CASE REPORT

Pseudo-renal failure: bladder rupture with urinary ascites

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Accepted 31 October 2015

SUMMARY

We report a case of pseudo-renal failure caused by urinary ascites due to spontaneous bladder rupture following transurethral resection of a bladder tumour (TUR-BT). A 63-year-old man presented with 2 months of abdominal distension due to ascites. Laboratory findings showed elevated serum creatinine and hyperkalaemia. Peritoneal fluid urea, creatinine and potassium levels were greater than those in serum levels. CT scan showed partial wall thinning in the bladder wall, and cystography indicated fragility in the dome where the latest TUR-BT was performed. Pseudo-renal failure (laboratory abnormalities of acute kidney injury in the setting of normal kidney function) from urinary ascites and reverse intraperitoneal dialysis was diagnosed. Symptoms and laboratory abnormalities improved promptly with insertion of a urinary catheter. This report aims to increase recognition of urinary ascites when a patient with genitourinary surgical procedures or radiation therapy, or blunt abdominal trauma, presents with ascites and elevated creatinine simultaneously.

BACKGROUND

Pseudo-renal failure describes a rare condition, with laboratory abnormalities that mimic acute kidney injury (AKI) in the setting of normal kidney function. Administration of intravenous dexamethasone, creatine supplement consumption, and interference with automatic creatinine measurement by cefoxitin and acetone have been reported as causes of pseudo-renal failure.^{1–4} The most common cause is urinary ascites, which occurs when upper or lower urinary tract rupture causes intraperitoneal urine leakage followed by reverse intraperitoneal dialysis of urine.^{5–10} Laboratory findings in urinary ascites closely mimic AKI, including elevated blood urea nitrogen, elevated creatinine, hyperkalaemia and metabolic acidosis,⁵ and the concomitant presence of ascites may suggest hepatorenal syndrome. However, kidney and liver function are maintained in this condition, and renal replacement therapy is not generally required.⁵ Management of rupture of the urinary system is the key to recovery.

CASE PRESENTATION

A 63-year-old Japanese man presented with 2 months of abdominal distension.⁶ He had diabetes mellitus and a 7-year history of transitional cell carcinoma of the bladder, which had been treated by nine episodes of transurethral resection of a bladder tumour (TUR-BT). Six months before admission, the latest TUR-BT had been performed.

Two months earlier, the patient had experienced dysuria, which had improved spontaneously within several days. Shortly thereafter, abdominal distension was noted and progressed gradually over 2 months. At an outpatient visit 4 days before admission, a 2.4 kg body weight increase was observed, and the level of serum creatinine was 0.8 mg/dL. The next day, dysuria recurred. Abdominal distension worsened on the day of admission. The patient denied abdominal trauma, fever, dyspnoea, abdominal pain or haematuria.

INVESTIGATIONS

Physical examination showed temperature 36.6°C, pulse 105 bpm, blood pressure 140/94 mm Hg and respiratory rate 24 breaths/minute. The patient's abdomen was markedly distended with shifting dullness, but tenderness was not noted. Cutaneous signs of cirrhosis were absent. Laboratory findings disclosed serum albumin 5.0 g/dL, aspartate aminotransferase 14 IU/L, alanine aminotransferase 14 IU/L, urea nitrogen 87 mg/dL, creatinine 6.1 mg/dL and potassium 5.5 mmol/L. Urinalysis showed 3+ proteinuria and 1+ occult blood with 30–49 white cells per high-power field and no red cell casts. Urine biochemistry revealed protein 1.5 g per gram of creatinine, urea 612 mg/dL, creatinine 110 mg/dL and potassium 25 mmol/L. Fractional excretion of sodium was 3.2%. Urine culture was not obtained. CT scan disclosed massive ascites (figure 1) without hydronephrosis or cirrhosis. Clear yellow-coloured peritoneal fluid obtained by abdominal paracentesis revealed albumin 0.3 g/dL, urea 134 mg/dL, creatinine 21.4 mg/dL and potassium 7.6 mmol/L. Serum–ascites albumin gradient (SAAG) was 4.7 g/dL. White blood cell count of peritoneal fluid was 170/μL with 92% macrophages, 4.5% neutrophils and 3.5% mesothelial cells. Urine cytology disclosed mesothelial cells, which appeared similar to the mesothelial cells in the peritoneal fluid.

DIFFERENTIAL DIAGNOSIS

The most common causes of ascites are cirrhosis, chronic heart failure, infection (eg, tuberculosis) and malignancy. Less common causes include constrictive pericarditis, nephrotic syndrome, pancreatitis and urine. Ascites is best characterised by analysis of peritoneal fluid including SAAG. In this case, urinary ascites was suggested by levels of peritoneal fluid urea, creatinine and potassium that surpassed serum levels. Detection of mesothelial cells in urine cytology strongly suggests existence of urinary ascites.

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To cite: Matsumura M, Ando N, Kumabe A, *et al.* *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2015-212671

Figure 1 CT scan disclosed massive ascites without hydronephrosis or cirrhosis. Wall thinning was suspected in the dome of the urinary bladder (arrow).

