Original Article

Catastrophic Spontaneous Bladder Rupture In Late Puerperium

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Abstract

Bladder necrosis after vaginal delivery is a rare but insidious occurrence. Bladder rupture during labor or postpartum is extremely rare. Patients usually complain of distension of the abdomen, suprapubic pain, anuria, or haematuria. Few patients may have no symptoms so the diagnosis of bladder rupture may be difficult in these situations. Bladder rupture is a challenge for obstetricians that should be diagnosed early and represents a surgical emergency.

We report a case of bladder rupture in a 29-year-old primigravida with gestational diabetes with fetal macrosomia which manifested late about 25th days after full-term normal vaginal delivery. There was an intraperitoneal rupture of the bladder diagnosed by ascitic fluid typing revealing high levels of urea and creatinine in the fluid which was further confirmed by Computerised Tomography (CT) imaging. Our case report aims to focus on early diagnosis and timely management, which may lead to good prognostic outcomes and prevent maternal and fetal morbidity and mortality.

Keywords: Bladder Rupture; Spontaneous; Vaginal Delivery, Computerised Tomography (CT)

INTRODUCTION

The term spontaneous bladder rupture is applied to those cases in which there is neither a history of antecedent trauma nor any underlying bladder pathology. (1,2) Spontaneous bladder rupture is extremely rare during labor or postpartum. Normal vaginal delivery in the presence of a distended bladder may lead to bladder rupture (3)

Catastrophic event of bladder rupture can be prevented by regular practice of evacuating the bladder before a patient goes to the second stage of labor.

Surgical intervention is crucial for the resolution of the clinical picture and consists of urine removal from the peritoneal cavity and closing of the ruptured bladder (4)

CASE:

A 29-year-old primigravida had full-term normal vaginal delivery where a male child of 3.8 kg was born at hospital A. She was discharged on 3rd day of delivery with normal vitals and a healthy baby.

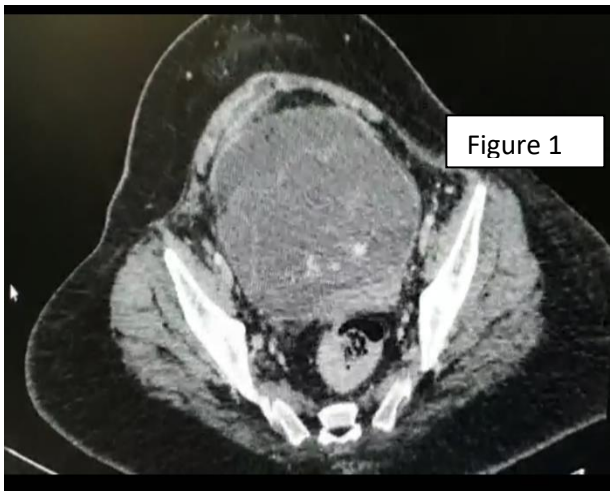
Two days later, the patient presented with abdominal distension, decreased urine output and altered sensorium so was taken to hospital B. On examination, she was disoriented, pulse rate of 100 bpm, and Blood pressure of 150/100 mmHg. The provisional diagnosis at hospital B was thought to be postpartum psychosis. On the sixth day postpartum due to a low GCS score she was intubated at hospital B, documented from the previous center. Foley's Catheterization was done, which drained 3 liters of turbid urine. Because of her deteriorating condition, she was referred to our hospital in the intubated state on day 11 of postpartum for further management.

At the arrival of our emergency department, her vitals were as follows; Pulse rate of 130 bpm, Blood pressure of 150/98 mmHg Spo2: 99% on FiO2 100%, abdominal distension with guarding present. Vaginal examination revealed no active bleeding with healthy episiotomy, on auscultation sinus tachycardia with bilateral wheeze was present. Foleys was in situ. She was shifted to our surgical intensive care unit. Her Blood parameters revealed raised WBC 15800 per microliter Hb 9.1 gm/dl, S. Urea- 47mg/dl S. Creatinine- 2mg/dl. HbA1c 7.4 % Random BSL 188 mg/dl. Urine analysis showed increased pus cells with proteinuria and *Proteus mirabilis* on culture.

