

Case Report

Pseudo-azotaemia due to intraperitoneal urine leakage: a report of two cases

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Abstract

Ascites, oliguria and increasing serum creatinine levels are often noted in patients with acute kidney injury. However, these presentations are also observed in patients with intraperitoneal urinary leakage. Bladder perforation without obvious trauma is sometimes mistaken for acute kidney injury. We report two cases of bladder perforation resembling acute kidney injury. The first case was a 37-year-old woman with delayed intraperitoneal urinary leakage following total abdominal hysterectomy, and the second was a 70-year-old woman with spontaneous bladder perforation. Although the initial diagnosis in both cases was acute kidney injury, rupture of the urinary bladder was later identified.

Keywords: acute kidney injury; ascites; bladder perforation

Background

Rupture of the urinary bladder frequently occurs after blunt trauma. When detection is delayed or the rupture occurs spontaneously, it sometimes resembles acute kidney injury because ascites, oliguria and increasing serum creatinine levels are observed in patients with intraperitoneal urinary leakage [1,2]. We report two cases of bladder perforation resembling acute kidney injury.

Case report 1

A 37-year-old woman presented with post-operative acute abdominal distension and an increasing serum creatinine level 7 days after total abdominal hysterectomy for uterine myoma. She was afebrile. Abdominal examination revealed abdominal distension with shifting dullness and diffuse tenderness. Abdominal rebound tenderness was not exhibited. Her serum creatinine and urea nitrogen levels were elevated, but the serum beta2-microglobulin level was within normal limits. Urinalysis revealed pro-

teinuria and microhaematuria (Table 1). The patient had no history of kidney disease, and her serum creatinine level on post-operative Day 1 was normal (0.76 mg/dL). The urinary catheter had been removed 24 h after the operation. Paracentesis demonstrated ascitic fluid that was clear, light yellowish and non-bloody. Sodium, potassium and chloride levels in the ascitic fluid were 37, 19 and 76 mEq/L, respectively, which differed markedly from the serum electrolyte levels (Table 1). Ultrasound examination revealed normal kidney size bilaterally without evidence of hydronephrosis.

Because these symptoms may also be caused by drug-induced nephropathy, all medications were stopped. However, no decrease in ascites or in the serum creatinine level was observed. Since the patient's urine volume had decreased, a urinary catheter was inserted. Ascites subsequently resolved, and the serum creatinine level decreased to the normal range. We performed retrograde cystography, which revealed bladder perforation (Figure 1A). The perforation was closed surgically, and a subsequent retrograde cystography did not reveal urinary leak.

Case report 2

A 70-year-old woman with a history of radiotherapy for cervical cancer 16 years earlier presented with progressive abdominal distension over a 2-week period. On admission, she was found to have oedema and massive ascites. She was afebrile. Abdominal examination revealed abdominal distension with shifting dullness and diffuse tenderness. Abdominal rebound tenderness was not exhibited. Serum creatinine and urea nitrogen levels were elevated. Urinalysis revealed proteinuria and microhaematuria (Table 1). Paracentesis demonstrated clear, light yellowish and non-bloody ascitic fluid. Sodium, potassium and chloride levels in the ascitic fluid were 21, 23 and 78 mEq/L, respectively, which differed from the serum electrolyte levels (Table 1). Ultrasound examination revealed normal kidney size bilaterally without evidence of hydronephrosis. The patient was not taking medications.

Table 1. Laboratory findings of two cases of intraperitoneal urine leakage

Variables	Normal range	Case 1	Case 2
Serum			
Urea nitrogen (mg/dL)	8–20	45.9	21.9
Creatinine (mg/dL)	0.32–0.84	3.70	1.95
Sodium (mEq/L)	135–147	131	135
Potassium (mEq/L)	3.3–4.8	5.2	2.9
Chloride (mEq/L)	98–108	97	112
Beta2-microglobulin (mg/L)	1.1–2.5	1.1	No data
Urine			
Protein		3+	1+
Occult blood		3+	1+
Sodium (mEq/L)		15	No data
Potassium (mEq/L)		9	No data
Chloride (mEq/L)		20	No data
Beta2-microglobulin (μ g/L)	<230	49	No data
Ascites			
Sodium (mEq/L)		37	21
Potassium (mEq/L)		19	23
Chloride (mEq/L)		76	78

After placement of the catheter in the bladder, the ascites disappeared, and the serum creatinine level decreased to the normal range. We performed retrograde cystography, but bladder perforation was not detected. Because the patient's condition had improved, she was discharged from the hospital.

Three months after discharge, the patient was re-admitted with massive ascites and an increasing serum creatinine level. To determine the cause of serum creatinine elevation, we performed technetium-99m diethylenetriamine-pentaacetic acid (Tc-99m DTPA) renography, which showed extravasation of Tc-99m DTPA into the peritoneal cavity. Retrograde cystography revealed a small perforation in the bladder (Figure 1B, arrow). The patient declined surgical treatment and was discharged with catheter placement. No sign of recurrence had been noted during the 3-year follow up.

Discussion

Intraperitoneal urinary leakage is characterized by an increase in the serum creatinine level caused by reabsorption of creatinine in the urine through the peritoneal membrane, oliguria and ascites. Because most cases of intraperitoneal urinary leakage are the result of blunt trauma, leakage without obvious trauma may be misdiagnosed as acute kidney injury [1,2].

