

The Case | Ascites with oliguric acute renal failure

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Figure 1 | An intravenous urography made 15 min after contrast injection.

A 50-year-old woman presented to the hospital with poor appetite, abdominal pain, and decreased urine output for several days. She had a history of endometrial cancer and received radical hysterectomy, radiotherapy, and chemotherapy 8 years ago. She had been receiving non-steroidal anti-inflammatory drugs frequently for chronic lower-back pain in the days preceding admission. On admission, blood pressure was 121/75 mm Hg and heart rate was 80 beats per minute. Physical examination revealed distended abdomen with shifting dullness and diffused tenderness. Mild abdom-

inal rebound pain was also noted. Laboratory examination disclosed elevated blood urea nitrogen (48.5 mg/dl) and creatinine (2.2 mg/dl) levels, hyponatremia (122 mmol/l), and hyperkalemia (5 mmol/l). Non-steroidal anti-inflammatory drugs were discontinued. Abdominal echo revealed massive ascites and bilateral normal kidney size, without hydronephrosis. Paracentesis demonstrated clear, light yellowish ascites with an unusual biochemistry profile, prompting us to perform intravenous urography, which is shown in Figure 1.

**What unusual biochemistry tests were performed in the ascitic fluid?
What was the cause of acute renal failure and how would you confirm the diagnosis?**

SEE NEXT PAGE FOR ANSWERS

The Diagnosis | Bladder rupture with urinary ascites

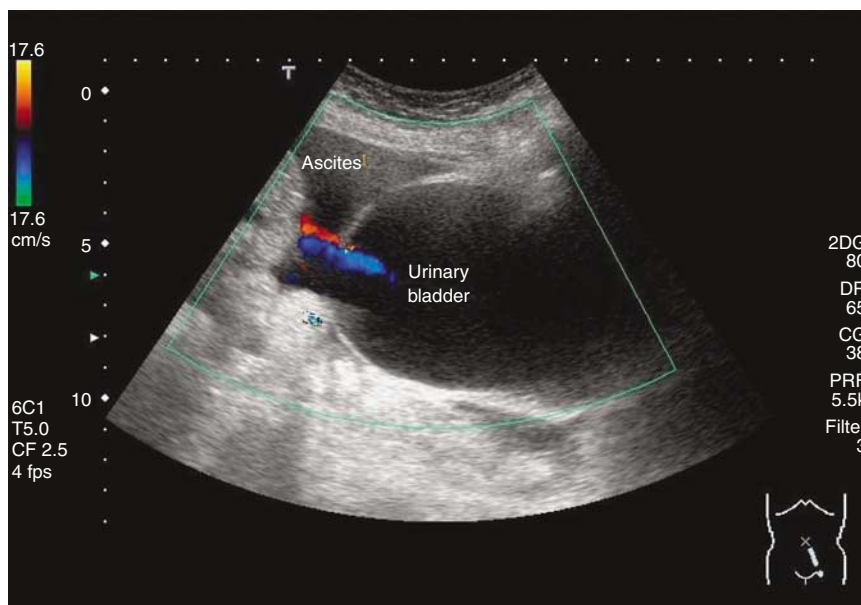


Figure 2 | Color duplex displayed flow between bladder and peritoneal space.

The biochemical profile of ascites revealed transudative fluid with levels of urea nitrogen and creatinine (61 and 5 mg/dl, respectively) higher than serum levels. Intravenous urography showed contrast leakage from the left upper wall of the urinary bladder (Figure 1). An ultrasonography documented a 0.7-cm defect over the left upper wall of the urinary bladder; color duplex revealed to-and-fro flow between the bladder and peritoneal space (Figure 2). Intraperitoneal bladder rupture was confirmed. She received transurethral catheterization for decompression and was discharged with normal renal function (blood urea nitrogen 9.3 mg/dl and serum creatinine 0.9 mg/dl) and electrolyte levels (serum sodium 139 mmol/l and potassium 4.3 mmol/l). Follow-up cystoscopy 2 months later revealed no vesicle-peritoneal fistula, and the Foley catheter was removed.

Possible etiologies for spontaneous bladder rupture include infravesicular obstruction, infectious lesions of bladder (emphysematous cystitis, tuberculosis, schistosomiasis, and candidiasis), bladder diverticulum, bladder carcinoma, and chemotherapy (prolonged cyclophosphamide treatment and intravesicle instillation of mitomycin C).¹ Pelvic irradiation is also known to cause spontaneous rupture of the urinary bladder, which can often develop several years after the initial radiation exposure.² In our case, bladder rupture occurred 8 years after radiotherapy.

The clinical presentations associated with spontaneous bladder rupture into the peritoneum space include ascites with abdominal distention and lower-abdominal pain associated with peritoneal signs. Impaired micturition and symptoms of

diaphragmatic irritation may also be diagnosed.¹ Due to reabsorption of urinary ascites across the peritoneum, a condition of oliguric renal failure with hyponatremia and hyperkalemia is typical.^{3,4} Blood urea nitrogen and creatinine levels in ascitic fluid are higher than serum levels and the fistula can be further confirmed by imaging studies such as contrast cystography or duplex sonography. However, sometimes the rupture may remain unrecognized until cystoscopy is performed.¹ Treatment of bladder perforation includes laparotomy for repair of the perforation, peritoneal lavage, and drainage of bladder through a suprapubic or transurethral catheter. Recently, laparoscopic closure of bladder perforation has also been performed.⁵

DISCLOSURE

All the authors declared no competing interests.

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