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Case Report

Delayed Presentation of Bladder Rupture Presenting as Ascites and Acute Kidney Injury

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Abstract

We report the case of a middle-aged male who presented with an acute abdomen with imaging findings suspicious for ascites and bloods suggestive of an acute kidney injury. Our case demonstrates that in-patient with acute abdomen, with imaging consistent with ascites and no history of liver disease urinary ascites and bladder perforation should be considered.

Introduction

The urinary bladder is usually protected by pelvic structures owing to its deep location in the pelvis with a low injury rate of 0.87 to 1.6% among the blunt abdominal trauma cases [1,2]. Mechanisms of bladder injuries includes direct force towards a distended bladder, shearing force secondary to pelvic fracture, penetrating trauma and iatrogenic causes [2-4]. Traumatic urinary bladder injuries can be further classified into bladder contusion, extra peritoneal rupture (60%), intraperitoneal rupture (25%), and combined intraperitoneal and extra peritoneal ruptures (6%) [1,5,6]. Extra peritoneal bladder rupture (EBR) is two-fold more common than intraperitoneal rupture, and intraperitoneal bladder rupture (IBR) often results from blunt force compressing on a distended bladder. In rare circumstances, IBR can occur spontaneously. Reported causes and risk factors of spontaneous bladder rupture (SBR) include chronic bladder disease, prolonged urinary retention, alcohol binge drinking, postpartum period and radiotherapy for pelvic malignancies [7]. We report a case of a middle-aged male without any history of trauma presenting to a rural facility with signs and symptoms consistent with acute peritonitis. CT scan findings were initially thought suggestive of perforated bowel with pneumoperitoneum and large volume free fluid, with blood analysis indicating significant acute kidney injury. On exploratory laparotomy the patient was found to have a 4cm posterior bladder wall injury. Our case highlights that in patients with acute abdomen, with imaging consistent with ascites and no history of liver disease urinary ascites and bladder perforation should be considered as prompt surgical repair reduces complications including intra-abdominal sepsis.

Case Presentation

A middle-aged Australian male presented to the emergency department with a 12-hour history of acute severe generalised abdominal pain on waking. The patient gave a history of excessive alcohol consumption the night prior to presentation however denied abdominal trauma. Patient had no significant past medical history (no known history of liver or renal disease), no medications, and surgical history was only significant for a colonoscopy one month prior to investigate per rectal bleeding which demonstrated internal haemorrhoids and a 3mm hyperplastic polyp in sigmoid colon. He had no history of intravenous drug use, no history of blood transfusions or recent tattoos. The patient was normotensive with a blood pressure of 138/81mm Hg, heart rate of 90 beats per minute, had a temperature of 37.0 degrees Celsius and a respiratory rate

of 20 breaths per minute. On abdominal examination, he was generally peritonitic with board like rigidity with generalised guarding in all four quadrants. An in-dwelling urinary catheter (IDC) was inserted prior to surgical review and the urine drained clear. Biochemical analysis of blood for electrolytes, renal and liver function on presentation were indicative of an acute kidney injury with a potassium of 5.2mmol/L (135-145mmol/L), creatinine of 271 umol/L (40-100 umol/L), eGFR 24 mL/min/1.73m^2, urea 8.6mmol/L (2.7-7.1 mmol/L) and normal liver function with a total bilirubin 13 umol/L (<20 umol/L), conjugated bilirubin <4 umol/L (<4umol/L), alkaline phosphatase 80 U/L (30-110U/L), gamma-glutamyl transferase 20 U/L (<38U/L), alanine aminotransferase 32 U/L (<34 U/L), aspartate aminotransferase 35 U/L (<31U/L) and lipase of 24 U/L (<60 U/L). Baseline eGFR was >90 mL/min/1.73m^2 and creatinine level was 82 umol/L. Full blood count demonstrated a white cell count (WCC) of 17.7 x 10^9/L (4.0 – 11.0x10^9/L), Haemoglobin of 146g/L and a C-reactive protein of 32 mg/L (<2 mg/L). A venous blood gas was performed which demonstrated an elevated lactate at 2.6 mmol/L, which normalised to 2.3 on repeat venous blood gas after fluid resuscitation. Urine analysis for microscopy demonstrated >500 leukocytes, 350 red blood cells and 30 epithelial cells, with urine myoglobin of 19. Computed tomography (CT) abdomen (after bladder catheterisation) with intravenous contrast and images in portal venous phase demonstrated pneumoperitoneum, with free air in left upper and lower abdomen, large volume free fluid with concern for perforation of bowel (Figure 1 and Figure 2).

