



New-Onset Massive Ascites With Acute Kidney Injury Due to Spontaneous Rupture of the Urinary Bladder

Chien-Hua Chen^{1,2}, Yun-Te Chang^{2,*}, Neng-Chyan Huang², Fong-Dee Huang²

¹Department of Emergency Medicine, Chiayi Branch, Taichung Veterans General Hospital, Chiayi, Taiwan

²Department of Emergency Medicine, Kaohsiung Veterans General Hospital, Kaohsiung, Taiwan

Spontaneous rupture of the urinary bladder (SRUB) is a rare urological emergency associated with urinary ascites and apparent acute kidney injury. The clinical course can be severe or lethal if no early diagnosis is achieved. We present a 44-year-old woman with type 2 diabetes mellitus had progressive abdominal fullness, poor appetite, dyspnea, and lower legs edema for two days. Laboratory studies present as acute kidney injury. Abdominal sonogram and computed tomogram (CT) revealed massive ascites. Her ascites relieved dramatically after Foley catheter indwelling, and daily urine amount was about 5,000 mL. Urinary bladder rupture was found after CT cystography. She received surgical repair of the ruptured urinary bladder, and cystometry revealed detrusor areflexia. Then she was discharged uneventfully.

Key words: *spontaneous urinary bladder rupture, SUBR, acute kidney injury, massive ascites*

Introduction

Spontaneous or idiopathic rupture of urinary bladder (SRUB) was defined as the ruptured urinary bladder occurred in a patient who had no evidence of trauma or instrumentation.¹⁻⁴ There were only limited articles describing the SRUB.¹⁻⁴ This report described an unusual case of a urinary bladder rupture who did not have any recent history of trauma or operation, but associated with type 2 diabetes mellitus bladder dysfunction.

Case Report

A 44-year-old woman visited our emergency department with progressive abdominal fullness, poor appetite, dyspnea, and lower legs edema for two days. She has hypertension, type 2 diabetes mellitus, and hyperlipidemia for 10 years under regular drugs control. She received tubal ligation about 10 years ago, and she had no other surgery thereafter. She denied

drug abuse or trauma recently. Physical examination revealed distended abdomen with ovoid appearance, mild and diffuse abdominal tenderness, shifting dullness, and lower legs edema. Initial laboratory studies reported white blood cells 9,500/ μ L with neutrophil 65% and lymphocyte 27%, blood urea nitrogen 65.0 mg/dL, serum creatinine 5.1 mg/dL, normal activity of liver enzymes, and normal concentrations of bilirubin, urea, and electrolytes. Sonography revealed much lower abdominal ascites, and 1,500 mL of ascites was drained. The analysis of ascites revealed white blood cells 30/ μ L with lymphocyte 98% and neutrophil 2%, red blood cells 40/ μ L, protein 2.0 gm/dL, and lactate dehydrogenase 114 U/L (serum lactate dehydrogenase 134 U/L). No air-fluid level was noted in plain abdominal film (Fig. 1A). Abdominal computed tomogram (CT) was arranged for suspecting hollow organ perforation, but it only revealed much ascites without other specific finding. Then ascites was drained about 2,000 mL for two days to relieve the abdominal distension and dyspnea. But oliguria

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*Corresponding author: Yun-Te Chang, MD, MPH, Department of Emergency Medicine, Kaohsiung Veterans General Hospital, No. 386, Taichung 1st Rd., Kaohsiung 813, Taiwan. E-mail: yuytchang@vghks.gov.tw

and progressive azotemia with elevated blood urea nitrogen (91.0 mg/dL) and serum creatinine (9.1 mg/dL) occurred. Foley catheter was indwelled with daily urine amount 5,120 mL, and ascites relieved dramatically. Spontaneous urinary bladder rupture was highly suspected. Cystogram revealed pneumohydroperitonium and extravasation of contrast medium (Fig. 1B). The abdominal CT cystogram presented perforational hole at left upper portion of urinary bladder (Fig. 2) with accumulation of contrast medium at the cul-de-sac, paravesical space, and intramesenteric space (Figs. 2 and 3). Surgery was performed emergently to repair the ruptured urinary bladder. The postoperative course was uneventful, and ascites, legs edema, and azotemia were also improved. Cystometry revealed detrusor areflexia (Fig. 4). Before discharge, laboratory studies re-

ported white blood cells 8,330/ μ L with neutrophil 80% and lymphocyte 13%, blood urea nitrogen 8.0 mg/dL, serum creatinine 0.6 mg/dL. She was discharged home with a regular follow-up in the out-patient clinic.

