

Trauma and reconstruction

A case of spontaneous urinary bladder rupture secondary to urinary retention due to an urethral stricture



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ABSTRACT

A spontaneous, non-traumatic, urinary bladder rupture is a rare condition. We describe a case of a 23-year-old male with a spontaneous bladder rupture secondary to urinary retention, due to an urethral stricture. He presented to the emergency department with voiding difficulties, severe abdominal pain and renal failure. Abdominal ultrasound revealed large amounts of ascites. After an unsuccessful attempt to place a Foley catheter a cystoscopy was performed which showed an urethral stricture. On CT-cystogram an intra-peritoneal bladder rupture was diagnosed and the patient underwent laparoscopic repair of the bladder wall. The postoperative course was uneventful.

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1. Introduction

Urinary bladder ruptures are commonly associated with blunt abdominal trauma, but can also occur spontaneously. A spontaneous urinary bladder rupture however, is a rare condition.¹ Predisposing factors to the occurrence of spontaneous bladder rupture are associated with an increase in intra-vesical pressure or decreased strength of the bladder wall.¹ Diagnosing a spontaneous urinary bladder rupture can be challenging, even with the aid of Computed Tomography (CT). If untreated, it can lead to severe complications such as sepsis, renal failure and hyperkalaemia, and can eventually cause death.

We describe a case of a young male who presented with voiding difficulties, severe abdominal pain, renal failure and ascites on admission, which was later diagnosed as an urinary bladder rupture secondary to a bladder outlet obstruction due to an urethral stricture.

2. Case presentation

A 23-year old Caucasian male was referred to the emergency department with a 3-day history of voiding difficulties. The patient's history consisted of a motor vehicle accident two years ago for which he had been admitted to the hospital for a prolonged period of time. There was no history of previous urinary

catheterization or urethral trauma. He reported voiding difficulties and abdominal pain, both increasing in the course of the last three days. An attempt by the general practitioner to place a Foley catheter was unsuccessful. On presentation at the emergency department we saw a hemodynamically stable, afebrile patient. Physical examination revealed a painful abdomen with signs of peritonitis. A second attempt by the urologist to place a Foley catheter also proved to be unsuccessful. His blood tests revealed elevated white blood cells of $18.7 \times 10^9/L$, C-reactive protein of 32mg/L and a creatinine of 831 $\mu\text{mol/L}$, urea of 33.2mmol/L, sodium of 130mmol/L and a potassium of 5.9mmol/L. Abdominal ultrasound showed a dilated bladder (>500 cc) with a thickened wall and large amounts of ascites (Fig. 1). No hydronephrosis was observed. Since the origin of the ascites was unknown the urologist refrained from placing a suprapubic catheter. Instead, a cystoscopy was performed, which showed an urethral stricture. The stricture was dilated and a Foley catheter was successfully placed over a guidewire. After catheterization the patient's condition improved. Due to the large quantities of ascites the patient was first suspected to suffer from an abdominal malignancy. Therefore, blood was tested for leukocyte differentiation and gonadal tumour markers, which both came back negative. The fluid was aspirated and sent for testing, showing a creatinine of 11 $\mu\text{mol/L}$. During admission the patient was polyuric, producing 4.800 cc in the first 24 hours after admission. Over the course of the next two days his creatinine improved (creatinine 128 $\mu\text{mol/L}$, Urea 11.2mmol/L) and the electrolyte disturbances normalized (sodium 135mmol/L, potassium 4.6mmol/L). Also, his white blood cells and CRP normalized. CT-cystogram was done two days after admission, showing a normal

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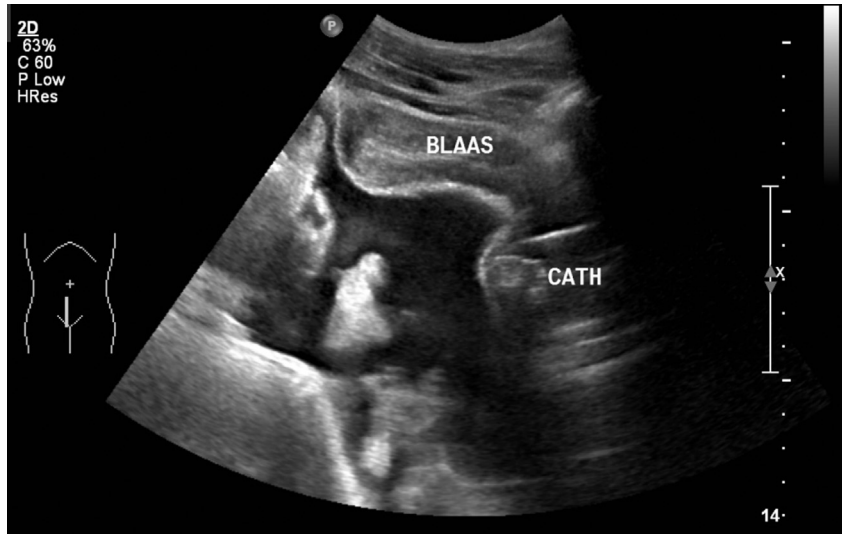


Fig. 1. Abdominal ultrasound after catheter placement showing a thickened bladder wall and large amounts of ascites.

aspect of the kidneys and a decrease of ascites. Furthermore, an interruption of the bladder wall was seen, with leakage of intra-vesical contrast, suggesting a rupture of the ventral bladder wall (Fig. 2). Therefore the patient was scheduled for a laparoscopic repair of the bladder wall the next day. After surgery the patient had an uneventful recovery and was discharged after 2 days. One week after surgery the catheter was removed, he experienced no voiding problems. Two months after surgery the patient reported excellent voiding with good results on the uroflowmetry with a Qmax of 19.4 ml/s and no postvoid residual.

