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# URINARY ASCITES SECONDARY TO RECURRENT SPONTANEOUS BLADDER RUPTURE MIMICKING RENAL FAILURE: A DIAGNOSTIC ODYSSEY IN A POST-RADIATION PATIENT

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## ABSTRACT

*Introduction:* Urinary ascites secondary to spontaneous rupture of the urinary bladder (SRUB) is a rare clinical entity presenting with non-specific symptoms, which poses a significant diagnostic challenge. A specific presentation involves pseudo-renal failure because of peritoneal reabsorption of urinary solutes.

*Case description:* We present the case of a female in her sixties with recurrent severe abdominal pain and ascites, concomitated with apparent acute kidney injury. Her medical history was significant for Wertheim-Meigs hysterectomy and pelvic radiation for cervical cancer. She underwent multiple hospitalisations with extensive investigations and even exploratory surgeries, driven by misleading CT findings of diffuse bowel wall thickening and gross ascites. The definitive diagnosis was made by extensive review of the patients' medical records and research of similar cases in the literature, as also by paracentesis, revealing an ascites-to-serum creatinine ratio of 3.76. A computed tomography cystogram confirmed an intraperitoneal bladder rupture with contrast extravasation. The patient was successfully managed conservatively with intermittent self-catheterisation.

*Conclusion:* In patients with urogenital comorbidities developing large-volume ascites without preceding trauma, urinary ascites should be considered an important differential diagnosis, typically defined by an ascites-to-serum creatinine ratio of >1. This case explores the diagnostic pitfalls of urinary ascites secondary to SRUB in a patient with late radiation cystitis focusing on the phenomenon of reverse auto peritoneal dialysis and a normal cystatin C level as key diagnostic clues.

## KEYWORDS

Spontaneous rupture of the urinary bladder (SRUB), urinary ascites, pseudo-renal failure

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## LEARNING POINTS

- Urinary ascites by spontaneous bladder rupture is a rare but critical late complication of pelvic radiation therapy, often misdiagnosed by its non-specific symptoms.
- Urinary ascites drives the diffusion of urinary solutes back into the systemic circulation, mimicking acute kidney injury by reverse auto peritoneal dialysis.
- The following constellation of laboratory parameters can be found in uroperitoneum: high serum creatinine but normal serum cystatin C, high serum-ascites albumin gradient and a high ascites-to-serum creatinine ratio.

## INTRODUCTION

Urinary ascites secondary to spontaneous rupture of the urinary bladder (SRUB) represents a rare urological emergency. SRUB is characterised by a urinary bladder perforation without preceding trauma and it is frequently misdiagnosed due to non-specific symptoms such as abdominal pain and difficulty voiding[1], which are often overshadowed by concomitant findings such as acute peritonitis, gross ascites and bowel wall thickening. A particularly confounding aspect is the development of pseudo-renal failure. This metabolic derangement occurs when urinary solutes diffuse from the peritoneal fluid back into the circulatory system resulting in reverse auto peritoneal dialysis[2]. This report details the intricate clinical course of a patient who suffered from undiagnosed spontaneous recurrent bladder ruptures for over a year and serves as a textbook example of the diagnostic complexity of urinary ascites secondary to recurrent spontaneous bladder rupture.

## CASE DESCRIPTION

The patient, a female in her sixties, initially presented to the emergency department after experiencing sudden lower abdominal pain following straining. She had multiple similar episodes before, starting eighteen months prior to the current hospital admission.

During the first episode the computed tomography scan of the abdomen had revealed free fluid in the pelvis and wall thickening of small bowel loops. Percutaneous drainage of the free fluid had yielded rusty-brown fluid with creatinine level of 67 µmol/l, comparable to serum levels. Even though uroperitoneum had already been suspected then, a definitive diagnosis could not be made. A temporary urinary catheter was inserted; she was treated for cystitis with para infectious ascites and the pain and ascites resolved. After discharge she underwent a cystoscopy, which revealed bladder trabeculation and two discrete areas of thinned mucosa with visible underlying perivesical fat, interpreted as trophic changes post-radiation. A colonoscopy was also performed and unremarkable. Urodynamics diagnosed a high capacity (>600 ml), hyposensitive bladder with no detrusor contractions, consistent with a neurogenic bladder. The recommended management consisted of prompted voiding and physiotherapy.

Further hospitalisations followed due to recurrent colicky lower abdominal pain. Exploratory laparoscopy 15 months prior to the current admission revealed fibrin and pus in the lower abdomen with growth of *Enterococcus faecalis*. She also developed a large retroperitoneal haematoma involving the right colon and duodenum, for which an exploratory laparotomy with hemicolectomy and appendectomy was performed. On another pain-episode, a computed tomography (CT) scan demonstrated massive ascites extending to the upper abdomen and marked, long-segment thickening of the jejunal wall *(Fig. 1)*.