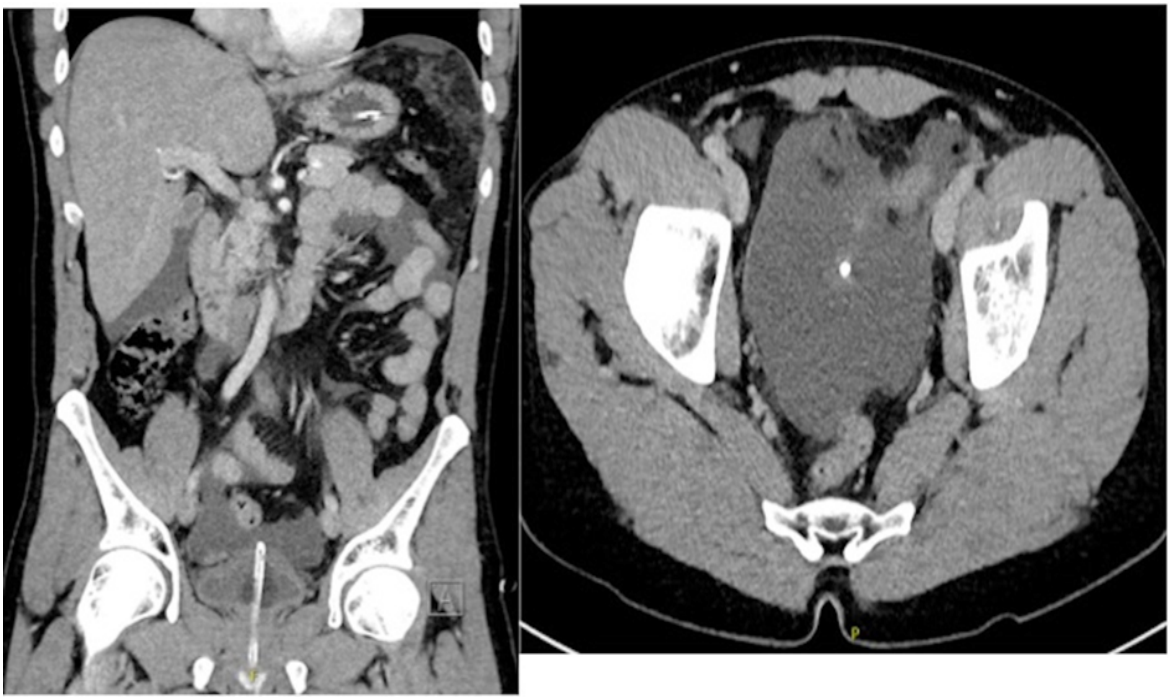

Figure 1: Coronal views of a CT abdomen pre-operatively showing a catheterised bladder with tip of catheter appear to be external to bladder dome.