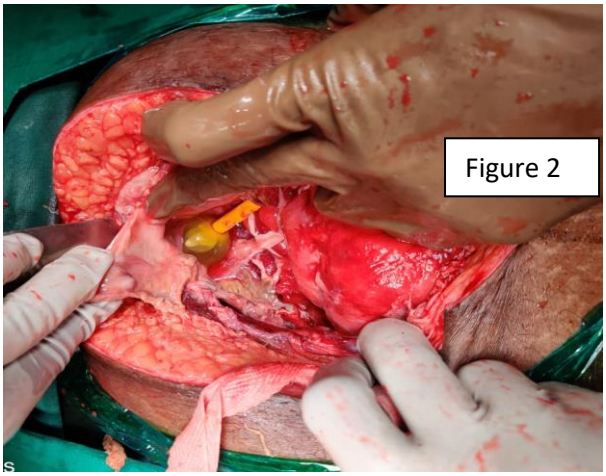
A contrast-enhanced CT imaging of the Abdomen Pelvis was performed. The findings in imaging were Urinary bladder showed hyperdense contents possibility of hemorrhagic fluid with wall edema sludge suggestive of cystitis. Few air pockets were also noted in the inferior and anterior walls suggestive of bladder mucosal injury.
A detailed history was noted where she was diagnosed with Hypertension at around 10 weeks of gestation with Gestational diabetes diagnosed at 24 weeks of gestation at Hospital A. However, she had not taken her antihypertensive drugs and diabetic treatment regularly.

Provision diagnosis of Urospecticemia with Diabetic Ketoacidosis with Chronic hypertension with superimposed Pre-Eclampsia was made at our hospital. She was managed at SICU, and broad-spectrum antibiotics with gram-negative coverage were started. Blood Sugar was controlled with Insulin. Anti-hypertensives and thromboprophylaxis were also given. The patient was in good general condition after two weeks of treatment at SICU thereafter she was extubated. The Foleys catheter was removed.

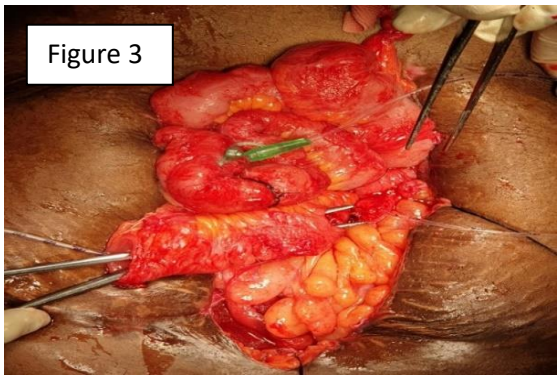
However, on the 25th day postpartum, she had abdominal distention with lower abdomen discomfort and difficulty voiding urine. Ultrasonography of the abdomen and pelvis was performed which reported moderate Ascites. Ultrasound-guided abdominal fluid tapping was done which revealed high levels of Urea and Creatinine in the fluid. Uro-surgeons were consulted. A contrast-enhanced CT imaging of the Abdomen-pelvis (Figure 1) showed infective loculated collections in the peritoneum with peritonitis. Urinary bladder outline could not be made out. The status of bladder continuity cannot be commented upon as the Urinary bladder lumen is not filled with contrast. Due to the concern of bladder injury with peritonitis, the patient underwent emergency exploratory laparotomy on the 26th day postpartum. Intra-operatively dept of General Surgery and Urology was consulted. Laparotomy findings

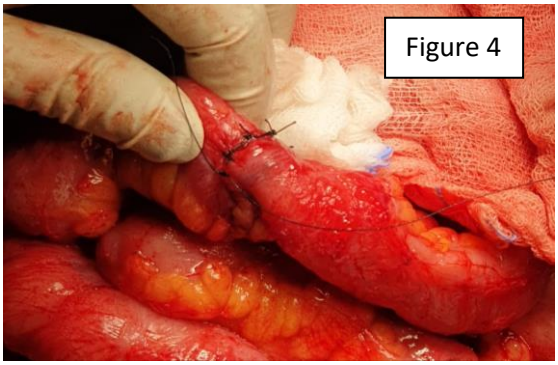


(Figure 2); Complete necrosis and sloughing-off of the bladder wall, only trigone, and ureteric orifices were intact with Foleys catheter seen. Adhesions of bowel loops were seen covered with flakes. Diversion procedures were impossible with adherent bowel and sepsis, so thorough lavage was given. The simplest procedure of Bilateral ureteric catheterization was done by placing a Foleys catheter per urethral and suturing the available bladder trigone to lower abdominal musculature.

