

Two cases of intraperitoneal bladder rupture following vaginal delivery

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ABSTRACT

We report two rare cases of isolated spontaneous intraperitoneal bladder rupture following normal vaginal delivery without concomitant uterine rupture. Two primigravidas, aged 32 years and 34 years, presented following vaginal delivery with abdominal distension and acute renal failure. The initial diagnosis in both cases was severe urosepsis. Definitive diagnosis was achieved after 48 hours. Comparison was made with two of the cases found in the literature and all four cases were reviewed. Laparotomy and bladder repair were performed in all patients with no adverse outcome reported. Two of them were diagnosed only during laparotomy. Key diagnostic clinical features were acute renal failure, new-onset ascites and bowel ileus with sepsis. This rare but life-threatening condition is often diagnosed late. Prompt laparotomy and bladder repair are necessary to prevent an adverse outcome.

Keywords: bladder perforation, bladder repair, obstetric complication, spontaneous bladder rupture, urological emergency, vaginal delivery complication

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INTRODUCTION

Isolated intraperitoneal bladder rupture following vaginal delivery is a rare urological emergency. We report two such cases, and comparison was made with the two previously-reported cases. Diagnostic features are discussed and appropriate imaging options are suggested.

CASE REPORTS

Case 1

The first patient was a 32-year-old primigravida, who presented to her obstetrician with acute lower abdominal pain and distension five days following a vacuum-assisted vaginal delivery. The baby's birth weight was 2,850 g. The delivery was described to be uneventful and labour was not prolonged. Her obstetrician performed a pelvic ultrasonography which revealed gross ascites.



Fig. 1 Axial CT image shows perforation of the bladder dome (arrowed) with intraperitoneal extravasation of the contrast agent.

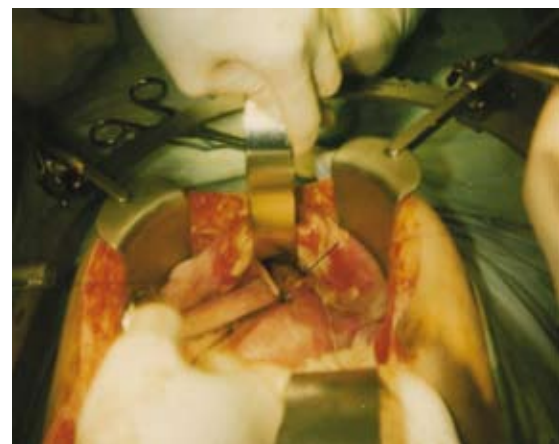


Fig. 2 Operative photograph shows a 10-cm bladder perforation (arrowed) following normal vaginal delivery.

Upon catheterisation, 1.5 L of purulent urine was drained from the bladder. She was transferred urgently to an acute general hospital for further management. At presentation, she was afebrile and her abdomen was grossly distended. Her suprapubic region was warm, erythematous with tenderness on palpation. The Foley catheter was draining lightly-bloodstained urine. Lochia was non-offensive. White cell count was $12.1 \times 10^9/L$. Serum sodium was 132 mmol/L, potassium was 5.3

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Table I. Comparison of four patients with intraperitoneal bladder rupture following vaginal delivery.

	Kibel et al ⁽¹⁾	Kekre et al ⁽²⁾	Png et al [*]	
			Case 1	Case 2
Age (years)	28	23	32	34
Days postpartum	3	9	5	2
Previous caesarean section	No	Yes	No	No
No. of previous vaginal delivery	0	Not available	0	0
Length of labour (hrs)	10	1 (2nd stage)	Not available	0.5 (2nd stage)
Birth weight (g)	4,200	2,600	2,850	2,685
Assisted delivery	Vacuum	Forceps	Vacuum	Nil
Catheterised during labour	No	Not available	Not available	Yes
Abdominal distension	Yes	No	Yes	Yes
Abdominal tenderness	Yes	Yes	Yes	Yes
Bowel ileus	Not available	Yes	Yes	Yes
Serum urea (mmol/L)	31.8	24.3	24.1	15.8
Serum creatinine (umol/L)	910	530	536	433
Ascitic fluid creatinine (umol/L)	2,743	Not available	Not available	Not available
Intensive care support	Yes	Yes	No	Yes
Site of perforation	Posterior wall	Fundus	Dome	Dome
Mode of diagnosis	Cystogram	Laparotomy	CT urogram	Laparotomy

* Current study

mmol/L, creatinine 536 umol/L and urea 24.1 mmol/L. Arterial blood gases showed pH of 7.36, pCO₂ 27 mmHg, bicarbonate 15 mmol/L and base excess was -9 mmol/L.

The initial diagnosis was “severe cystitis with acute renal failure”. She was hydrated and empirical intravenous antibiotics were started. Contrast-enhanced computer tomography (CT) was withheld due to the renal impairment. With intravenous hydration, creatinine normalised to 73 umol/L after 24 hours. CT was done eventually after 48 hours using intravenous and oral contrast. An intraperitoneal perforation of the bladder dome was detected, showing extravasation of contrast into the peritoneal cavity (Fig. 1). At exploratory laparotomy, there were large amounts of fibrinous uroascites. The bladder wall was thinned out with areas of necrosis over the dome and posterior wall. There were two 1-cm perforations in the dome and posterior wall. The peritoneal cavity was lavaged, areas of bladder necrosis excised and the defects were closed primarily in two layers. The bladder was drained via urethral and suprapubic catheters. Recovery was uneventful and the urethral catheter was removed prior to discharge. CT cystogram after four weeks showed no urinary leak and the suprapubic catheter was removed.

