

PSEUDO-ACUTE KIDNEY INJURY SECONDARY TO SPONTANEOUS BLADDER RUPTURE AND URINARY ASCITES: A CASE REPORT

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Abstract

The concurrent presentation of oliguria, massive ascites, and severe azotemia typically suggests true acute kidney injury (AKI), such as hepatorenal syndrome. However, this clinical picture can be mimicked by the rare condition of pseudo-AKI. We report a 42-year-old woman with CKD stage 3 and prior subtotal hysterectomy who presented with progressive oliguria, abdominal distension, weight gain, and a serum creatinine of 6.4 mg/dL. An initially high serum–ascites albumin gradient (SAAG) misleadingly pointed toward portal hypertension despite no clinical evidence of liver disease. Contrast-enhanced pelvic MRI demonstrated small-volume pelvic ascites with suspected contrast extravasation adjacent to the bladder, consistent with urinary leakage, and a following ascitic fluid analysis confirmed urinary ascites with an ascitic fluid-to-serum creatinine ratio of 6.4. Azotemia resolved completely with conservative management via continuous bladder drainage. This case underscores the importance of considering urinary ascites in the differential diagnosis of AKI with ascites and past pelvic surgery, while also highlighting the diagnostic value of ascitic fluid analysis, especially when imaging studies are inconclusive.

Key Words: pseudo-acute kidney injury, urinary ascites, bladder rupture, peritoneal dialysis, case report

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Introduction

The clinical triad of acute azotemia, oliguria, and ascites presents a formidable diagnostic challenge, often prompting a presumptive diagnosis of true acute kidney injury (AKI).¹ In patients without overt signs of post-renal obstruction, conditions such as hepatorenal syndrome, cardiorenal syndrome, or acute tubular necrosis are typically considered.²⁻⁵ However, this clinical picture can be mimicked by a rare but critical condition known as pseudo-AKI, where laboratory abnormalities suggest renal failure but intrinsic glomerular filtration is preserved.^{2,3,5,6}

The pathophysiology of pseudo-AKI in this context is most commonly due to urinary ascites, a condition resulting from the intraperitoneal extravasation of urine, typically following a rupture of the urinary bladder or ureter.^{1,7} The peritoneum, with its large surface area, functions as a semipermeable membrane. This allows for the passive diffusion of highly concentrated solutes, such as creatinine and urea, from the collected urine in the peritoneal cavity into the much more dilute bloodstream. This phenomenon, termed “reverse peritoneal dialysis,” artificially elevates serum creatinine and BUN levels, creating a biochemical profile indistinguishable from true AKI.⁸⁻¹⁰ While blunt trauma is a frequent cause of bladder rupture, spontaneous rupture can occur, particularly in bladders weakened by prior pathology or surgery, such as hysterectomy.¹¹

This report describes the case of a 42-year-old woman with a remote history of emergent subtotal hysterectomy as a lifesaving procedure for fatal, massive post-partum hemorrhage, who presented with severe azotemia and massive ascites. We aim to detail the diagnostic pathway, emphasizing the pivotal role of ascitic fluid analysis in differentiating pseudo-AKI from true renal failure, and to highlight the efficacy of conservative

management. This case underscores the importance of maintaining a high index of suspicion for urinary ascites in patients with a history of pelvic surgery who present with apparent AKI.

Case Presentation

Patient Information and Past Medical History

The patient was a 42-year-old woman with a medical history significant for chronic kidney disease (CKD) stage 3, with a baseline serum creatinine of approximately 1.1 mg/dL, and hypertension. Her history was notable for a critically complicated pregnancy two years prior to the current presentation, specifically a massive postpartum hemorrhage complicated by disseminated intravascular coagulopathy (DIC) in August 2019. Her life-saving management included an emergency subtotal hysterectomy, cystoscopy with right ureteral catheter insertion, and supportive care with veno-venous extracorporeal membrane oxygenation (VV-ECMO) for respiratory failure and continuous veno-venous hemofiltration (CVVH) for AKI and shock liver. She eventually recovered and was discharged in November 2019.

