Silent bladder perforation

Introduction

This article presents a rare case of spontaneous intraperitoneal bladder perforation in a 70-year-old man who presented to the emergency department of a regional Australian hospital with a 12-day history of difficulty passing urine preceded by an episode of acute abdominal pain. The diagnosis was delayed because of the rarity of this condition and was only considered following the discovery of ascites on computed tomography. The patient had emergent drainage of extravasated urine and excision of a perforated bladder diverticulum.

Discussion

Spontaneous perforation of a bladder diverticulum is rare with only 14 cases reported in the literature (Leahy and Grummet, 2013). The clinical presentation varies but intraperitoneal perforation typically presents with acute onset of abdominal pain, peritonism, distension, oliguria and acute renal failure (Keeler and Sant, 1990; Itoh and Kounami, 1994; Benchekroun et al, 1997; Nishimura et al, 2000; Leahy and Grummet, 2013). Diagnosis can be delayed because it is often not considered in the differential diagnosis of an acute abdomen or oliguria and thus requires a high index of suspicion. This case demonstrates the difficulty in diagnosis, with two other provisional diagnoses considered before the final diagnosis of bladder perforation.

A bladder diverticulum is a mucosal herniation through a defect in the detrusor muscle. It does not have a muscle layer and consists of only urothelium, lamina propria and adventitia. Bladder diverticula are mostly acquired (Moreno Sierra et al, 2010), with the most common causes being bladder outlet obstruction, neuropathic bladder dysfunction, malignancy or radiotherapy (Keeler and Sant, 1990; Itoh and Kounami, 1994; Jorion and Michel, 1999; Shakeri et al, 2007). In this case the bladder diverticula are most likely a result of chronic bladder outlet obstruction from benign prostatic hyperplasia causing detrusor overactivity and high pressures within the bladder. The history of traumatic bladder perforation 30 years ago may also have contributed.

Urinary ascites from bladder perforation presents an interesting diagnostic dilemma. The gold standard for diagnosis is extravasation of contrast demonstrated with a cystogram (Aber et al, 2011). However, cystograms may not always be available in the acute setting and delayed presentation and control of the leak by an indwelling catheter may limit the role of the cystogram (as demonstrated in this case report). Biochemical analysis of a diagnostic peritoneal tap has been proposed (Ramcharan et al, 1987). An ascites:serum creatinine ratio >1.0 is highly suggestive of an intraperitoneal urinary leak. However, the sensitivity of the ascites:serum creatinine ratio may be impaired as a result of reverse autodialysis through the peritoneal membrane after the first 24 hours following perforation. Reverse autodialysis occurs when metabolic waste products (primarily urea and creatinine) from the urine are reabsorbed through the peritoneum resulting in a sudden and marked elevation of serum levels of urea and creatinine, as demonstrated by this case (Aber et al, 2011). Severe elevation of the serum creatinine level in the presence of ascites may therefore be helpful in the diagnosis of delayed presentation of bladder perforation (Aber et al, 2011).

Complications include urinary tract infection, urinary ascites, sepsis and chemical or infective peritonitis (Leahy and Grummet, 2013). Treatment of intraperitoneal bladder perforation requires emergent

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Case Report

A 70-year-old man presented to the emergency department with a 12-day history of difficulty voiding preceded by an acute episode of abdominal pain. His GP diagnosed a urinary tract infection 9 days before presentation and started him on antibiotics. Urine culture performed at that time was eventually found to be negative.

On presentation he reported mild generalized abdominal discomfort and was only able to void small amounts of urine. His history was significant for longstanding lower urinary tract symptoms and a previous traumatic bladder rupture following a severe motor vehicle accident 30 years earlier. Examination revealed a distended abdomen which was soft and non-tender. Digital rectal exam revealed an enlarged prostate. Bladder scan showed 600 ml in the bladder and an indwelling catheter was inserted. His blood tests suggested severe renal impairment with a serum creatinine of 1187 µmol/litre and urea of 37.3 mmol/litre. A diagnosis of acute kidney injury following bladder outlet obstruction was made and a joint admission under urology and nephrology was arranged. Nephrotoxic medications were ceased. Urine output over the next 12 hours was over 2 litres, consistent with a post-obstructive diuresis.

