the initial non-contrast CT. The bladder wall defect was also revealed on the coronal reconstructed images. These findings were consistent with the surgical findings, and could have led to the correct preoperative diagnosis if we had had sufficient knowledge of spontaneous bladder rupture.

Under urinary catheterization, ascites and free intraperitoneal air were observed in both patients. These findings are indistinguishable from those of bowel perforation. Even after review, identification of bladder rupture in our first patient was quite difficult. Free intraperitoneal air and a small amount of ascites were observed, but signs of either a clot or a bladder wall defect were not observed. Although free air was present near the urinary bladder, its origin was not determinable. In our second patient, a significant change in the ascites volume between pre and post urinary catheterization was interesting and reasonable because of the result of fluid excretion through the bladder wall defect and the urinary catheter. Dramatic change in the ascites volume after urinary catheterization can be indicative of spontaneous urinary bladder rupture.

For detecting bladder rupture, basic intravenous contrast-enhanced CT might not be beneficial compared with non-contrast CT. Even contrast-enhanced CT with an excretory phase, which is slightly time-consuming, will not necessarily lead to detection of a small bladder rupture, because a blood clot or omentum may temporarily seal a small perforation. CT retrograde cystography is the most satisfactory radiological procedure. Chan and coworkers [9] described the diagnostic usefulness of CT cystography for patients with traumatic bladder rupture. The authors reported that the sensitivity, specificity, positive predictive value, and negative predictive value of CT cystography for detection of intraperitoneal bladder rupture were 100, 99.6, 85.7, and 100%, respectively. CT cystography might be beneficial for detection of bladder perforation even in cases of spontaneous rupture. CT cystography, however, might not be performed for nontraumatic patients, because suspicion of bladder perforation is unlikely.

Conclusion

Spontaneous intraperitoneal bladder rupture is rare but should be included in differential diagnosis when CT shows ascites and blood attenuation near the bladder, especially when such findings occur in patients with oliguria or anuria, renal dysfunction, and lower abdominal pain and tenderness. Radiologists should realize that CT findings mimicking bowel perforation can be seen in patients with spontaneous bladder rupture under urinary catheterization.

References

1. Labanaris AP, Zugor V, Nützel RR, Kühn R. Spontaneous rupture of the urinary bladder 10 years after curative radiotherapy. Sci World J. 2008;8:405–8.
2. Marbury WB, Fry W. Spontaneous rupture of the urinary bladder: report of case of second rupture. Ann Surg. 1943;117:760–5.
3. Evans RA, Reece RW, Smith MJ. Idiopathic rupture of the bladder. J Urol. 1976;116:565–7.
4. Wystrychowski A, Nowicki M, Kokot F. Hyponatraemic renal pseudofailure—don't forget the possibility of uroperitoneum. Nephrol Dial Transpl. 1996;11:2491–2.
5. Heyns CF, Rimington PD. Intraperitoneal rupture of the bladder causing the biochemical features of renal failure. Br J Urol. 1987;60:217–22.
6. Kato A, Yoshida K, Tsuru N, Ushiyama T, Suzuki K, Ozono S, et al. Spontaneous rupture of the urinary bladder presenting as oliguric acute renal failure. Intern Med. 2006;45:815–8.
7. Basiri A, Radfar MH. Conservative management of early bladder rupture after postopera-tive radiotherapy for prostate cancer. Urol J. 2008;5:269–71.
8. Shin SS, Jeong YY, Chung TW, Yoon W, Kang HK, Kang TW, et al. The sentinel clot sign: a useful CT finding for the evaluation of intraperitoneal bladder rupture following blunt trauma. Korean J Radiol. 2007;8:492–7.
9. Chan DPN, Abujudeh HH, Cushing GL, Novelline RA. CT cystography with multiplanar reformation for suspected bladder rupture: experience in 234 cases. AJR Am J Roentgenol. 2006;187:1296–302.