Case 1 was a patient who presented with bladder injury following total abdominal hysterectomy. The incidence of bladder injury after total abdominal hysterectomy is 0.1% [3]. Because most bladder injuries are identified intraoperatively, delayed appearance of urinary leakage is extremely rare. However, the literature includes one report of delayed intraperitoneal urinary leakage after Caesarean section [1]. Case 2 was a patient with spontaneous bladder perforation. Although spontaneous bladder perforation is uncommon, several cases have been reported in association with intravesicular obstruction, infectious lesion of

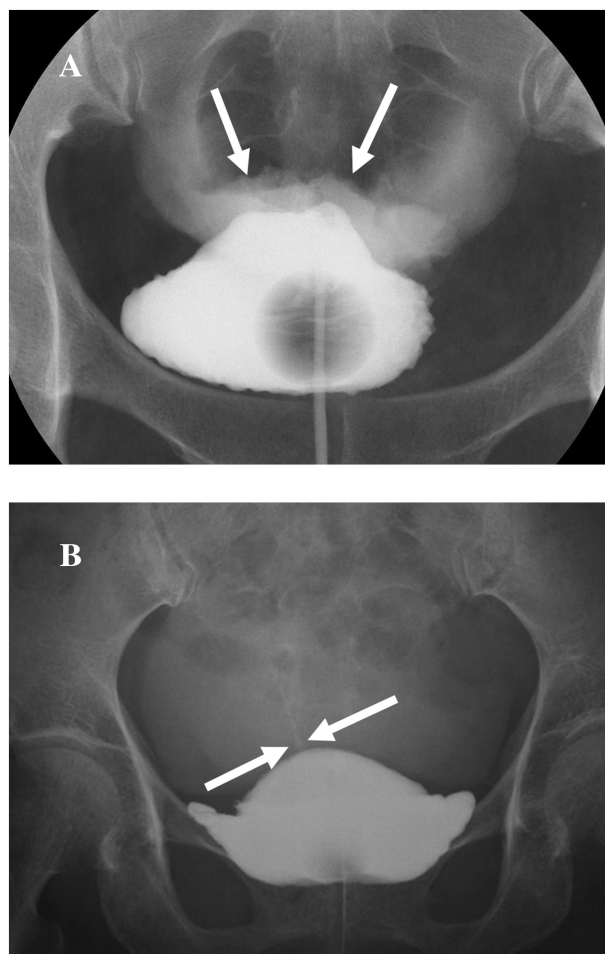


Fig. 1. Retrograde cystography exhibiting bladder perforation in Case 1 (A) and Case 2 (B).

the bladder, bladder diverticulum, bladder carcinoma, chemotherapy, and alcohol or substance abuse [2,4]. Pelvic irradiation is another cause of spontaneous bladder perforation. The reported incidence of spontaneous bladder perforation following radiotherapy for cervical cancer is 2.0% [5]. Perforation often develops several years after the initial radiotherapy [5].

Urinary leakage usually presents with signs of peritonitis [2,6]. Therefore, acute kidney injury with massive ascites and peritonitis should be distinguished from urinary leakage. However, our two cases had abdominal pain, but rebound tenderness, which is a sign of peritonitis, was not noted. Because bladder rupture without signs of peritonitis has been reported [4], symptoms of peritonitis may sometimes be unclear.

Although a correlation between serum and ascitic electrolyte levels has not been clearly established, some papers have reported this correlation [7–9]. Sodium, potassium and chloride levels in ascitic fluid are nearly identical to those in serum (ascites vs. serum; sodium 133.1 ± 6.6 vs. 131.8 ± 6.3 mmol/L, potassium 4.1 ± 0.8 vs. 4.3 ± 0.9 mmol/L and chloride 107.2 ± 7.6 vs. 101 ± 7 mmol/L) in cirrhosis patients [7]. In peritoneal dialysis patients, electrolyte levels in peritoneal dialysate that are retained

longer than 24 h are nearly identical to those in serum (peritoneal dialysate vs. serum; sodium 141.4 vs. 140.6 mEq/L, potassium 4.7 vs. 5.1 mEq/L and chloride 108.5 vs. 102.7 mEq/L) [8]. Another report noted that the ratio of sodium, potassium and chloride between serum and transudates which include ascites is 0.95, 0.74–0.80, and 0.95–0.99, respectively [9]. However, in our two cases, the ascitic sodium, potassium and chloride levels differed markedly from their serum levels. These differences were probably caused by the leakage of urine into the abdominal cavity. Ascitic electrolyte levels may therefore be helpful in diagnosing intraperitoneal urinary leakage.

Both serum beta2-microglobulin and creatinine levels are usually elevated in patients with acute kidney injury [10]. This was not the case in our first patient, and this discrepancy may be a clue to the presence of intraperitoneal urinary leakage. The reason for this discrepancy has not been elucidated; however, beta2-microglobulin, due to its larger molecular size, may not be absorbed through the peritoneal membrane similar to creatinine. In addition, although we did not measure urea nitrogen and creatinine in the ascitic fluid, the higher levels in ascites than in the blood are additional clues suggesting the possibility of intraperitoneal urinary leakage [11].

In summary, we report two cases with intraperitoneal urinary leakage resembling acute kidney injury. These cases suggest that bladder perforation should be considered in the differential diagnosis of acute kidney injury with massive ascites.

Conflict of interest statement. None declared.

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Received for publication: 1.2.10; Accepted in revised form: 21.5.10