



Case Report

Spontaneous Rupture of the Urinary Bladder: A Case Report

Mohammad Haydar^{1,2*}, Shiraz Abudahesh¹, Uriel Levinger^{1,2}

¹Internal Department C, Laniado Medical Center, Netanya, Israel

²Adelson School of Medicine at Ariel University, Israel

*Correspondence author: Mohammad Haydar, MD, Specialist Physician of Internal Medicine, Laniado Medical Center Netanya, Israel;
Email: mhaydar@laniado.org.il; hider326@gmail.com

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Abstract

Spontaneous rupture of the bladder (SRUB) is a rare occurrence globally (<1%). The most common cause of bladder rupture is trauma (96%). We present a case of a 34-year-old man with a history of heavy alcohol consumption and significant smoking history. He presented with diffuse abdominal pain and urinary retention. On blood tests, he was found to have increased creatinine. A urinary catheter was inserted and there was improvement in his kidney function. However, on removal of the catheter, his creatinine function would elevate. This occurred two times before further investigations were performed. CT cystography was performed and there was a significant amount of free intraperitoneal fluid found in the pelvis. After retrograde filling of the bladder with contrast material, a tear in the dome of the bladder was discovered. The patient underwent a surgical repair of the tear and there were no complications during surgery. In summation, spontaneous urinary bladder rupture is a rare emergency which can cause acute abdomen, oliguria, peritoneal effusion and abnormal renal function. In patients with heavy alcohol consumption and especially without an associated trauma history, physicians should consider SRUB. CT scan can help in diagnosis. Diagnostic laparoscopy or explorative laparotomy should not be delayed.

Keywords: Trauma; Spontaneous Rupture of the Bladder; Urology; High Creatinine; Surgery

Abbreviation

AKI: Acute Kidney Injury; ATN: Acute Tubular Necrosis; BUN: Blood Urea Nitrogen; CRP: C-Reactive Protein; ER: Emergency Room; GFR: Glomerular Filtrate Rate; NSAID: Non-Steroidal Anti-Inflammatory Drugs; SLE: Systemic Lupus Erythematosus; SRUB: Spontaneous Rupture of the Bladder

Introduction

Spontaneous Rupture of the Bladder (SRUB) is a rare occurrence globally (<1%). The incidence is around 1 in every 126,000 people. The most common cause of bladder rupture is trauma (96%). This occurs most often at the time the bladder is fully distended and vulnerable before micturition and can rupture from the weakest point. Extra peritoneal rupture of the bladder occurs in ~60-65% of cases and intraperitoneal rupture in 25% of cases [1]. Extra peritoneal bladder rupture most commonly presents with abdominal pain and dysuria [2]. In contrast, intraperitoneal rupture has a classic triad consisting of macroscopic hematuria, abdominal pain, difficulty or inability to void. Other presentations of bladder rupture could be non-specific, ranging from anuria with free fluid to the abdomen, abdominal distention and oliguria. The diagnosis of SRUB is challenging; therefore, most patients are misdiagnosed with acute abdomen and inflammation of the digestive system.

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34 years old, Ethiopian, kitchen worker. No underlying diseases, no previous surgeries or hospitalizations. No known sensitivity. History of heavy alcohol consumption and smoking.

First visit to the Emergency Room (ER), he presented with diffuse abdominal pain and urinary retention that started the day before his admission. Vital signs were within the normal range. In his abdominal examination, there was soft diffuse tenderness but no signs of peritoneal irritation and no tenderness when tapping the flank on both sides. The rest of the examination was without acute findings. Blood tests were taken and he was found to have leukocytosis (14 thousand K/uL). C-Reactive Protein (CRP), electrolytes, renal and liver function were normal. Alcohol levels were elevated. A urinary catheter was inserted and there was a residual volume of 100 mL of urine with a cloudy appearance, positive for leukocytes and blood. In addition, he was examined by a Urologist who found no acute urological problems.

