



Case Report

A rare case of spontaneous intraperitoneal rupture of urinary bladder after normal delivery

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ABSTRACT

We report an uncommon case of postpartum women, who presented to us 20 days after a normal delivery with anuria, abdominal pain & distension, huge ascites and renal failure. Which on evaluation and emergency laparotomy was found to be due an intraperitoneal tear of bladder probably that has happened during a vaginal delivery with a full bladder. Purulent urine in the peritoneal cavity was suctioned, a thorough lavage was done and the tear in the dome of the bladder was repaired with vicryl 3.0 in 2 layer followed which she was kept on continuous drainage of bladder for 3 wks. This is an absolutely preventable condition, provided proper care was taken in second stage by emptying the bladder. High index of suspicion and prompt diagnosis could have reduced the suffering, psychological trauma, financial burden and most importantly the morbidity in this case.

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1. Introduction

The first case of bladder rupture is reported in 1995 by Kibel AS et al. It is not uncommon to have bladder rupture in patients with and underlying diseases bladder. But spontaneous rupture during labour or in the immediate postpartum period is very rare.¹⁻⁶ So term "spontaneous rupture of bladder" can be used only when there is a bladder rupture/tear without an antecedent history of trauma or known bladder pathology, the incidence of which is found to be 1:126000.¹⁻³

The clinical presentation is usually as that of an acute abdomen. It may be associated with suprapubic pain, hematuria and anuria. Surprisingly, few may not have any symptoms and may have a late and unusual presentation which may even complicate the picture and delay the diagnosis.⁴ So, a detailed history of intrapartum and postpartum events and a high index of suspicion is needed

from the side of treating doctor as the condition is an emergency.⁴ A multidisciplinary team approach including an obstetrician, surgeon and urologist, and an emergency laparotomy with thorough lavage, repair of tear, bladder drainage and supportive management with antibiotics and analgesics would reduce the morbidity.^{7,8}

2. Case Report

A 25yr old lady(G2A1), on her 20th postpartum day after a normal delivery elsewhere was admitted with pain abdomen, fever, anuria, vomittings and abdominal distension. Her antenatal period was uneventful, with no complaints related to urination. Was in latent labour for 24 hrs, later induction of labour was done with PGE1, labour lasted for 8 hrs, and delivered a male baby, wt 2.5kg & APGAR score 8. After 24 hrs she was discharged home in good condition.

She was asymptomatic for 3 days after delivery. On 4th postpartum day, she had difficulty in voiding with which

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she went to a clinic, where her bladder was catheterised. Following which she developed abdominal pain, vomitings, distension and fever with chills & rigors which worsened gradually. Was treated with antibiotics elsewhere with which there was no improvement, hence referred to our hospital for multidisciplinary care.

On admission, she looked sick, with pallor, and was febrile with temp of 101f, pulse rate was 150 bpm and blood pressure was 120/90. Per abdomen examination revealed - distension with guarding, generalized and rebound tenderness and sluggish bowel sounds, uterus was sub-involved and was of 14 weeks size. On Vaginal examination, cervix was health and no signs of infection, swab taken. There was no history of previous blunt trauma to abdomen. Urinary catheter was changed & was found to be blocked with thick pus and plaques & 500ml of purulent urine was drained. Was shifted to ICU, investigations were sent, a working diagnosis of sepsis with UTI/ peritonitis was made and was started on empirical broad spectrum antibiotics. Based on the culture sensitivity report the antibiotics were changed inspite of which there was no clinical improvement.

Ultrasound scan abdomen and pelvis showed a bulky uterus, bilateral ovaries were normal, foley's bulb in situ and massive ascites. Ultrasound guided ascitic fluid drainage was done, sent for analysis and culture sensitivity.

Her investigations were as below:

Hb-11gm %, WBC - 10,100/cc with predominant leucocytes.

Her renal function tests and serum electrolytes were abnormal with blood urea and serum creatinine - 28 mg/dl and 2.3 mg/dl respectively; serum sodium and potassium - 140 meq/l and 7.1 meq/l respectively.

Pt/aptt & INR, viral markers, ECG & 2D-echo, LFT 's(except LDH-530), x-ray chest and erect abdomen were normal

Urinalysis showed microhaematuria with plenty of pus cells.

A CT (contrast enhanced) scan of the abdomen did not reveal any abnormality, except gross ascites. Arterial blood gas showed a PH of 7.43, PCO₂ - 32.60 mm Hg, PO₂-92.7, HCO₃ 20.9 mmol /L and a base excess of 3.4mmol/l. Ascitic fluid was sent to biochemistry and it was confirmed to be urine. Creatinine content of the drained fluid was 5.8 mg/dl, ascitic fluid for ADA was normal.

HVS showed E*+.coli, urine C/S showed enterococcus, blood and ascitic fluid showed no growth */

On suspicion of bladder rupture, plan for exploratory laparotomy was made. Intraoperatively, about 1.5 L of purulent fluid (urine) was suctioned. The uterus was subinvolved and there was no evidence of perforation. About a 2mm laceration was identified on the bladder dome (intraperitoneal rupture), which was sealed by pus/plaque partially and thus preventing the urine to escape into the

peritoneal cavity (Figures 1 and 2). The edges of the rent were freshened & bladder was repaired in two layers with vicryl no 3.0. A flap of omentum was mobilized and grafted on the repaired site. A suprapubic cystostomy was done away from the injury and a separate bladder draining catheter was inserted through urethra. Abdomen closed after leaving an intraperitoneal drain.