Case 2

The second patient was a 34-year-old primgravida who delivered a 2,685 g baby girl following a normal vaginal delivery. The second stage was not prolonged and took 30 minutes. She was catheterised before labour and a median episiotomy was made during delivery. Antenatally, she had preeclampsia, but blood pressure was well-controlled with no proteinuria. Two days after delivery, while still in the obstetric ward, she developed

fever, vomiting and abdominal pain. Her abdomen was distended and tender in the suprapubic region. White cell count was $23.9 \times 10^9/L$. Serum creatinine was 433 umol/L, potassium 5.1 mmol/L, sodium 131 mmol/L and urea 15.8 mmol/L. There was significant pyuria on urinalysis. Abdominal radiographs showed dilated small bowel loops. The working diagnosis was that of “urosepsis causing acute renal failure and paralytic ileus”. On the fourth day, the patient deteriorated and developed tachypnoea and tachycardia, requiring monitoring in the intensive care unit.

A general surgical consult was made and contrast-enhanced CT was done. Free fluid was found in the abdomen, with fat stranding and inflammatory changes in the pelvis. A pocket of gas was seen in the lower abdomen, raising the possibility of a perforated viscus. At exploratory laparotomy, the abdomen was found to have purulent uroascites and there was a 10-cm perforation of the bladder dome (Fig. 2). The bladder was repaired in two layers and drained via suprapubic and urethral catheters. A cystogram after ten days showed no urinary leak and the suprapubic catheter was removed.

DISCUSSION

Bladder rupture in the puerperium is commonly associated with concomitant uterine rupture in the obstetric setting. Isolated intraperitoneal bladder rupture from a vaginal delivery is extremely rare. Of the handful of cases reported in the literature, only two full-text details were available for review. Kibel et al reported the first case in 1995, in which the initial diagnosis was thought to be “sepsis secondary to uterine rupture with renal failure on the bases of sepsis and nonsteroidal anti-inflammatory drug use”.⁽¹⁾ Non-contrast CT was not useful for diagnosing the perforation. Diagnosis was

made only during subsequent cystogram. The other case reported by Kekre et al was diagnosed as "peritonitis with septicaemia".⁽²⁾ Paracentesis was done for qualitative analysis, but fluid creatinine was not reported. No cross-sectional imaging was done. Bladder perforation was detected only during laparotomy. The diagnosis of spontaneous bladder rupture in the puerperium is therefore often delayed. Two of these cases were diagnosed only during exploratory laparotomy.

The aetiology of this condition is multifactorial. Sustained pressure of the foetal head against the intraperitoneal portion of the bladder during forceful uterine contractions may cause pressure necrosis of the bladder dome. This is more likely if the patient is not catheterised, resulting in a distended bladder during labour. Other contributory factors include prolonged second stage and high birth weight babies. We therefore recommend that the bladder be catheterised or drained before labour.

Table 1 compares some of the important clinical and biochemical features in all four cases. It can be seen that there was no clear identifiable factor which predisposed to this condition. Even in the most seemingly straightforward case, as in our second patient (catheterised bladder, 30-minute second stage, 2,685 g baby, unassisted delivery), bladder perforation may still occur. A high index of suspicion is therefore required. Clinical features common to all four cases were: (1) presence of urinary ascites; (2) bowel ileus; and (3) acute renal failure secondary to systemic reabsorption of urea and creatinine. With renal shutdown, life-threatening hyperkalaemia and metabolic acidosis ensued.⁽³⁾ Three out of four patients required intensive care support prior to definitive diagnosis and laparotomy.

The ideal choice of imaging should be a static retrograde cystogram with contrast instilled via a Foley catheter.⁽⁴⁾ A contrast-distended bladder is required to obtain a definitive diagnosis of bladder rupture. A basic intravenous contrast-enhanced CT without a bladder phase will miss the perforation, as was the case in our second patient. Even a contrast-enhanced CT with an excretory phase (CT urogram) may miss a small bladder rupture, as a blood clot or omentum may temporarily seal a small perforation. The presence of acute renal failure also precludes the use of intravenous (IV) contrast. We therefore suggest using non-IV contrast CT in conjunction with retrograde cystogram (non-IV contrast CT cystogram), looking for intraperitoneal extravasation

of contrast. This provides the benefit of cross-sectional images in addition to the ability to distend the bladder to detect small perforations while avoiding contrast nephrotoxicity.

However, it must be emphasised that the approach to an acute surgical abdomen is always based on clinical findings rather than depending on radiological proof. Decision for laparotomy, in the presence of signs of peritonitis, should not be delayed. Imaging should be reserved only for equivocal cases. Laparotomy is required for peritoneal lavage, excision of devitalised bladder tissue and primary repair of the bladder perforation.⁽⁵⁾ The bladder should be drained postoperatively via urethral and/or suprapubic catheters. Dual drainage allows flushing to be done via either catheter to remove any postoperative blood clot. This prevents clot retention which will compromise the repair. The urethral catheter can be removed once patient is fit to ambulate a few days after surgery. A cystogram is done ten days after surgery to check the integrity of the repair. The suprapubic catheter is removed if the cystogram showed no leakage of contrast.

Unlike bladder perforation occurring in blunt trauma victims, there is no associated visceral injury in postpartum bladder perforation. In addition, as these patients are all young women in the childbearing age, there is almost no mortality if the condition is treated early, as all four patients showed good postoperative outcomes. It will be a pity that such a highly treatable condition should go unnoticed at the initial presentation. The presence of acute renal failure, ascites and bowel ileus should prompt the physician to this rare complication. It is hoped that this collection of cases would enhance the awareness of this potentially life-threatening yet treatable urological emergency.

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