Given the patient's abdominal pain, he was also examined by a General Surgeon. The abdomen was completely soft, mild diffuse sensitivity without signs of irritation, without hernias and had a normal rectal examination. A bedside ultrasound was performed and demonstrated a small amount of free fluid in the peritoneum. CTA abdomen and pelvis was ordered: liver and spleen with normal dimensions and density. Gallbladder preserved bile ducts of normal width. Normal pancreas, normal adrenals, normal-sized kidneys and a good and symmetrical nephrogenic effect, with no evidence of expansion in the collecting system. There is no evidence of lymphadenopathy, bowels with no abnormal findings, a catheterized and partially filled bladder, normal prostate. Peritoneal fluid in a small amount - without evidence of ruptures in parenchymatous organs in the abdomen. He was admitted to the Internal Medicine Department with a diagnosis of urinary tract infection - under antibiotic treatment with Ceftriaxone. After a few days, there was clinical and laboratory improvement. The pain passed and the patient micturated spontaneously after removing the urinary catheter - he was discharged with ciprofloxacin treatment.

The patient returned to the emergency room due to similar complaints within 24 hours of discharge (abdominal pain and difficulty urinating). Physical examination was unchanged from previous examination and vital signs were within the normal range. Abdominal x-ray demonstrated - no free air, no expansion of the intestinal loops. Laboratory tests demonstrated an increase in creatinine from 0.9 to 4.2 mg/dL. A urinary catheter was inserted with a residual 70 cc. The patient was again admitted to the Internal Medicine Department and series of investigations were performed to elucidate the onset of acute renal failure. An ultrasound examination of the kidneys and urinary tract was performed with no abnormal findings including obstruction. Urinary sediment and serological investigations were negative. In the differential diagnosis, Acute Tubular Necrosis (ATN) was suspected on the basis of drug treatment with Non-Steroidal Anti-Inflammatory Drugs (NSAID) and contrast induced nephropathy from his previous admission. Indeed, it was appropriate in terms of timing - an increase in creatinine within 24-48 hours after exposure, peaking within 3-5 days. He was treated with aggressive hydration. After treatment with fluids, the patient's creatinine improved to 0.8 and then the urinary catheter was removed. However, the patient continued to complain of abdominal pain. He was once again examined by a surgeon who felt that the abdominal pain was due to constipation. He was treated with enemas and there was improvement in his symptoms.

On repeated testing during treatment, an increase in creatinine to 4 mg/dL was observed. The following questions were raised given the increase in creatinine despite treatment with fluids and cessation of nephrotoxic medications: Is it due to the use of enemas? vasculitis? Obstructive disorder? Nephrotoxic treatment?

To help elucidate this quandary, the patient was tested for urine sediment level, serologies for Systemic Lupus Erythematosus (SLE) and vasculitis. All tests returned negative. A urinary catheter was inserted - residual urine 500 cc. Ultrasound was performed again "Normal kidneys, empty bladder contains a catheter, small to medium amount of ascites, fatty liver". After collecting all the data, it became clear that the decrease in creatinine happened at the same time as the insertion of a urinary catheter. After a multidisciplinary discussion, it was decided to perform CT cystography. We found there was a significant amount of free intraperitoneal fluid in the pelvis. After retrograde filling of the bladder with contrast material, a tear in the dome of the bladder was discovered, through which contrast material leaked into the peritoneal cavity (Fig. 1). The patient was transferred to the Urology Department and underwent Exploratory Laparotomy. About a liter of slightly cloudy yellow fluid was removed and a tear 12 cm long was found on the back wall of the urinary bladder. The margins of the wound were slightly edematous, without plaques on the intestines and the peritoneum. The tear was closed and there were no complications during surgery. The patient was successfully weaned from the catheter and discharged.