Discussion

SRUB is uncommon with a low incidence of 1:126,000 but a high mortality (47%).⁵ The significant mortality could be attributed to the delayed diagnosis in lower urinary tract leakage.⁶ A mean 5.4 day delay between an identifiable incident or presentation and diagnosis in 44 patients with bladder rupture was reported by Mokoena and Naidu.⁷ Common causes of SRUB were bladder wall lesion, bladder outflow obstruction, drug or substance, pregnancy or labor, and idiopathic (Table 1).⁸⁻¹⁰

Classical symptoms of SRUB may include triad of pain, difficulty or inability to void, and rigidity of the abdominal wall.² But acidosis, azotemia, fever and sepsis, low urine output, peritonitis, ileus, urinary ascites, or respiratory difficulties could be present if bladder injury was unrecognized.¹¹ Reabsorption of urea and creatinine through peritoneal dialysis could increase the blood urea nitrogen and serum creatinine and resemble reversible acute pseudo-renal failure.^{4,12} Diabetic patients could have a wide variety of bladder dysfunction from overactive bladder and urge incontinence to decreased bladder sensation and overflow incontinence,¹³ and detrusor hyperreflexia, reduced detrusor contractility, or detrusor areflexia could be found in cystometry.¹⁴ In this case, the detrusor are-

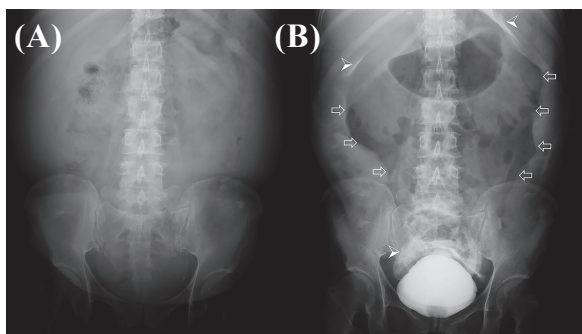


Fig. 1. Cystogram revealed pneumohydroperitonium (arrows) and extravasation of contrast medium (arrow heads) (B), in contrary to the plain abdominal film studied at emergency department, which did not show these abnormalities (A).