With unexplained recurrent abdominal pain, ascites and bowel wall thickening, an inflammatory bowel disease was suspected. Further investigations including a push-enteroscopy and a hydro- magnetic resonance imaging (MRI) showed no pathological lesions, leaving the aetiology obscure. Repeated episodes of acute renal failure in the context of severe lower abdominal pain were notable, interpreted as prerenal kidney injury in the context of systemic inflammation, decreasing to baseline after inserting a temporary urinary catheter.

After eighteen months of recurrent abdominal pain, the patient presented again with the same symptoms. With all prior investigations at hand, a gastrointestinal disease or infectious cystitis seemed unlikely. Careful clinical assessment showed that she experienced suprapubic pain



***Figure 1.*** *Abdominal CT demonstrating diffuse circumferential wall thickening of bowel loops with four-quadrant ascites.*

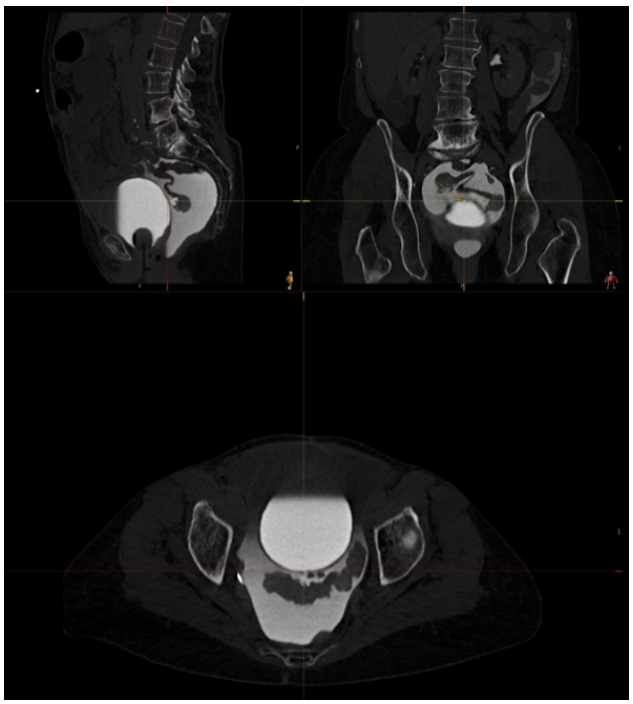


*Figure 2. CT cystography revealing contrast leakage from the urinary bladder.*

after forceful micturition, followed by temporary relief and then generalised abdominal pain. She also reported that the placement of urinary catheters often alleviated the pain. Clinically, a bladder pathology seemed to be the most probable cause.

This assumption was backed up by the past medical history. The patient reported a long-standing history of voiding difficulty, characterised by a loss of bladder sensation and the need to use manual suprapubic compressions and Valsalva manoeuvre. Her medical history was notable for FIGO stage IIb cervical carcinoma 23 years prior, treated with radical abdominal hysterectomy (Wertheim-Meigs procedure), followed by adjuvant radio chemotherapy. She also had required the placement of a tension-free vaginal tape for stress urinary incontinence and had already undergone open cystolithotomy for the removal of bladder stones, a complication indicative of chronic urinary stasis[3].

Laboratory investigations suggested acute kidney failure, as in previous episodes, with a serum creatinine peaking at 193 µmol/l on day three of admission (baseline creatinine 66 µmol/l). In contrast, cystatin C was within normal range (0.84 mg/l, estimated glomerular filtration rate 88 ml/min/1.73m$^2$). A CT-angiography again revealed voluminous four-quadrant ascites and diffuse, long-segment oedematous thickening of the small bowel and ascending colon.

With suprapubic pain, difficulty voiding, creatinine-rise and ascites without apparent liver or cardiac disease, recurrent spontaneous bladder ruptures were hypothesised. An ascitic fluid puncture was performed, which yielded yellow turbid fluid with a high serum-ascites albumin gradient of 2.7 g/dl, a low ascitic protein of 1.3 g/dl, a highly elevated ascites creatinine level of 725 µmol/l and an ascites-to-serum creatinine ratio 3.76.

CT cystography ultimately proved our hypothesis, revealing the posterior leak from the urinary bladder *(Fig. 2)*. Other investigations such as ascites cytology, urinary porphyrin-profile and C1-esterase inhibitor activity for abdominal angioedema showed no pathological results.