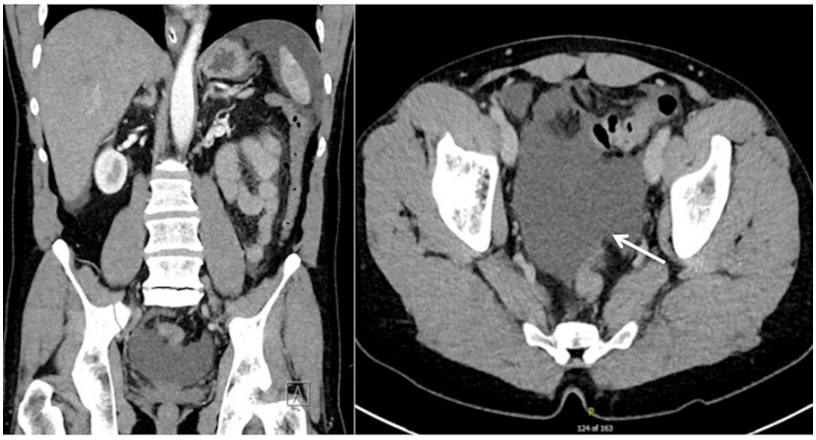

Figure 2: Axial and coronal views of a computed tomography (CT) scan Preoperative showing moderate volume ascites (arrow) without haemoperitoneum.

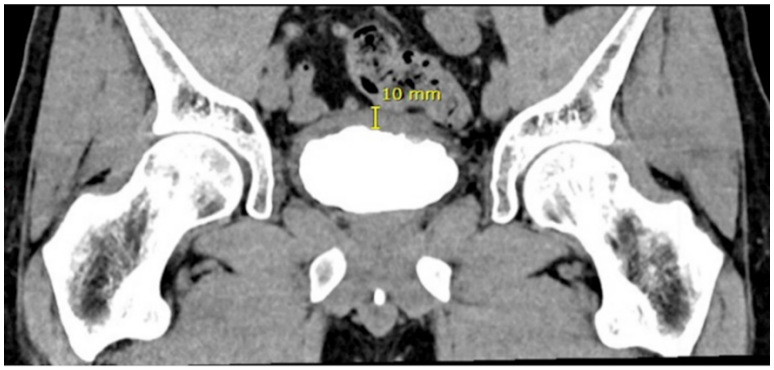

Figure 3: Coronal views of a postoperative CT Cystogram particularly showing 10mm thickening along superior bladder surface without contrast extravasation.

After aggressive fluid resuscitation in emergency, a decision was made for urgent exploratory laparotomy. A midline laparotomy was performed with initial findings of 850mls of blood tinged free fluid in the peritoneal cavity, which had macroscopic appearance of blood stained urine. All large and small bowel looked macroscopically normal. A 4cm posterior aspect bladder perforation without ureteric involvement was found with a three-layer bladder wall closure performed and a post repair leak test demonstrating no evidence of leak. A thorough abdominal washout was performed with 1L of normal saline prior to closure of the laparotomy wound. Free fluid was sent for microscopic analysis, which demonstrated 1+ leucocytes. No epithelial cells or organisms were noted. The patient was admitted to the surgical ward post operatively with patient controlled anaesthetic infusion and rectus sheath blocks for analgesia. Post operatively, the IDC was draining clear fluid. The creatinine normalised on post-operative day one (97 umol/L) and eGFR returned to baseline of >90 mL/min/1.73m^2 on day 2 post operation. The patient was discharged home on day 7 post operation with an IDC in situ. He was reviewed in the urology outpatient clinic 2-weeks post discharge with a CT cystogram prior to review (Figure 3). CT cystogram demonstrated no extravasation of contrast, with bladder wall superiourly appearing thickened at 10mm and no free fluid in pelvis (Figure 3). The patient recovered well and a trial of void and Catheter removal was performed successfully at initial post operative review. Patient was discharged back into care of his general practitioner without further urology follow up. Imaging preoperative showed free air under the diaphragm, resulting in initial suspicious for bowel perforation. Free air seen on imaging is presumed secondary to introduction of air into the bladder during catheterisation and movement of this air into the peritoneal cavity through the bladder wall defect. Despite significant urine leakage into the peritoneal cavity demonstrated on CT imaging, the patient was never anuric, the bladder defect did not involve the ureters and thus some urine draining into the bladder was drained via the indwelling catheter.