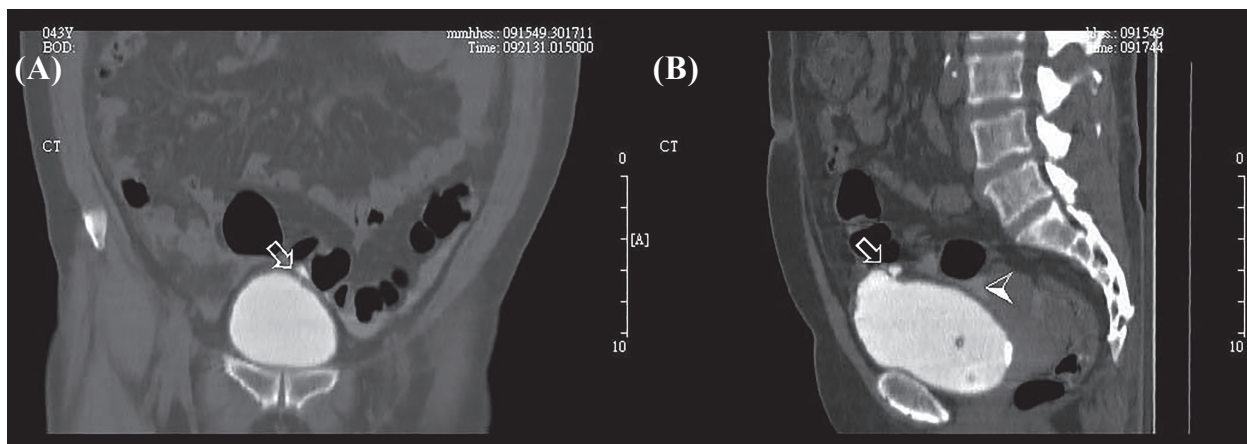


Fig. 2. Abdominal computed tomogram cystogram revealed a perforational hole at left upper portion of urinary bladder (arrows), coronal (A), and sagittal sections (B). Contrast medium filled the cul-de-sac space (arrow heads) can be seen.

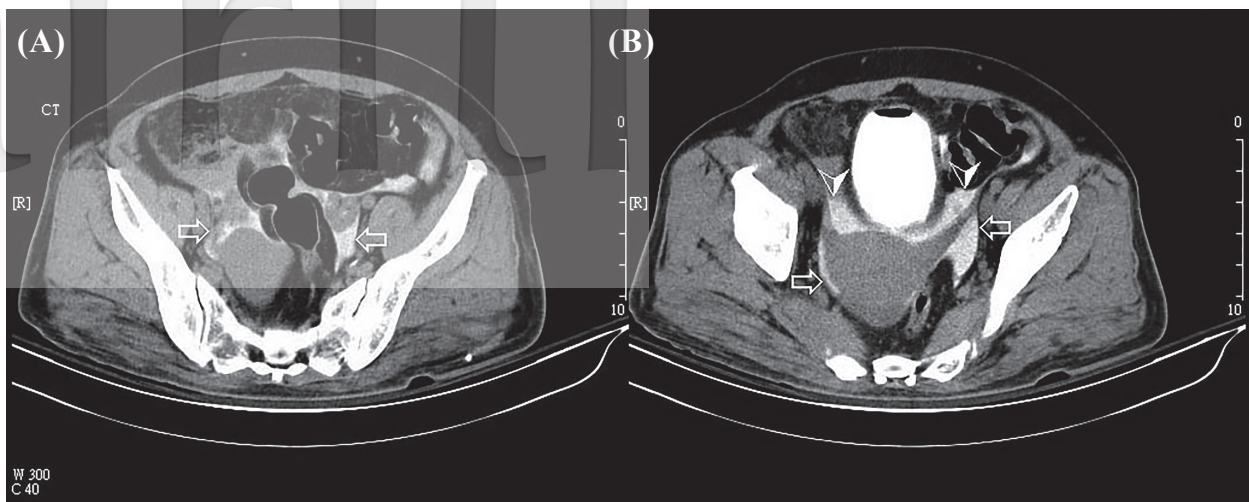


Fig. 3. Abdominal computed tomogram cystogram revealed accumulation of contrast medium at the intramesenteric space (arrows) (A), the cul-de-sac (arrows) (B), and the paravesical space (arrow heads) (B).

