

A case of pseudorenal failure – spontaneous rupture of the urinary bladder

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Abstract

A 64-year old woman was admitted to the hospital for severe abdominal pain and distension after gardening and chopping wood the day before. She had a medical history of superficial transitional cell carcinoma (TCC) of the urinary bladder and a laparoscopic nefro-ureterectomy for an invasive TCC of the upper urothelial tract in 2012. Clinical examination showed an acute abdomen. Laboratory analysis revealed a plasma creatinine level of 4.23 mg/dl. Computed tomography (CT) imaging of the abdomen showed hypodense free intraperitoneal fluid, suspicious for ascites. Serum creatinine level decreased to 1.8 mg/dl after placement of a urinary catheter. Laparoscopic exploration revealed a perforated ulcer in the urinary bladder dome, the intraperitoneal fluid showed abnormally high levels of urea and creatinine, confirming uroperitoneum. This case shows that uroperitoneum must be included in the differential diagnosis of patients with diffuse/acute abdominal pain, abdominal distension and elevated levels of serum creatinine, and that vigilance is indicated. (*Acta gastroenterol. belg.*, 2017, 80, 419-421).

Key words : uroperitoneum, pseudorenal failure, ascites, acute kidney insufficiency.

Introduction

Spontaneous urinary bladder rupture is a rare cause of uroperitoneum. We present a case of spontaneous rupture of the urinary bladder, presenting as acute abdominal pain with distension of the abdomen and high serum creatinine levels.

Case report

A 64-year old woman was admitted to the hospital because of acute abdominal pain and swelling after gardening and chopping wood the day before. The pain was colic-like. She had a medical history of superficial transitional cell carcinoma (TCC) of the urinary bladder and laparoscopic nefro- ureterectomy for an invasive TCC of the upper urothelial tract in 2012. Weekly mitomycin instillations were started postoperatively, but were discontinued after one month because of hand-foot syndrome. Follow up with cystoscopy six months before admission showed no recurrence of the superficial and invasive TCC lesions. Clinical examination showed a distended abdomen, painful on palpation. There were signs of peritonitis, with rebound tenderness. She didn't mention any problem with micturition. At first sight, laboratory results revealed acute kidney failure injury AKIN (Acute Kidney Injury Network) stage 3, with a serum creatinine of 4.23 mg/dl, without any electrolyte

disturbances. C-reactive protein was 3.61 mg/L (0-5 mg/L), leukocytosis was 7.82 x10⁹/L (4.5-11x10⁹ mg/L), urea was 99.9 mg/dL (15-49 mg/dL). Urgent computed tomography (CT) imaging without contrast enhancement of the abdomen showed hypodense free intraperitoneal fluid, radiologically suspicious for ascites (Fig. 1). A urinary bladder defect was not visualised. There were no signs of hydronephrosis.

The patient was hospitalized. Prerenal azotemia was suspected and IV fluid therapy and analgesia were started but it was not until placement of a urinary catheter the following morning, that serum creatinine began to decline. Two days after admission, serum creatinine level had already decreased to 1.8 mg/dl. Tumor markers (CA 125 and alpha fetoprotein) were normal.

A diagnostic laparoscopy was performed. This revealed a perforated ulcer in the urinary bladder dome with suspicion of malignant origin in view of the medical history of superficial TCC of the bladder. Biopsies were taken at the margin of the ulcer before suturing the perforation. Evaluation of the intraperitoneal fluid showed abnormally high levels of urea and creatinine, thus confirming the diagnosis of uroperitoneum.

Anatomopathological examination of the biopsies of the urinary bladder wall could not reveal any signs of dysplasia or malignancy.

The clinical condition of the patient quickly improved, with disappearance of the abdominal pain, and normalisation of serum creatinine levels.

She was discharged after ten days.

Cystoscopic evaluation six weeks after surgery showed a normal postoperative evolution with a complete healing of the ulcer. There was normal bladder mucosa without any suspicion of superficial TCC.

Discussion

Urinary bladder rupture is a rare entity, if not recognized in time, it is associated with a high morbidity and mortality. Without trauma, mortality rates

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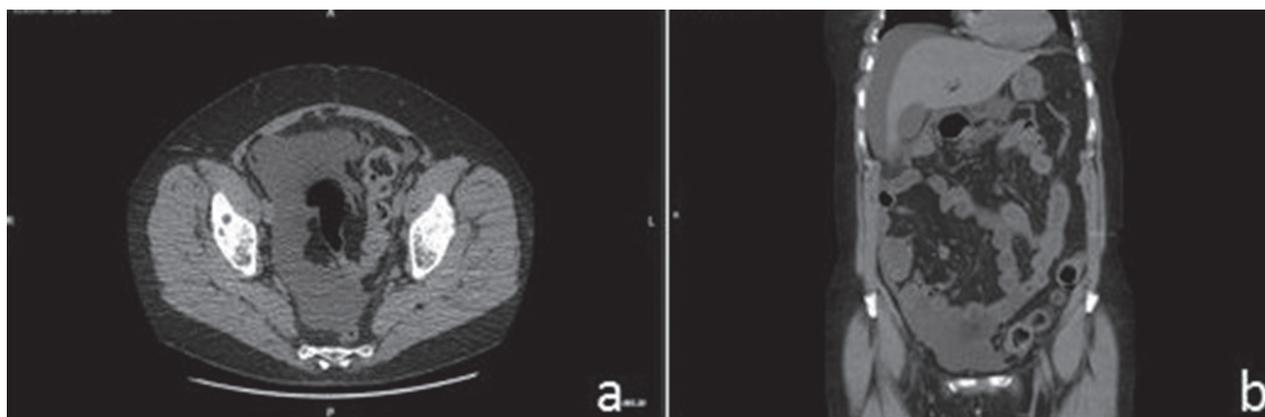