Clinical Presentation and Initial Investigations

In August 2021, two years after her hysterectomy, the patient presented to the emergency department (ED) with a two-week history of progressively decreasing urine output, intermittent lower abdominal pain, and significant abdominal distention with a 5 kg weight gain, accompanied by nausea and vomiting but not fever. On physical examination, her abdomen was distended and tense with tenderness in the lower quadrants but without signs of peritonitis or fluid accumulation at other body parts.

Initial laboratory studies on admission (September 3, 2021) revealed a serum creatinine (Cr) of 6.4 mg/dL and a blood urea nitrogen (BUN) of 74.8 mg/dL, indicating severe acute-on-chronic kidney injury, while serum alanine transaminase (ALT) among other liver function tests remained within normal limits. Urinalysis also disclosed hematuria of red blood cell (RBC) ≥ 100 /HPF, and a fractional excretion of urea nitrogen of 51.2%. A non-contrast computed tomography (CT) scan of the abdomen and pelvis showed a moderate volume of ascites but unremarkable kidneys with no evidence of hydronephrosis, and no findings suggestive of liver cirrhosis or portal hypertension.

Diagnostic Assessment

The initial diagnostic picture was perplexing. A diagnostic paracentesis performed on September 3 drained 3,740 mL of serosanguinous fluid. Analysis of this fluid yielded a serum-ascites albumin gradient (SAAG) greater than 3 g/dL, a finding that strongly suggests portal hypertension as the etiology of the ascites. However, this was inconsistent with the patient's clinical history and imaging, which showed no evidence of liver disease. At this moment, portal vein thrombosis could not be excluded, necessitating contrast-enhanced imaging studies and tests of coagulation panel; the latter disclosed no abnormalities except for an elevated IgG anticardiolipin of 82 GPL-U/ml.

Following the large-volume paracentesis, the patient became completely anuric. On September 4, a bedside ultrasound revealed re-accumulation of a large volume of ascites and a distended urinary bladder. Subsequently, contrast-enhanced pelvic MRI demonstrated no intravascular thrombosis but focal extravasation of gadolinium from the urinary bladder—most conspicuous at the dome—into the peritoneal cavity, consistent with an intraperitoneal urinary leak (Fig. 1 and 2). A Foley catheter was inserted, which yielded an immediate return of 700 mL of urine that was visually identical to

the previously drained ascitic fluid. This was followed by a profound diuresis, with a total urine output of 7,335 mL over the subsequent 24 hours. Concurrently, her renal function began to improve dramatically (Table 1).

This striking clinical response prompted a targeted diagnostic paracentesis on September 9 to compare fluid chemistries, and also a trial to remove her Foley catheter. The results were definitive: the ascitic fluid creatinine was 17.9 mg/dL and BUN was 66.4 mg/dL, while the simultaneous serum creatinine was 2.8 mg/dL and BUN was 26.6 mg/dL after transient removal of Foley catheter. This yielded an ascitic fluid-to-serum creatinine ratio of 6.4 and a BUN ratio of 2.5. A ratio greater than 1.0 is pathognomonic for urinary ascites, confirming that the peritoneal fluid was, in fact, urine from a bladder leak.^{2,3,8,10}

Therapeutic Intervention and Outcome

With the diagnosis of pseudo-AKI from urinary ascites confirmed, the patient was managed conservatively. A urology consultation recommended continuous bladder drainage instead of surgical repair to promote healing of the presumed bladder defect. The Foley catheter was replaced with a 20-French nephrostomy tube, which was maintained on low-pressure suction (10 mmHg). The patient's serum creatinine rapidly returned to her baseline of approximately 0.9 mg/dL, and the ascites completely resolved. A retrograde cystography with CT scan performed on September 17, after one week of continuous bladder drainage, showed no further evidence of contrast extravasation, suggesting the bladder wall defect had sealed (Fig. 3). The patient was discharged in stable condition on September 18 with the catheter in situ for an additional period of bladder rest, with outpatient urology follow-up arranged. The patient remained stable 4 years after this event, with a baseline serum creatinine of 1.0 mg/dL.