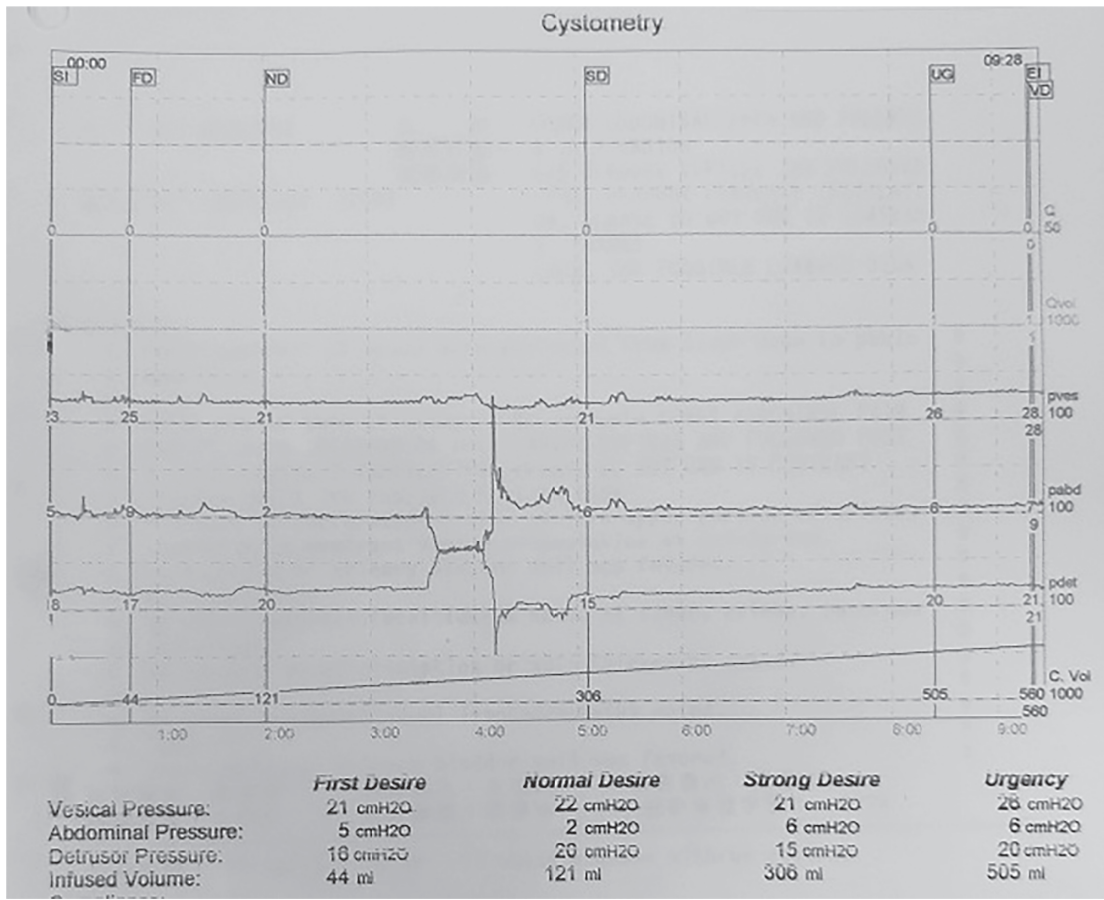


Fig. 4. Cystometry revealed that detrusor pressure was not increased at first desire, normal desire, strong desire, or urgency, indicating detrusor areflexia.

Table 1. Common causes of spontaneous urinary bladder rupture

Category	Common causes
Bladder wall lesion	(1) Inflammation or infection: gonococcal, tuberculous, schistosomias, tubo-ovarian abscess, chronic/fungal cystitis, etc. (2) Carcinoma (3) Bladder diverticulum (4) Previously repaired bladder perforation (5) Pelvic irradiation
Bladder outflow obstruction	(1) Obstructive: prostatic hypertrophy, Foley catheter, calculus, etc. (2) Neurologic: stroke, tabes dorsalis, spinal cord lesion, autonomic neuropathy
Drug or substance	Alcohol, cocaine, methamphetamine, etc.
Pregnancy or labor	Retroverted uterus, obstructed labor, postpartum
Idiopathic	

flexia could cause overdistended bladder, and then the bladder could rupture spontaneously.

The diagnostic accuracy of abdominal CT (60.6%) was relative lower than that of retrograde cystogram (95.9%).¹⁵ CT cystography is equivalent to retrograde cystogram for detecting bladder injury.¹⁶ Intraperitoneal bladder injury should be managed by immediate operative repair, but uncomplicated extraperitoneal bladder ruptures could be conservatively managed with urethral catheter drainage alone.¹¹

Conclusions

Spontaneous urinary bladder rupture is difficult to diagnose clinically, even with advanced techniques such as sonogram or CT. Rapid resolution of ascites and acute renal failure after Foley catheterization could assist to find the correct diagnosis, but to make the decision of Foley catheter indwelling is the most difficult if the patient still can urinate. However, early diagnosis and surgery, but not focused on the treatment of ascites or acute kidney injury, are suggested in such clinical situation.

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