3. Discussion

Urinary bladder ruptures are commonly associated with blunt abdominal trauma or iatrogenic causes like catheterization.² Patients who suffer from spontaneous ruptures usually have one or

more risk factors. Literature provides only a few case reports of patients with spontaneous bladder ruptures due to a variety of underlying conditions. Predisposing conditions are associated with increased intra-vesical pressure, such as (chronic) urinary retention due to neurologic or obstructive disorders, while other predisposing conditions are associated with a decreased strength of the bladder wall, such as diverticula, chronic inflammation, tumours and radiationcystitis.¹ Another underlying cause of spontaneous urinary bladder rupture is excessive alcohol consumption (binge drinking), which has been increasing over the last couple of years.³ In our case, an increase in intra-vesical pressure due to an obstructive disorder resulting in an urinary retention, was the cause of the rupture. A predisposing condition which decreased the strength of the bladder wall was not found.

Initial presentation of bladder ruptures is often unspecific, and may differ from mild voiding complaints with small amounts of

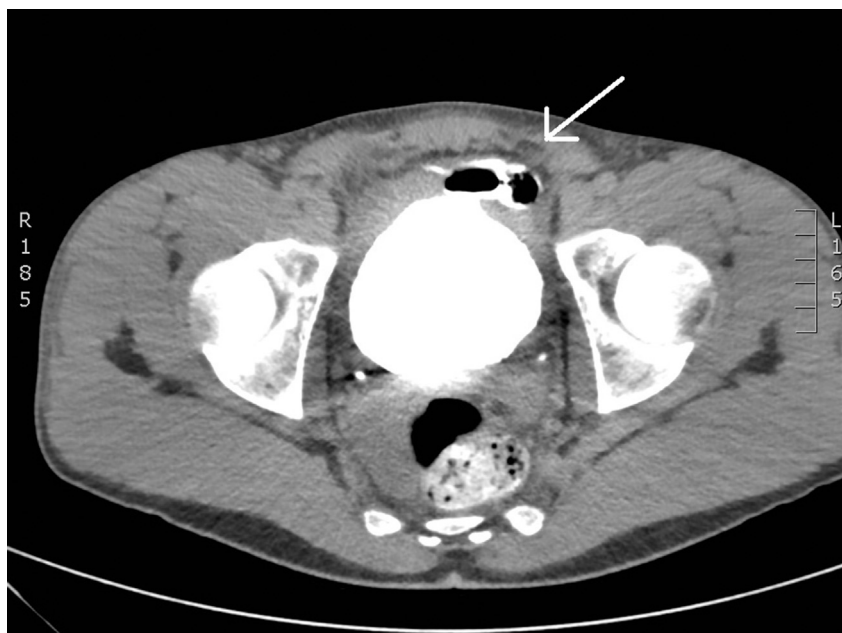


Fig. 2. CT-image showing intra-abdominal leakage of intravesical contrast due to a ventral bladder rupture.

ascites, to peritoneal irritation with increased creatinine due to reabsorption.² If a rupture exists for an extended period of time (>24 hours), urine tests may show microscopic haematuria. Furthermore, an elevated serum creatinine due to reuptake of urine creatinine through the peritoneum is commonly seen.⁴

Cystoscopy was only used to place the Foley catheter, the bladder wall was not meticulously inspected. If this would have been possible, the rupture might have been diagnosed at presentation. A cystoscopy however is not the golden standard in diagnosing a bladder rupture. Both conventional retrograde cystography and CT-cystography are currently the golden standard, whereas on ultrasound or conventional CT the diagnosis is often missed.⁵ When diagnosed, surgical intervention is required for patients with intraperitoneal ruptures. Extraperitoneal ruptures can be managed conservatively with a Foley catheter.³ Furthermore, underlying causes of the rupture may require additional treatment. Treatment of severe complications, such as kidney failure, hyperkalaemia and sepsis, has improved, resulting in a dramatic decrease in mortality compared to the '50s. However, bladder ruptures still can be fatal, especially in patients who are diagnosed at a later stage.³

4. Conclusion

Spontaneous bladder ruptures are very uncommon, and accurate diagnosis may be difficult due to the variation in complaints at initial presentation. Diagnosis in early stage is important to prevent severe complications such as sepsis and eventually death. This case highlights the fact that a bladder rupture should be suspected in

patients presenting with voiding difficulties, severe abdominal pain and renal failure. A CT-cystogram should be used to confirm the diagnosis of a bladder rupture.

Conflicts of interest

Declarations of interest: none.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at <https://doi.org/10.1016/j.eucr.2018.01.009>.

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