



PSEUDO RENAL FAILURE POST ROAD TRAFFIC ACCIDENT

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ABSTRACT Blunt abdominal trauma encompasses a broad spectrum of internal injuries, amongst which bladder rupture remains relatively infrequent, occurring in less than 2% of cases. Intra-peritoneal rupture, while rare, is clinically significant—it results in urinary ascites, notable electrolyte disturbances, and azotaemia secondary to the peritoneal absorption of urinary constituents. Early identification, appropriate imaging, and rapid surgical management are critical for optimising patient outcomes. Here, we present the case of a 66-year-old male who arrived at the emergency department two days post a handlebar injury to the lower abdomen, sustained during a motor vehicle incident. The patient reported escalating abdominal pain, progressive distension, and visible ecchymosis in the suprapubic area. On examination, vital signs were stable (HR 98/min, BP 130/90 mmHg, RR 22/min, SpO₂ 98% on room air). There was marked suprapubic tenderness and fullness, raising suspicion of urinary retention. Focused Assessment with Sonography in Trauma (FAST) detected free fluid in Morrison's pouch. Laboratory studies noted hyponatremia, hyperkalaemia, and elevated creatinine—findings consistent with urinary ascites. Foley catheterisation yielded 350 mL of gross haematuria. Resuscitative measures included intravenous fluids, calcium gluconate, and insulin with dextrose for hyperkalaemia. Haemodialysis was performed due to persistent azotaemia. CT cystogram demonstrated intraperitoneal bladder rupture. The patient underwent emergency laparotomy with two-layer bladder repair and placement of an abdominal drain. Postoperative recovery was favourable: creatinine normalised, the drain was removed on postoperative day one, and the patient was discharged in stable condition. This case underscores the necessity for high clinical suspicion, targeted imaging, and aggressive metabolic correction in patients with blunt abdominal trauma and haematuria. Prompt surgical intervention remains essential for a favourable prognosis, even in the context of significant metabolic derangement.

KEYWORDS : Blunt abdominal trauma, bladder rupture, haematuria, urinary ascites, intraperitoneal injury, laparotomy

INTRODUCTION

Blunt abdominal trauma is a major contributor to morbidity and mortality in trauma settings. While injuries to solid organs—particularly the liver and spleen—are more common, bladder trauma is rare and typically associated with concomitant pelvic fractures. Intra-peritoneal bladder rupture commonly results from direct trauma to a distended bladder, predisposing to urinary ascites, electrolyte abnormalities, and azotaemia, due to the peritoneal reabsorption of urinary solutes.

Haematuria serves as a crucial clinical indicator of bladder injury in trauma patients. CT cystography remains the diagnostic modality of choice. Management of intra-peritoneal bladder rupture necessitates surgical repair, while extraperitoneal injuries may often be managed conservatively with catheter drainage.

This case highlights diagnostic and therapeutic challenges in an elderly patient with delayed presentation following blunt abdominal trauma.

Case Presentation

A 66-year-old male presented to the emergency department 48 hours post a two-wheeler accident, following direct impact from the handlebar to the lower abdomen.

Presenting Complaints

He reported progressive lower abdominal pain and distension over two days. Suprapubic ecchymosis developed on day one post-injury. There was no loss of consciousness, vomiting, seizure activity, or otorrhagia/rhinorrhagia. He remained ambulatory post-trauma.

Primary Survey

Airway: Maintained; no cervical spine tenderness.

Breathing: Spontaneous, bilateral air entry equal, no added lung sounds. RR: 22/min. SpO₂: 98% on room air.

Circulation: Radial pulse regular at 98/min, BP 130/90 mmHg. Notable suprapubic ecchymosis extending to the right iliac fossa. Abdomen distended, with suprapubic fullness/tenderness. Pelvic compression test negative. No active external bleeding.

Disability: GCS 15/15 (E4V5M6). Pupils were equal/reactive.

Exposure: Afebrile (98.6°F).

Primary Adjuncts

IV access: 18G in right metacarpal vein.

Arterial Blood Gas: pH 7.35, Na⁺ 124 mmol/L, K⁺ 6.03 mmol/L, Hb 15.1 g/dL, glucose 138 mg/dL, lactate 1.0 mmol/L.

ECG: Sinus rhythm, unremarkable.

Foley catheter (16F): 350 mL haematuria.



Figure 1: A) Suprapubic ecchymosis B) Haematuria

Secondary Survey

No pallor, icterus, pedal oedema, or lymphadenopathy.

Cardiovascular: S1/S2 normal, no murmurs.

Respiratory: Bilateral air entry equal, no added sounds.

CNS: No focal deficits.

Imaging And Clinical Progression

An extended Focused Sonographic Assessment in Trauma (eFAST) showed Free fluid visualised in the Morrisons pouch.



Figure 2: Free fluid in morrison's pouch

There was a creatinine rise from 2.1 to 8.0 mg/dL, and blood urea from 5.9 to 139 mg/dL.