Table 1. Timeline of Key Clinical and Laboratory Events

Date (2021)	Key Event	Serum Creatinine (mg/dL)	Serum BUN (mg/dL)	Urine Output (mL/24h)	Key Fluid Analysis
Aug 28	ED visit for abdominal Pain	1.6	-	Decreased	-
Sep 01	Urology outpatient clinic	5.5	-	Oliguria	Renal echo: Massive ascites
Sep 03	Admission	6.4	74.8	Anuric	Paracentesis #1 (3740 mL)
Sep 04	Foley catheter inserted	4.8	64.2	7,335	-
Sep 06	Post-drainage	0.7	8.1	~3235	-
Sep 09	Diagnostic paracentesis #2	2.8	26.6	~680	Ascites Cr: 17.9 mg/dL; Ascites BUN: 66.4 mg/dL
Sep 13	Continued drainage	0.9	13.8	~1840	-
Sep 18	Discharge	0.9	16.4	Stable	Foley in situ

Abbreviation: BUN: blood urea nitrogen; ED: emergency department; Cr: creatinine

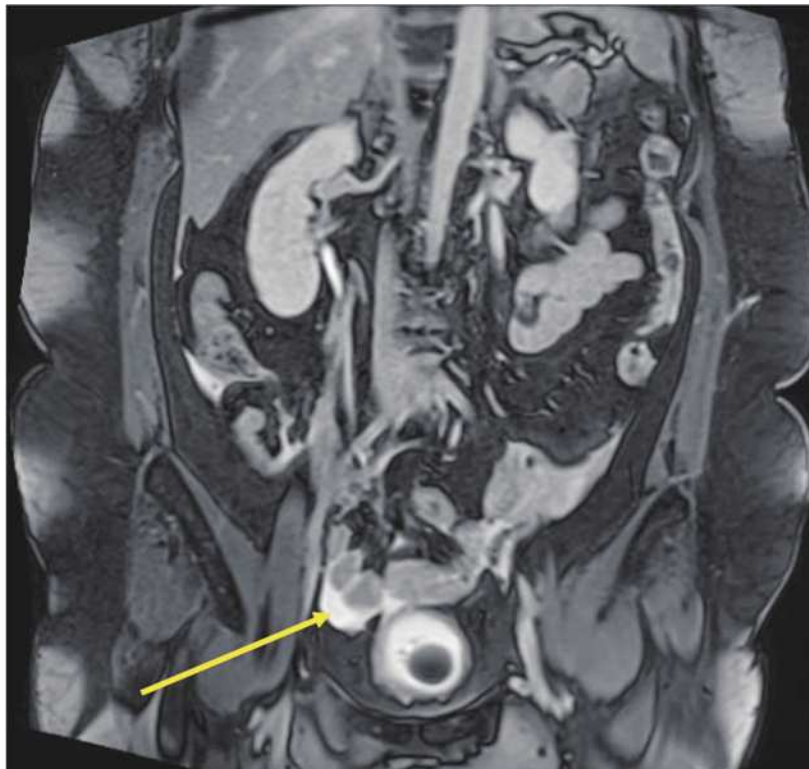


Fig. 1. MRI of the pelvis with intravenous contrast demonstrates a small amount of ascites in the pelvic cavity. Contrast extravasation is visible adjacent to the bladder, raising suspicion of urine leakage. A Foley catheter is noted in situ. (Abbreviation: MRI: magnetic resonance imaging)

