Spontaneous rupture of urinary bladder in second trimester of pregnancy

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Citation

Abstract
A rare case of spontaneous rupture of urinary bladder in the second trimester of pregnancy is described. This report highlights the rarity of this complication and its diagnostic challenge. The patient presented with acute abdominal pain, voiding difficulty and biochemical features of renal failure. Four days prior to her presentation, she was treated empirically as urinary infection when she complained of retention like symptoms.

At laparotomy, around 3 liters of urine found in abdominal cavity with a 2 cm hole in the vault of bladder. TB culture was negative and bladder biopsy showed non specific inflammation.

Laparotomy, bladder repair with antibiotics cover and indwelling catheter constitutes the basis of a successful outcome.

CASE REPORT
We report a spontaneous intraperitoneal rupture of urinary bladder in early second trimester of pregnancy. Only two similar cases have been reported in the literature [1,2].

The patient was a 36 years old Indian lady, in her fourth pregnancy at 15th weeks of gestation. All her three pregnancies were uncomplicated term vaginal deliveries. She was diagnosed with hypothyroidism and was on thyroxine 150mcg daily.

She was referred from her GP as an emergency with generalised abdominal pain of four days duration which increased in severity that morning. This was associated with low backach. There was no history of trauma or vaginal bleeding.

A single live fetus was confirmed at 10 weeks gestation by ultrasound.

Four days prior to her admission, she was seen in accident and emergency with abdominal pain and voiding difficulty which was treated as urinary infection and oral antibiotics commenced. She continued to have hesitancy, feeling of desire to void but passing only small amounts of urine.

On examination, she was conscious and in pain, blood pressure: 100/40; pulse rate was 86bpm and she was hypothermic (Temp 34.9°C)

The abdomen was distended, tympanic with guarding. Vaginal examination revealed a tender fullness in pouch of Douglas and cervix was closed and directed anteriorly.

Abdominal ultrasound showed large amount of fluid in her abdominal cavity.

The patient was catheterised easily and 200ml clear urine drained. A provisional diagnosis of intraperitoneal haemorrhage was made and patient was resuscitated and prepared for emergency laparotomy.

At laparotomy, the peritoneal cavity was found to contain large amount of clear yellow fluid (2.5 liters). The uterus was deeply retroverted in the pelvis and both tubes and ovaries looked normal. Surprisingly, a hole was found in the vault of urinary bladder around 2 cm in diameter with ragged edges. There were no signs of trauma in other abdominal organs. Bladder perforation was confirmed by methylene blue dye test from urethra.

Bladder repair was completed by the urologist and urogynaecologist. The rent was trimmed and closed in two layers 3/0 PDS suture. Omentum patch was used to cover the repair and a tube drain was left in the vesicouterine pouch. Intravenous antibiotics including gentamicin, cephuroxime and metronidazole were commenced for 48 hours then oral antibiotics for ten days. Indwelling Foley catheter (16F) was inserted and to stay for the duration of pregnancy.
Urine cytology showed no abnormality and was negative for TB and conventional cultures.

Blood Investigations showed biochemical picture of acute renal impairment. Urea was 8.5mmol/L (2.5-7.5) and creatinine 362umol/L (60-129), Albumin 43(35-50), Hb 10.2 gm/dl, Sodium 133mmol/L, Potassium 4.5 mmol/L.

The following day her kidney function recovered (Urea: 2.6, creatinine: 65) but Albumin dropped to 17 g/L.

Bladder wall histology showed acute and chronic inflammation, oedema and congestion. There was extensive denudation of the urothelium with only basal layers remaining at places which shows mild reactive atypia. The features were consistent with biopsy from adjacent to a perforation/hole. There was no evidence of vasculitis, granulomata, carcinoma in situ or invasive neoplasia in the biopsy.

The patient had an uneventful postoperative course and showing good recovery clinically and biochemically. The pregnancy is ongoing smoothly and continued on long term catheter which agreed to stay till after delivery.

DISCUSSION

Spontaneous rupture of urinary bladder in pregnancy is a rare occurrence. It is defined as rupture of the urinary bladder into the peritoneal cavity or pelvic cellular tissue without a history of trauma [3]. In pregnancy, this clinical emergency is even rarer. It is more often intraperitoneal than extraperitoneal.

Several causes have been implicated including bladder tumors, post radiotherapy, chronic infective diseases and necrotising cystitis [4]. Serious maternal bladder injury at the time of uterine rupture remains a risk of attempted vaginal delivery after prior caesarean section.