TREATMENT

In total 4800 mL of urine was collected after urinary catheter placement, and the abdominal distension resolved. On hospital day 2, the patient's weight decreased by 2.5 kg. Serum creatinine decreased to 1.5 mg/dL and urinalysis revealed no proteinuria, no occult blood, no red cell casts and no white cells. On hospital day 3, serum creatinine decreased to 0.8 mg/dL. Retrospective evaluation of the CT scan on hospital day 4 showed partial wall thinning in the dome of the urinary bladder (figure 2A, B). Wall thinning was also suspected in the same part of the urinary bladder on the CT scan at admission (figure 1, arrow). On hospital day 8, cystography disclosed no perforation. However, wall thinning of the bladder in the location of the latest TUR-BT (figure 2C) was observed. The patient was discharged with a urethral catheter.

OUTCOME AND FOLLOW-UP

One month later, cystoscopy revealed no defects of the bladder wall. At the time of writing, the patient's serum creatinine level

has been stable at approximately 0.7 mg/dL, and ascites has not recurred in the subsequent 18 months.

DISCUSSION

This case highlights three interconnected phenomena: bladder rupture, urinary ascites and pseudo-renal failure. Bladder rupture can occur following blunt trauma,⁷ irradiation to the pelvis,⁵ or surgical procedures,^{8 9} or may be spontaneous.¹⁰ Bladder rupture following binge alcohol drinking is multifactorial including increased urine volume causing bladder distention, decreased perception of desire to void and blunt trauma.¹¹ Therapeutic procedures also cause bladder rupture. Hayashi *et al*⁵ described an elderly woman with spontaneous bladder rupture 10 years after pelvic irradiation for uterine cancer. Kim and Yang¹² reported an elderly patient with delayed bladder perforation following radical TUR-BT for invasive urothelial cancer.

We suspect that the patient had intermittent, low volume, self-resolving leaks over the months preceding admission and then developed a continuing leak in the few days preceding admission. When a bladder leak spontaneously seals, a kidney with near normal glomerular filtration rate can easily excrete the excess creatinine that is resorbed in the serum from the urinary ascites. When the bladder leak is ongoing, however, the excretory capacity is overwhelmed by the serum creatinine load from the urinary ascites, and therefore the serum creatinine increases. This may explain how the patient had a normal serum creatinine level 4 days before admission, but not at the time of admission.

Mokoena and Naidu¹³ reported a mean 5.4 day delay between an incident or presentation and diagnosis in 44 patients with bladder rupture including both intraperitoneal rupture (urinary ascites, n=36) and extraperitoneal rupture (n=8). The gold standard for the diagnosis of bladder rupture is exploratory laparotomy. However, this procedure is invasive for a stable patient without peritonitis. Alternative tests to support the diagnosis of urinary ascites include an ascites-to-serum creatinine ratio >1.0.^{10 14} In patients with urinary ascites, the serum-urinary ascites albumin gradient will be >1.1 g/dL, because urine does not contain significant albumin. Mesothelial cells in urine cytology suggest the transposition of mesothelial cells from the peritoneal cavity to the urinary bladder.⁵ CT cystography is the most sensitive and accurate diagnostic test for bladder rupture. Deck *et al*¹⁵ reported that the sensitivity and the specificity for diagnosing intraperitoneal bladder rupture using CT cystography were 78% and 99%, respectively. In this case, bladder perforation was suggested but not confirmed by cystography. However, gradual urine leakage from the fragile part of the bladder wall in the location of the latest TUR-BT was

Figure 2 Retrospective evaluation of the CT scan on hospital day 4 showed partial wall thinning in the dome of the urinary bladder (A: coronal plane, B: sagittal plane, arrows). Cystography revealed wall thinning at the site of the latest transurethral resection of a bladder tumour treatment (C: sagittal plane, arrowhead).

strongly suspected based on history, laboratory data and imaging studies. If CT cystography had been performed on the day of admission, it may have demonstrated a small bladder wall perforation.

The prognosis for bladder rupture is excellent when diagnosis and treatment are prompt. Normalisation of laboratory abnormalities usually occurs in <24 h. However, lower urinary tract leakage can lead to significant morbidity when diagnosis is delayed.¹⁶ Surgical repair is recommended for intraperitoneal rupture, although conservative therapy is a treatment option for selected patients, and cases of bladder rupture successfully managed without operative treatment have been reported.^{17 18}

Pseudo-renal failure due to urinary ascites closely mimics AKI and hepatorenal syndrome. This diagnostic error can lead to inappropriate medications or haemodialysis. Physicians should consider pseudo-renal failure in a patient with genitourinary surgical procedures or radiation therapy, or blunt abdominal trauma, who develops anuria or oliguria, ascites and an elevated creatinine level.

Learning points

- Pseudo-renal failure is a rare condition with laboratory abnormalities that mimic acute kidney injury in the setting of normal kidney function.
- Pseudo-renal failure with urinary ascites due to bladder rupture should be considered in a patient with genitourinary surgical procedures or radiation therapy, or blunt abdominal trauma, who develops anuria or oliguria, ascites and an elevated creatinine.

Contributors MM and AK contributed to the patient's management. MM, NA, AK and GD contributed to writing and reviewing the report. All the authors read and approved the final version of the manuscript.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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