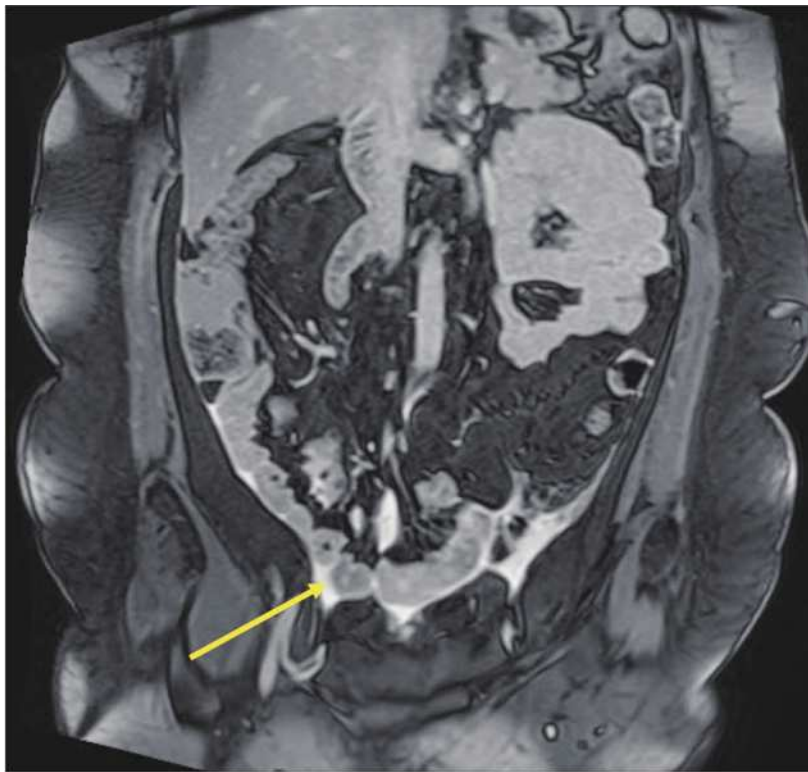


Fig. 2. Coronal MRI of the abdomen and pelvis shows small pelvic ascites with suspected contrast extravasation, consistent with urinary leakage. No hydronephrosis or renal lesions are observed. (Abbreviation: MRI: magnetic resonance imaging)

Discussion

This case provides a compelling illustration of pseudo-AKI secondary to remote spontaneous bladder rupture and urinary ascites. It demonstrates how a clinical syndrome mimicking end-stage renal failure can arise from a correctable mechanical issue, and it highlights how adherence to fundamental pathophysiological principles can guide clinicians away from a misdiagnosis and unnecessary, invasive interventions like hemodialysis.^{2,3,7,12}

The underlying mechanism of this phenomenon is “reverse peritoneal dialysis”.^{2,9} The peritoneal membrane, with its extensive surface area and rich vascular supply, allows for the bidirectional transfer of solutes. In the setting of urinary ascites, the

extremely high concentration of waste products in the urine—creatinine levels 30 to 100 times higher than in serum—creates a steep concentration gradient that drives the passive diffusion of these solutes from the peritoneal cavity into the bloodstream, artificially inflating serum creatinine and BUN levels,^{3,8,10} as well as hyperkalemia, hyponatremia, and acidosis in some patients.^{2,7,11} Serum creatinine typically rises not immediately but within 24 hours after initial urinary leakage,^{1,7} which explains why the patient’s serum creatinine underwent such a dramatic rise between the two ED visits.

A key diagnostic challenge in this case was the initial finding of a high SAAG, which typically points toward portal hypertension.^{5,12,13} This finding was a significant red herring, as the patient had no clinical or imaging evidence of liver disease. The