Discussion

The urinary bladder is usually protected by pelvic structures owing to its deep location in the pelvis with a low injury rate of 0.87 to 1.6% among the blunt abdominal trauma cases [1,2]. Its position slightly rises above the pelvis into the lower abdomen upon filling, hence increasing the risk of bladder injury when distended [1]. Mechanisms of bladder injuries includes direct force towards a distended bladder, shearing force secondary to pelvic fracture, penetrating trauma and iatrogenic causes [2-4]. Traumatic urinary bladder injuries can be further classified into bladder contusion, extra peritoneal rupture (60%), intraperitoneal rupture (25%), and combined intraperitoneal and extra peritoneal ruptures (6%) [1,5,6]. Bladder contusion, also known as bruised bladder, is a relatively benign condition resulting from damage to the bladder mucosa muscular is without complete loss of wall continuity [1]. Its incidence is unknown and remains as a diagnosis of exclusion as patients may remain undiagnosed or present with transient haematuria without receiving any treatment [1,3]. Extra peritoneal bladder rupture (EBR) is two-fold more common than intraperitoneal rupture and urinary leakage is contained within the per vesical space. It is usually associated with pelvic fracture when the displaced pelvic bones transmit force, which tears the anterolateral bladder wall near its base and its fascial attachments [1]. CT cryptogram is the preferred choice of imaging and recognizing the CT Fat Triangle Sign which shows extra peritoneal fluid collection helps to confirm the presence of EBR [2,8]. Uncomplicated EBR can be managed with urinary catheter drainage and a repeat cystogram on day 10 as 85% of patients recover within this time frame [3]. A second repeat cystogram needs to be done on day 21 if the former cystogram shows an ongoing leak [9]. Intraperitoneal bladder rupture (IBR) often results from blunt force compressing on a distended bladder. This increases the intravesical pressure causing intraperitoneal rupture of the bladder dome [1]. The clinical presentations of IBR include gross haematuria, abdominal pain, rigidity and difficulty voiding [10]. CT scan shows the extravasation of contrast medium from the bladder and the sentinel clot sign which refers to a clot accumulating at the injury site [11]. IBR requires immediate surgical repair with two-layer closure as delayed management may lead to intra-abdominal sepsis and death [12]. In rare circumstances, IBR can occur spontaneously. Reported causes and risk factors of spontaneous bladder rupture (SBR) include chronic bladder disease, prolonged urinary retention, alcohol binge drinking, postpartum period and radiotherapy for pelvic malignancies [7]. SBR usually occurs intraperitoneally among alcohol-intoxicated patients due to alcohol-induced diuresis and impaired sensorium removing voiding cues leading to an overly distended bladder [13]. The bladder dome becomes thinner and weaker upon distension and a further increase in intraabdominal pressure from nausea, vomiting or a fall eventually leads to SBR [7,14]. Delayed diagnosis and treatment could lead to severe complications such as urinary ascites, peritonitis and pseudo-acute kidney injury [12,15]. SBR causes urinary leakage into the peritoneal cavity, forming urinary ascites which include clinical presentations of peritonitis and rebound tenderness [7,14]. Metabolic waste products from the urine are reabsorbed across the peritoneum into the circulation based on the 'reverse auto-dialysis' concept [7,15,16]. This leads to an increase in the serum urea and creatinine levels, hyperkalemia and oliguria which mimics the presentation of acute kidney injury [7,15]. A peritoneal fluid creatinine to serum creatinine ratio of >1 should raise suspicion of intraperitoneal urinary leakage as both usually have equal creatinine level ratio [7]. IBR causes a significant elevation in blood urea nitrogen (BUN) as compared to EBR, hence a sudden increase in BUN from the baseline is an important point in diagnosing urinary ascites [7,15]. BUN also has a higher sensitivity than serum creatinine in detecting the presence of intraperitoneal urine [15]. The diagnosis of bladder rupture is easily missed hence the presence of trauma, abdominal pain and increased serum creatinine in an alcoholic should prompt suspicion of IBR.