A non-contrast computed tomography scan of the abdomen and pelvis showed a large amount of intraperitoneal free fluid with no intraperitoneal air. Evidence of chronic bladder outlet obstruction was present with diffuse mural thickening of the bladder wall and the presence of two large bladder diverticula. Bladder perforation was suspected and a computed tomography cystogram was performed, which did not demonstrate active contrast extravasation.

On rigid cystoscopy two large bladder diverticula and encroaching lateral lobes of the prostate were demonstrated. The larger diverticulum appeared to have been the source of intraperitoneal perforation. A laparotomy was then performed to drain the peritoneal cavity. The two diverticula were excised and an open transvesical prostatectomy was performed. A suprapubic and 3-way indwelling catheter were inserted for continuous bladder irrigation.

His creatinine level returned to normal by day 3 of his admission. Following his surgery he was discharged to a private hospital for postoperative rehabilitation. Pathology specimens from the surgery were consistent with bladder diverticula with no malignancy and benign prostatic hyperplasia.

evacuation of intraperitoneal urine and repair of the bladder defect. Extraperitoneal perforation may be managed conservatively with bladder drainage via an indwelling or suprapubic catheter. BJHM

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LEARNING POINTS

- A thick-walled bladder with diverticula can indicate detrusor overactivity secondary to bladder outlet obstruction. This can lead to high voiding pressures within the bladder resulting in the formation of diverticula that may be prone to perforation.
- Diagnosis of spontaneous bladder perforation is often delayed and requires a high index of suspicion.
- A history of lower urinary tract symptoms, combined with acute onset abdominal pain, oliguria, raised serum creatinine levels and ascites, should prompt one to consider spontaneous bladder perforation.
- Cystogram is the gold standard for diagnosis of bladder perforation but availability and delayed presentation may limit its utility.
- Intraperitoneal bladder rupture requires surgical intervention while extraperitoneal bladder rupture may be managed conservatively with bladder drainage via an indwelling or suprapubic catheter and rarely requires surgical repair.

Calcified pericardial haematoma causing heart failure

A 67-year-old woman was admitted with shortness of breath. Physical examination revealed a third heart sound, engorged neck veins, hepatomegaly and pedal oedema.

Chest X-ray (*Figure 1*) showed dense pericardial calcification. Echocardiogram showed an ovoid mass impinging on the dilated right ventricle with pulmonary hypertension. There was no pericardial effusion or any respiratory variation in the mitral valve inflow velocities.

Computed tomogram of the chest (*Figure 2*) showed a 5 x 3 cm ovoid area of calcification with central soft tissue density pressing upon the right ventricle, most likely secondary to pericardial haematoma in the past.

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Traumatic bleeding into the pericardium can initiate a process of inflammation, calcification and scarring that may eventually produce pericardial constriction (Manhas et al, 2008).

Post-traumatic formation of a large, organized, calcified haematoma is rare. In a literature review (Brown and Ivey, 1996) the time from injury to presentation with symptoms ranged from 3 to 20 years. BJHM

Figure 1. Chest X-ray showing dense pericardial calcification.

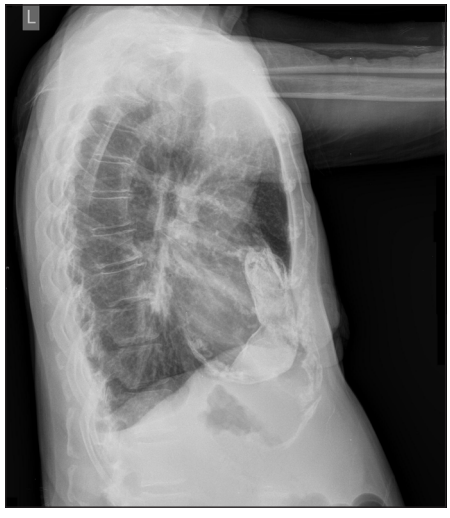

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Figure 2. Computed tomogram of the chest showing an ovoid calcified mass impinging on the right ventricle.