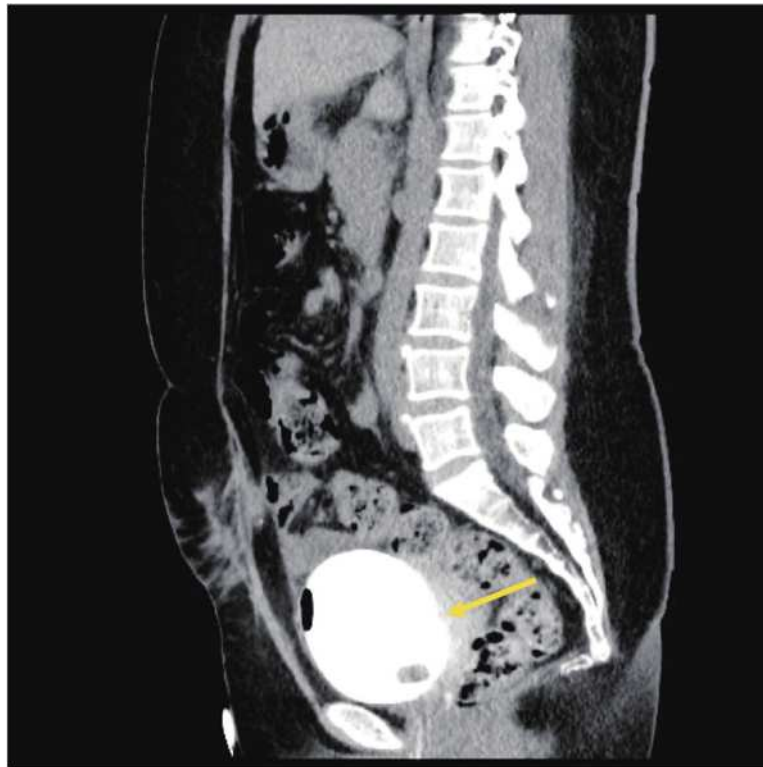


Fig. 3. Contrast-enhanced CT of the abdomen and pelvis after treatment shows no extravasation to the pelvic cavity, while displaying irregular thickening on the posterior wall (arrow), suggesting probable bladder healing after the rupture. (Abbreviation:CT: computed tomography)

literature notes that urinary ascites can, in fact, present with a high SAAG, likely due to peritoneal irritation or other factors altering fluid dynamics, thus serving as a crucial diagnostic pitfall.⁵ This case reinforces that laboratory tests, however specific, must be interpreted within the complete clinical context. The diagnostic test of choice was the direct comparison of ascitic fluid and serum creatinine levels. An ascitic fluid-to-serum creatinine ratio >1.0 is considered pathognomonic for urinary ascites, and in this patient, the ratio was a striking 6.4, unequivocally confirming the diagnosis.^{2,8,10,13} Retrospective studies regarding postoperative tracking of ascitic fluid-to-serum also verified the high correlation between the data to urinary leakage alongside its utility for preliminary detection.^{15,16}

Other laboratory findings that could be suggestive of urinary ascites include mesothelial cells found in urine, urothelial casts within peritoneal fluid, and elevated serum creatinine-to-cystatin C ratio.^{12,17,18} In particular, because the urinary cystatin C level is low compared to creatinine, reverse peritoneal dialysis would not affect the serum cystatin C level, leading to an elevated serum creatinine-to-cystatin C ratio during urinary ascites.^{12,19} Therefore, it can serve as a surrogate marker for ascitic fluid-to-serum creatinine ratio when peritoneal fluid data is lacking.^{12,20} Together, these simple and inexpensive tests should be considered as early as possible in any patient presenting with concurrent ascites and AKI of unclear etiology, since delayed diagnosis of urinary ascites could lead to high mortality.^{20,21}

Imaging has a decisive role in localizing the defect and defining the route of leakage. When intraperitoneal rupture is suspected, CT cystography—retrograde filling of the bladder with contrast followed by CT acquisition—provides high spatial resolution to demonstrate contrast extravasation and to distinguish intraperitoneal from extraperitoneal injury patterns that carry different management implications. Currently, CT cystography has supplanted conventional plain-film cystography as the first line radiological tool to evaluate bladder rupture because of comparable accuracy and better integration into abdominopelvic CT workflows with capturing of concomitant injuries, with both achieving 95% sensitivity and 100% specificity.^{22,23} Although considered the diagnostic gold standard by some, caution should be made especially when encountering negative results, as underdistended bladder and temporary sealing both result in false negative imaging.²⁴ In these cases, biochemical testing remains the crucial complementary information.