The patient was diagnosed with recurrent SRUB secondary to chronic radiation cystitis and neurogenic bladder dysfunction (post-Wertheim-Meigs)[4]. The urological and gynaecological consultation recommended a conservative management strategy to avoid risks associated with surgical repair in irradiated tissue. A transurethral catheter

| Parameters | Normal range | Before | After |
|---|---|---|---|
| Creatinine (µmol/l) | 45–84 | 193 | 75 |
| Urea (mmol/l) | 2.76–8.07 | 8.3 | 4.35 |
| Sodium (mmol/l) | 136–145 | 136 | 138 |
| Potassium (mmol/l) | 3.4–4.5 | 4.7 | 4 |
| Cystatin C (mg/l) | 0.61–0.95 | 0.84 | 0.78 |

*Table 1. Laboratory results before and <24 h after insertion of urinary catheter.*

| | Cirrhosis | Peritoneal carcinomatosis | Heart failure | Nephrotic syndrome | Patient |
|---|---|---|---|---|---|
| Appearance | Clear | Cloudy, turbid, mildly bloody | Clear | Clear | Yellow turbid |
| SAAG (g/dl) | ≥1.1 | <1.1 | ≥1.1 | <1.1 | 2.7 |
| Ascites protein (g/dl) | <2.5 | ≥2.5 | ≥2.5 | <2.5 | 1.3 |
| Ascites/serum creatinine ratio | <1 | <1 | <1 | <1 | 3.75 |

*Table 2. Comparison of key parameters between the most frequent causes of ascites.*

was placed for continuous drainage, resulting in the rapid resolution of abdominal pain and normalisation of serum creatinine within 24 hours *(Table 1)*.

The patient was later instructed in intermittent self-catheterisation to prevent bladder overdistension, the primary trigger for her ruptures. A follow-up cystography confirmed the sealing of the bladder wall defect. At four months follow-up, the patient remained asymptomatic, managing her bladder via self-catheterisation five to six times daily, with no recurrence of ascites or abdominal pain.

## DISCUSSION

This case represents an important example of the diagnostic challenges posed by spontaneous bladder rupture in the complex post-radiation patient.

The peritoneum is a semi-permeable membrane, richly vascularised by capillary networks, which is utilised for peritoneal dialysis in end-stage renal disease. In urinary ascites this physiology is inverted, and solutes move down their concentration gradient from the peritoneal fluid into the systemic circulation[2].

The average daily creatinine production is around 10,000 to 15,000 µmol/l. Depending on the fluid intake the concentration in the urine typically varies between 2,550 and 20,000 µmol/l, which is up to 300 times higher than in the serum. Furthermore, creatinine has a high peritoneal diffusion capacity. This is why in urinary ascites, serum creatinine often rises rapidly in the serum followed by a dramatic correction once the bladder is drained, confirming the functional integrity of the kidneys[5].

On the other hand, cystatin C is freely filtered at the glomerulus and subsequently reabsorbed and metabolised in the proximal tubule, resulting in only minimal amounts appearing in the urine (about 0.1 mg/l)[6]. In addition, it has a low peritoneal diffusion capacity. This explains, why our patient's serum cystatin C remained within the normal range.

Depending on the urine concentration and the osmotic gradient, electrolyte disturbances such as hyponatraemia and hyperkaliaemia can occur.

The serum-ascites albumin gradient (SAAG) plays a crucial role in differentiating ascites with a SAAG ≥1.1 g/dl being typical for portal-hypertensive ascites[7]. In addition, ascitic protein levels help differentiate a transudative process from an exudative origin (cut off 2.5 g/dl)[7]. Reflecting the low albumin content of urine, case reports of uroperitoneum have repeatedly reported a very high SAAG in combination with low ascitic protein levels in urinary ascites[8]. This pattern was also observed in our patient, with a SAAG of 2.7 g/dl and an ascitic protein concentration of 1.3 g/dl.

Nevertheless, the key finding in the diagnosis of urinary ascites is the ascites-to-serum creatinine ratio, with a ratio >1 being highly suggestive for uroperitoneum[9]. In this case, the ratio was 3.76 (ascites creatinine 725 µmol/l / serum creatinine 193 µmol/l) *(Table 2)*. It is worth noting that in an earlier admission, the ascites creatinine was reported as 67 µmol/l. This likely represented a period where the rupture had temporarily sealed, or the fluid sampled was predominantly inflammatory exudate rather than fresh urine.

## CONCLUSION

Spontaneous rupture of the urinary bladder (SRUB) is an important differential diagnosis in patients with acute abdomen, large-volume ascites and elevated serum creatinine, especially following a history of pelvic radiation. Urinary ascites should highly be suspected in the following constellation of laboratory parameters: Elevated serum creatinine with normal serum cystatin C, high SAAG (>1.1 g/dl) and low ascitic protein levels (<2.5 g/dl) and high ascites-to-serum creatinine ratio (>1).

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