Conclusion

Extraperitoneal bladder rupture (EBR) is two-fold more common than intraperitoneal bladder rupture (IBR). EBR often results from pelvic fractures with injuries at the anterolateral bladder wall and its fascial attachments while IBR occurs secondary to blunt force, which acts on a distended bladder, causing rupture of the bladder dome. Although rare, spontaneous bladder rupture can occur intraperitoneally post alcohol binge drinking. It is also associated with non-traumatic conditions such as chronic bladder disease, prolonged urinary retention, and radiotherapy for pelvic malignancies and in the postpartum period. This case highlights the importance of considering the diagnosis of bladder rupture in a patient who presents with an acute abdomen and abnormal serum electrolytes level. A sudden significant increase in the serum creatinine level should prompt the suspicion of IBR. The diagnosis of bladder rupture is often missed due to its clinical presentations, which mimics acute kidney injury. This case demonstrates that in-patient with acute abdomen, with imaging consistent with ascites and no history of liver disease urinary ascites and bladder perforation should be considered as a differential diagnosis. Early diagnosis and treatment are important in IBR as a delay in management can lead to intra-abdominal sepsis and death. Surgical bladder repair with two-layer closure remains the current standard management for IBR.

References

1. Guttmann I, Kerr HA (2013) Blunt bladder injury. Clin Sports Med. 32: 239-46.

2. Grünherz L, Startseva X, Kozomara-Hocke M, Barth BK, Simmen HP, et al (2017) Combined intra- and extraperitoneal urinary bladder rupture - a rare seat-belt injury: A case report. Int J Surg Case Rep. 38: 119-21.

3. Gomez RG, Ceballos L, Coburn M, Corriere JN, Dixon CM, Lobel B, et al (2004) Consensus statement on bladder injuries. BJU International. 94: 27-32.

4. Tsing V, Ng J, Wullschleger M (2017) Risk Factors Associated with Lower Urinary Tract Injuries in Traumatic Pelvic Fractures. Medical & Surgical Urology. 06.

5. Wirth GJ, Peter R, Poletti PA, Iselin CE (2010) Advances in the management of blunt traumatic bladder rupture: experience with 36 cases. BJU Int. 106: 1344-9.

6. Carroll PR, McAninch JW (1984) Major bladder trauma: mechanisms of injury and a unified method of diagnosis and repair. J Urol. 132: 254-7.

7. Zhou C, Ying X, Feng W (2015) Pseudo-acute Renal Failure due to Intraperitoneal Urine Leakage. Intern Med. 54: 1777-80.

8. Gross M, Mindelzun R, Jeffrey RB, Jr (2010) The Fat Triangle Sign: improving diagnostic accuracy of extraperitoneal bladder rupture. Abdom Imaging. 35: 253-5.

9. Urry RJ, Clarke DL, Bruce JL, Laing GL (2016) The incidence, spectrum and outcomes of traumatic bladder injuries within the Pietermaritzburg Metropolitan Trauma Service. Injury. 47: 1057-63.

10. Haddad FS, Wachtel TL (1987) Spontaneous intraperitoneal rupture of the bladder. Urol Int. 42: 467-9.

11. Shin SS, Jeong YY, Chung TW, Yoon W, Kang HK, et al (2007) The sentinel clot sign: a useful CT finding for the evaluation of intraperitoneal bladder rupture following blunt trauma. Korean J Radiol. 8: 492-7.

12. Corriere JN, Jr., Sandler CM (2006) Diagnosis and management of bladder injuries. Urol Clin North Am. 33: 67-71, vi.

13. Muneer M, Abdelrahman H, El-Menyar A, Zarour A, Awad A, et al (2015) Spontaneous Atraumatic Urinary Bladder Rupture Secondary to Alcohol Intoxication: A Case Report and Review of Literature. Am J Case Rep. 16: 778-81.

14. Kilari SK, Amancharla LY, Bodagala VL, Mulakala AJ, Bushan JV, et al (2007) Pseudo-renal failure due to intraperitoneal bladder rupture and silent subdural hematoma following a fall in an alcoholic. Int Urol Nephrol. 39: 947-9.

15. Gupta A, Biyani M, Gupta M, Saltel ME (2011) Hypercreatinemia: think beyond acute kidney injury. Can J Urol. 18: 6066-8.

16. Chennu KK, Rapur R, Vishnubotla SK (2013) Reverse autoperitoneal dialysis resulting in pseudo acute renal failure. Clin Kidney J. 6: 551.