The etiology of the bladder rupture in this patient was likely a delayed complication of her subtotal hysterectomy performed two years prior. Major pelvic surgeries, especially gynecological, colorectal, or urological procedures, can lead to scarring and the creation of a point of weakness, typically at the bladder dome, predisposing it to subsequent spontaneous rupture even without acute trauma.^{1,8,21,23} Despite the fact that most bladder leakage is discovered intraoperatively or within 30 days after a gynecological surgery,^{25,26} delayed rupture years after the operations is still well-documented,^{27,28} urging awareness to the patient's surgical history regardless of the remoteness of the event.

Ultimately, the management was remarkably straightforward. The rapid and complete resolution of severe azotemia with the simple placement of a

Foley catheter was both diagnostic and therapeutic, allowing serum creatinine to return to normal within 48 hours.^{1,10,13} This conservative approach allowed the bladder defect to heal spontaneously, avoiding the morbidity and cost of hemodialysis or surgical exploration. One retrospective study also demonstrated that in isolated intraperitoneal bladder perforation, conservative treatment is effective for over 90% of patients.²⁹ This case presents a powerful contrast between the patient's exceedingly complex medical history and the elegantly simple solution to her acute crisis, underscoring the enduring value of clinical reasoning grounded in pathophysiology.

Conclusion

Pseudo-AKI secondary to urinary ascites is a rare but critical diagnosis that can mimic severe intrinsic renal disease. Clinicians should maintain a high index of suspicion in patients presenting with oliguria, azotemia and ascites without hepatic failure, particularly in those with a history of pelvic surgery regardless of the extent of remoteness. The most valuable diagnostic clue is the rapid improvement of renal function following bladder drainage. The definitive diagnosis can be confirmed by a simple test: an ascitic fluid-to-serum creatinine ratio greater than 1.0, augmented by contrast-enhanced imaging modalities. Prompt recognition and conservative management with continuous bladder drainage can lead to complete resolution, preventing unnecessary and potentially harmful interventions.

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尿液性腹水導致偽急性腎損傷：病例報告

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摘要

臨床上病患若同時出現少尿、大量腹水及嚴重氮血症，常直覺聯想到急性腎損傷 (acute kidney injury, AKI)，如肝腎症候群。然而，這類表現亦可能由罕見之偽急性腎損傷所致。本報告呈現一例自發性膀胱破裂導致之尿液性腹水，造成偽急性腎損傷之個案，並探討其診斷思維與治療重點。本例為一名42歲女性，具慢性腎臟病第三期及子宮次全切除術病史，因持續進展之寡尿、腹脹與體重增加就診，入院時血清肌酸酐升至6.4 mg/dL。初步腹水分析顯示血清-腹水白蛋白梯度(SAAG)偏高，指向門脈高壓，然臨床上並無肝病證據。骨盆腔增強MRI顯示骨盆腔少量腹水，膀胱鄰近可見對比劑外滲，與尿液滲漏相符，結合後續腹水與血清分析顯示腹水肌酸酐/血清肌酸酐比值高達6.4，而確診為尿液性腹水。推測病因為先前婦科手術導致之膀胱壁弱點，發生自發性破裂。經持續膀胱引流治療後，腎功能完全恢復，無需進一步侵入性處置。此病例提示，對於有骨盆腔手術史且出現腹水與AKI之病患，應將尿液性腹水列入鑑別診斷。腹水肌酸酐與血清肌酸酐比值 >1.0 為簡便且具鑑別力之檢驗，應於第一時間施行以避免不必要處置。

關鍵詞：偽急性腎損傷，尿液性腹水，膀胱破裂，腹膜透析，病例報告

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