Acute Management

Management in the ED prioritised stabilisation. IV 10% calcium gluconate (10 mL) for hyperkalaemia correction. This was followed by IV regular insulin (10 units) in 100 mL of 25% dextrose to promote intracellular potassium shift. Maintenance IV normal saline (50 mL/hr) was commenced. Planned for CT Cystogram and hence, IV N-acetylcysteine (1.2 g in 100 mL NS) was given.

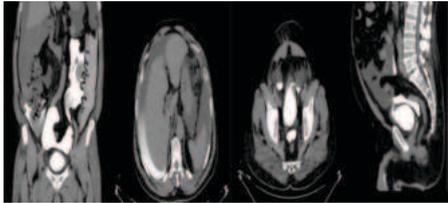


Figure 3: Bladder wall rupture

Due to refractory azotaemia (rising creatinine/urea), nephrology consultation was obtained, and the patient underwent a single session of haemodialysis preoperatively.

Surgical Management

Emergency Laparotomy: Two-layer bladder repair performed; surgical drain placed.



Figure 4: Two-layered bladder repair

Postoperatively, creatinine decreased to 2.1 mg/dL on the first postoperative day, and was further normalised to 1.0 mg/dL on postoperative day two. The drain was removed on POD1. Patient was discharged in a stable condition.

DISCUSSION

Bladder rupture following blunt abdominal trauma is rare, accounting for <2% of such cases. Intraperitoneal ruptures are less frequent than extra peritoneal injuries, typically resulting from direct force to a distended bladder, with the dome as the most susceptible site. In this patient, the mechanism was consistent with classic handlebar injury.

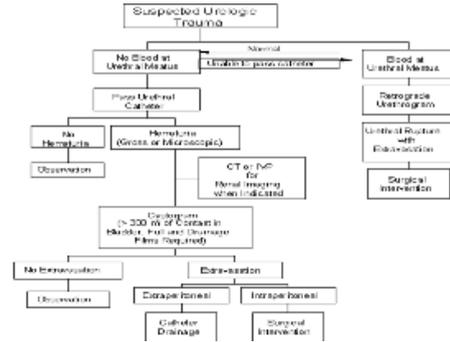
An intraperitoneal bladder rupture can cause urinary ascites resulting in creatinine-rich fluid within the abdominal cavity. The higher concentration of urinary waste products within the peritoneum compared with the blood creates a concentration gradient encouraging the reabsorption of azotaemia products back into the bloodstream, therefore resulting in the findings of electrolyte abnormalities (hyponatraemia and hyperkalaemia) and renal dysfunction (raised creatinine and an apparent reduction in estimated glomerular filtration rate (eGFR). The abnormalities seen on biochemical analysis do not represent deterioration in renal function but are in fact due to reverse dialysis across the peritoneal membrane.

It can provide a misleading picture of the patient's clinical state and leads to less-than-optimal care for the patient such as an under dosing of antibiotics. Thus, the Intraperitoneal bladder rupture leads to peritoneal absorption of urinary solutes (urea, creatinine), resulting in significant electrolyte disturbances—namely hyponatremia, hyperkalaemia, and azotaemia (“reverse auto dialysis”), most commonly named as pseudo renal failure post Road Traffic Accident (RTA). Haematuria is the most common clinical sign, but delayed presentations often manifest with abdominal distension, pain, and metabolic derangements rather than overt urinary symptoms.

eFAST identifies free intraperitoneal fluid but lacks specificity for bladder injury. CT cystography is the preferred diagnostic modality, given its high sensitivity and specificity.

Early identification and correction of life-threatening metabolic abnormalities are essential before definitive repair. Hyperkalaemia was managed with IV calcium gluconate, insulin-dextrose, and haemodialysis for refractory azotaemia. Early nephrology involvement expedited preoperative stabilisation. Definitive

management included a two-layered bladder repair and drainage, with uneventful postoperative recovery.



Clinical Takeaway: Intraperitoneal bladder rupture should be suspected in trauma patients with suprapubic pain, ecchymosis, haematuria, and unexplained azotaemia. Delayed recognition increases the risk of worsening metabolic derangement and renal dysfunction. Timely, multidisciplinary coordination between emergency medicine, surgery, and nephrology teams is critical for optimal outcomes.

Key Learning Points

Bladder rupture, while relatively rare in the context of blunt abdominal trauma, represents a critical diagnosis—particularly when haematuria is present. Intraperitoneal ruptures necessitate surgical intervention without exception; in contrast, extraperitoneal ruptures can often be managed conservatively, depending on the clinical scenario.

One must also be aware of possible electrolyte imbalances and elevated nitrogenous wastes (azotaemia) resulting from peritoneal absorption of urinary solutes, which can complicate the clinical picture.

For diagnostic accuracy, CT cystography remains the imaging modality of choice for suspected bladder injury.

Timely correction of metabolic disturbances and prompt surgical management, where indicated, are essential for optimising patient outcomes.

CONCLUSION

This case underscores the importance of maintaining a high degree of clinical suspicion for bladder injury in patients presenting with suprapubic pain, ecchymosis, and haematuria following blunt trauma. Early diagnostic imaging, prompt correction of electrolyte derangements, and timely surgical intervention are all crucial for favourable outcomes. Effective management relies on close collaboration between emergency medicine, nephrology, and surgical teams.

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