The incidence of spontaneous bladder rupture has been reported as 1 in 126 000 hospital admissions[3]. Only two similar cases have been reported by Chisholm[1] and Shroff et al[2]. The oldest review of literature by Stone in 1931, forty two cases of spontaneous bladder rupture were found[5]. The five cases of extraperitoneal rupture all reported in men.

Reasons or associated factors for recurrent rupture of urinary bladder reported in the literature were necrotising cystitis [4], alcohol abuse [4] and radiation cystitis following radiotherapy for cancer of the cervix [4].

Outside pregnancy, majority of cases were reported after the age of 50 years. This is clearly explained based on the commonest aetiology including bladder cancer, radiation cystitis and neurogenic bladder dysfunction.

Intraperitoneal bladder rupture have been classified into two major groups[4] depends on whether the rupture was due to a lesion of the bladder wall or caused by overdistension of the bladder from retention of urine.

The most common lesion in the first group is tuberculosis. Retention of urine can result from three reasons. First, neurological disorders like tabes dorsalis, second reason is urethral obstruction mainly in women from impacted gravid uterus or fibroids and third reason is ‘miscellaneous’ factors as puerperal and postoperative retention.

Bastable et al[5] demonstrated that the vault of the bladder is its weakest part. This has been confirmed by experimental rupture of the bladder by distension in the cadaver.

It is quite difficult to explain the reasons behind bladder rupture in our case. The patient presented with history of voiding difficulty a few days prior to her acute presentation with abdominal pain. At that time urine retention most have been developed most likely due to uterine retroversion. Retroversion of the uterus is common and is found to be the normal uterine position in about 20% of all women. If spontaneous displacement does not occur, fundal impaction may be manifest by 14-16 weeks. This had led to retention with overflow incontinence. Subsequently, this may have led to rupture of the bladder in its weakest part, the fundus.

The patient later presented with symptoms of acute abdomen due to urine peritonitis and biochemical features of kidney impairment (creatinine was 362umol/L). Urine leak into peritoneal cavity led to shrinkage of the bladder and possible disimpaction of the uterus therefore easy catheterisation.

The clinical features of spontaneous rupture of bladder are diverse. A history of unexplained urinary tract symptoms prior to the onset of the acute event is common to most of these patients. Patients with ruptured bladder present commonly with overt symptoms and signs of peritonitis. Due to its rarity, the diagnosis of this condition is difficult. Diagnosis is usually made at laparotomy.

The most common symptoms are acute abdominal pain associated with guarding and rigidity and voiding difficulty [4]. If they present after 24 hours, these patients have uraemia
Spontaneous rupture of urinary bladder in second trimester of pregnancy

and acidosis on investigations. The delay allows significant peritoneal reabsorption of urea and creatinine, which masqueraded as ‘acute renal failure’ on biochemical testing. Cystography has been recommended [4] although none of the reported cases had this investigation prior to laparotomy.

This diagnostic challenge has been highlighted by Mokoena et al in his review of forty-four patients, of mean age 33·3 years, over a period of 7 years [4]. The mean delay between an identifiable incident or presentation and diagnosis was 5.4 days. The mean admission or preoperative levels of blood urea and creatinine were raised to 19.6 mmol/1 and 362 mol/1 respectively in those with a delayed diagnosis. The diagnosis was made by voiding cystourethrography in 36 patients and by laparotomy in eight.

In our case, the diagnosis was made at laparotomy. A cystogram was not indicated due to pregnancy. Cystogram as a diagnostic tool, however, has been used successfully postpartum [4].

Intraperitoneal bladder rupture will always require open repair, while extraperitoneal injuries in traumatic cases can be managed with catheter drainage alone in a majority of cases, with some notable absolute exceptions.

In pregnancy, surgical repair of bladder and long term catheter drainage are the mainstay of treatment. Biopsy of bladder wall is essential to exclude infective aetiology or rare cases of bladder cancer [10]. After two-layer closure of bladder injuries, most patients will recover without complications. Urinary frequency, which is common after these injuries, should resolve after two months in the majority [11].

In the case reported, the novel approach was to leave the urethral catheter for the duration of pregnancy and to deliver by elective caesarean section. This aimed mainly to rest the bladder, maintain the repair and reduce the risk of recurrence due to possible inherent bladder weakness which played a role in this unusual complication.

References

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