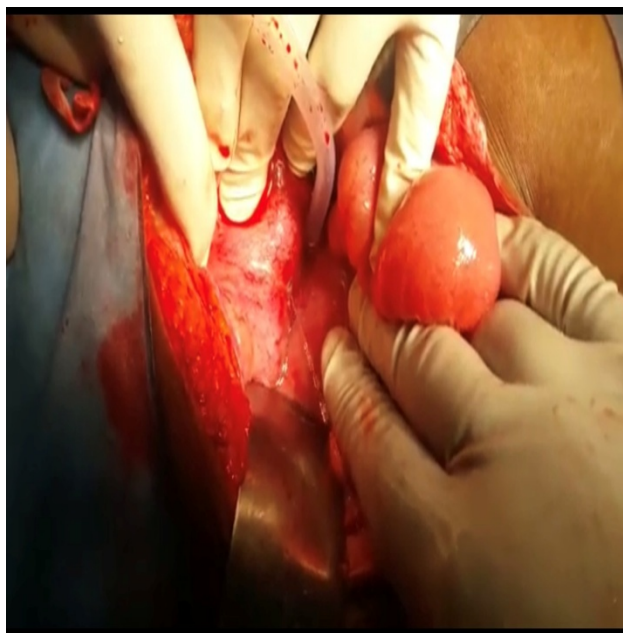


Fig. 1: Urine pouring out from the rent on the bladder

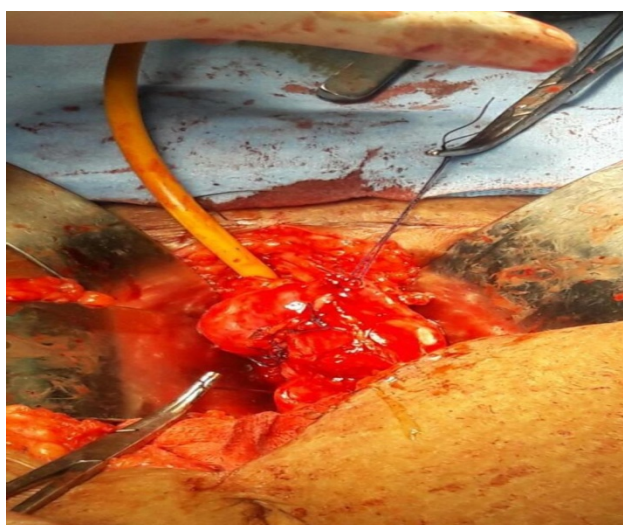


Fig. 2: Bladder repair

Her post-operative period is uneventful, recovered without any complications and was discharged with urinary catheter in situ on V post op day. Her follow up was uneventful. Bladder training was done and foley's catheter

was removed after 21 days. Her bladder function returned to normal after 28 days. A Cystoscopy was done after 28 days and the rent appeared to be healed.

3. Discussion

It is very rare to encounter a case of spontaneous bladder rupture in obstetric patients. In most instances they are associated with bladder diverticulum antenally or during cesarean following a trial of labour after cesarean section (TOLAC), where its also associated with rupture of uterus. In non obstetric patients, bladder rupture is associated with history of recent trauma, malignancy of bladder, outflow obstruction, during instrumentation, indwelling catheters, neurogenic bladder or a combination of these.

Bladder rupture in postpartum period without any predisposing factors or underlying bladder pathology is an emergency. The etiology of this condition is multifactorial.⁷ Sustained pressure of the fetal head against the bladder during forceful uterine contractions may lead to pressure necrosis of the bladder dome.^{2,5,7} This is more likely if the patient is not catheterized, resulting in a distended bladder during labour. The severity of pain in active phase and 3rd stage may mask the pain of rupture, because of which the condition is not suspected immediately.² Other contributory factors include prolonged second stage and high birth weight babies.⁵

Clinical picture mimics to that of an acute abdomen. Most common symptoms are abdominal pain, oliguria and anuria. The delay in diagnosis ranges from days to weeks if unsuspected, and even few patients continue to void normally for initial few days inspite of injury in the bladder. Failure to empty the bladder in the second stage of labour is probably the main reason for spontaneous rupture in this patient. High degree of suspicion, keeping in mind the probable clinical presentation, investigations such as retrograde cystoscopy, blood biochemistry suggesting abnormal renal parameters, and prompt decision for exploratory laparotomy will help in early diagnosis and thus reduces the morbidity.⁷ Operative treatment includes suction of peritoneal fluid, lavage, removal of devitalized tissue, immediate repair of bladder perforation and instituting good vesicle drainage.⁷

4. Conclusion

It's a totally preventable condition. Importance on emptying the bladder in the second stage before active pushing should

be taught to the training doctors and midwives. And a high degree of suspicion would reduce the morbidity attributed due to this.^{2-4,7}

5. Source of funding

None.

6. Conflict of interest

None.

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