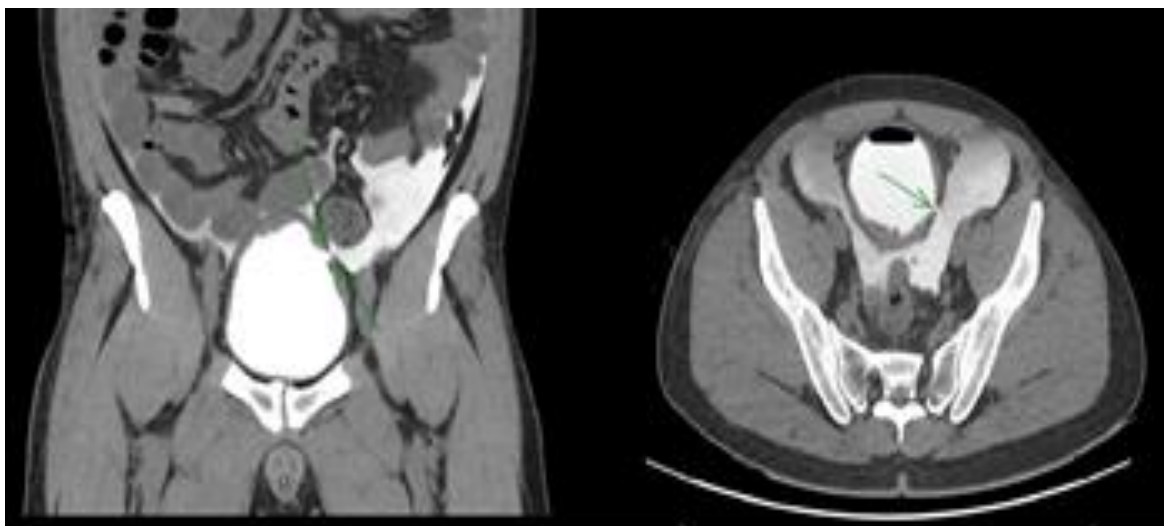


Figure 1: CT cystography demonstrated a tear in the dome of the bladder, through which contrast material leaks into the peritoneal cavity.

Discussion

Acute Kidney Injury (AKI) is characterized by a rapid decline in renal function measured by decreased Glomerular Filtrate Rate (GFR), increased serum creatinine, increased serum Blood Urea Nitrogen (BUN) and decreased urine output. Work up to uncover the etiology of AKI can involve evaluation of recent clinical history, past medical history, medication review, physical exam, serum and urine studies, imaging including renal ultrasound and renal biopsy. Our patient on the second visit to the ER was admitted due to abdominal pain and acute renal failure with high creatinine values. We followed a protocol to investigate abdominal pain and renal aggravation by taking anamnesis, in addition to relevant imaging including ultrasound. Possible differential diagnoses of renal failure include contrast induced nephropathy and acute tubular necrosis as a result of NSIAD usage. The patient received fluid as a treatment and a decrease in creatinine values began, but every time a urinary catheter was removed, we noticed an increase in the creatinine level.

During a multidisciplinary consultation including urologists, a decision to proceed to more advanced imaging was performed. Our patient underwent CT cystography that demonstrated significant amount of free intraperitoneal fluid and tear in the dome of the bladder. Our patient's medical history indicated he had consumed alcohol before symptom onset.

Patients may have altered bladder fullness sensations and abnormal behavioral responses after drinking alcohol, increasing SRUB risk. Urinary retention can occur after alcohol consumption, potentially due to rapid bladder filling caused by alcohol's diuretic effect, thinning the bladder wall and predisposing it to rupture [3].

We suspect that our patient's alcohol consumption caused the rupture. There are no specific guidelines for the treatment of SRUB. The European Association of Urology guidelines recommend that an intraperitoneal bladder rupture should always be managed by standard surgical repair because it might lead to a life-threatening condition such as abdominal sepsis and peritonitis. In contrast, conservative management can be considered for extraperitoneal bladder rupture [4,5]. Our patient underwent a laparotomy and Surgical closure of the tear as is customary to do in these cases.

Conclusion

Spontaneous urinary bladder rupture is a rare emergency which can cause acute abdomen, oliguria, peritoneal effusion and abnormal kidney function on blood test. In patients with heavy alcohol use and especially without an associated trauma history, physicians must consider SRUB. A CT cystography scan can help in diagnosis. Diagnostic laparoscopy or explorative laparotomy should not be delayed.

Conflict of Interests

The authors have no conflict of interest to declare.

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