Figure 1.



Figure 2.

approaching 50% have been described. When the urinary bladder rupture is caused by a blunt trauma, the mortality rate is around 11%, lower, because of the higher index of suspicion (1). Rupture of the urinary bladder is known to be more frequent in a bladder weakened by a disease process, in our case possibly because of multiple mitomycine instillations for the treatment of TCC. In the majority of cases of urinary bladder rupture, there is a traumatic mechanism involved, in this case possibly intense physical activity, or blunt trauma involved with chopping wood, but this wasn't mentioned by the patient. It usually presents as a triad of abdominal pain, abdominal distension and urinary ascites (2,3).

There are three types of urinary bladder rupture. Extraperitoneal (most frequent 80%), intraperitoneal (15-20%) and combined. The leakage of urine into the peritoneum gives rise to symptoms such as abdominal pain, oliguria or anuria and elevated serum creatinine levels, mimicking acute kidney injury (4).

The majority of cases of spontaneous urinary bladder rupture have had underlying bladder disease or outflow obstruction. The etiologic factors that are related to bladder rupture are, among others, a history of pelvic

radiotherapy, partial cystectomy, urinary bladder tumours, bladder stones, microvascular ischemia due to diabetes, neurogenic bladder, bladder infections, bladder outflow obstruction, an indwelling urethral or suprapubic catheter, alcohol intoxication or benzodiazepine overdose (5,6).

The differential diagnosis in this case included a gynaecological problem, for example rupture of an ovarian cyst or bleeding of an ovarian mass, diverticulitis, cholecystitis, acute appendicitis and pelvic inflammatory disease.

The diagnosis of urinary bladder rupture is difficult, especially when there is no trauma involved. For the detection of bladder rupture, contrast-enhanced CT offers no advantage over non-contrast CT. CT retrograde cystography is the best radiological procedure to detect urinary bladder rupture, with a sensitivity of 100% and a specificity of 99.6%. However, it may not be readily performed in non-traumatic patients, because of lower suspicion of bladder perforation (7).

Initially, in this patient, a multifactorial aetiology of her acute kidney injury was suspected: unique left kidney, dehydration and toxic drug reaction after use

of non-steroidal anti-inflammatory drugs (NSAIDs). But because of the triad of abdominal pain, abdominal distension and elevated levels of serum creatinine, rapidly ameliorating after urinary catheterisation, an underlying uroperitoneum was suspected.

The high serum levels of urea and creatinine, can be explained by 'reverse auto dialysis' of urine across the peritoneal membrane into the blood stream. The peritoneal membrane is a semipermeable membrane, which explains the high capacity of resorption. The raise in serum creatinine is referred to as 'pseudorenal failure'. If pseudorenal failure is suspected, a peritoneal fluid analysis with measurement of creatinine and urea has to be performed. In this case, an evacuating drainage of the fluid was not performed, because of the initial suspicion of malignancy and because the amount of fluid was limited (8,9,10).

The treatment of vesical ruptures and uroperitoneum is mainly surgical and urgent. Nevertheless in our case, laparoscopy was performed only after a couple of days, because the clinical image wasn't obvious and the general condition of the patient was rather good. Conservative management of urinary bladder perforation has been described, mostly in extraperitoneal ruptures. There are a few case reports of conservative management in intraperitoneal ruptures, but these were cases of small ruptures with little clinical symptoms. Management consists of urinary antibiotic prophylaxis and continued urinary drainage for a minimum of two weeks (11).

The exact cause of urinary bladder rupture in this case is not completely clear, it could have been the physical labour (of) the day before. Also, the patient had a medical history of TCC, with laparoscopic nephro- ureterectomy, transurethral resection of superficial TCC with adjuvant Mitomycin C instillations in 2012. Cases of bladder rupture after these instillations have been described, but in all of them this occurred in the first days after

transurethral resection (12,13). It would be logical to assume that this patient had a weakened bladder wall due to the history and therapy of TCC. There were no other risk factors such as diabetes or substance abuse.

To conclude, in a patient with acute abdominal pain, abdominal distention and intraperitoneal free fluid, accompanied by acute kidney injury, it is important to think of urinary bladder perforation, especially when there hasn't been a trauma. It is also of importance to take the medical history of the patient into account and to screen for risk factors.

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