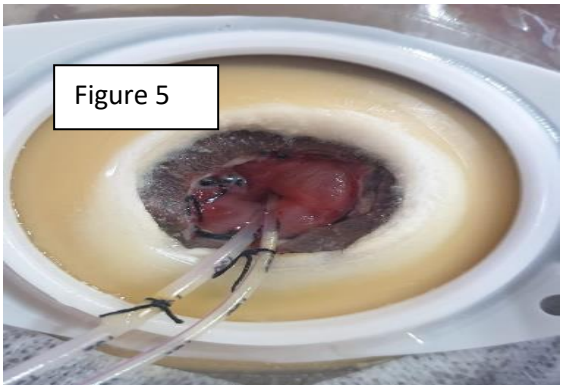


Output in the Foleys catheter improved for two weeks followed by peri catheter leak and leakage of urine through the laparotomy wound. Exploratory Laparotomy for possible bladder closure was planned on the 43rd day of postpartum. Intra-op findings showed no residual bladder, only trigone and ureter orifices, and small bowel adhesions with no free omentum. Thus, only ureteric catheters were replaced with DJ Stents. Bilateral PCN insertion was done under fluoroscopy guidance by Intervention Radiologist on the 53rd day. She was discharged with bilateral PCN-in-situ and planned for diversion after 3 months.





However, the patient lost to follow-up and reported to our hospital after almost 6 months. Bilateral PCNs were blocked with a small amount of urine leakage from the lower part of the wound, and PCNs were replaced. The patient underwent a third Exploratory laparotomy for the definitive procedure (Figure 3,4,5) of Ileal conduit with Bilateral uretero-ileal anastomosis with end-to-end ileo-ileal anastomosis which was performed successfully. Bilateral PCN was removed on day 10 of laparotomy. The abdominal drain was removed on day 11. She was discharged on day 14 of the third laparotomy with a healthy wound after suture removal.



Probable Risk factors (3,7,9)
Primiparous
Big-sized baby
Prolonged second stage of labor
Obstructed labor
Instrumental vaginal delivery (Forceps > Vacuum)
Blunt trauma
Maternal Diabetes
Chronic infection
Neuropathic bladder
Pelvic organs prolapse
Anatomical outflow obstructions
Binge alcohol drinking
Bladder diverticulum
Radiotherapy for cancer surgery

Discussion

Sisk and Wear first coined "spontaneous rupture of the urinary bladder" and defined it as if the bladder ruptured without external stimulation. (5)

In 1995, Kibel et al reported bladder rupture injury for the first time after normal vaginal delivery. (6) Bladder rupture during delivery can be classified into intraperitoneal or extraperitoneal types. The etiology of this condition is multifactorial (3,7,9)

Pathophysiology of the bladder rupture in puerperium includes sustained pressure from the fetal head against the bladder during forceful uterine contractions which may lead to pressure necrosis of the bladder dome (8).
Despite the low incidence and non-specific symptomatology, diagnosis is often delayed and associated with a high morbidity and mortality rate (10)

The patient usually presents with abdominal pain and oliguria. The most common clinical findings include supra-pubic pain, abdominal distension, gross haematuria, and inability to void. The triad of intraperitoneal bladder rupture includes abdominal pain, distention, and urinary ascites. A high index of suspicion is required if there is a sudden onset of abdominal pain, coincidental with bloody urine, generalized peritonitis, and abnormal renal biochemistry.
The inability to empty the bladder is one of the important presentations of bladder rupture, which our patient complained about after removing the initial catheter.

Diagnosis of intraperitoneal bladder rupture depends on retrograde cystoscopy, analysis of ascitic fluid for urea and creatinine, blood biochemistry, and exploratory finding. The imaging test of choice is cystography, demonstrating intraperitoneal contrast extravasation.

The decision for laparotomy in the presence of signs of peritonitis should not be delayed. Laparotomy is required for peritoneal lavage, excision of devitalized tissue, and primary repair of bladder perforation. (11)

In our case, the patient presented with typical symptoms and signs of bladder rupture and was in urosepsis. Correct diagnosis and timely intervention in the form of bilateral ureteric catheterization which was later replaced by DJ stents with percutaneous nephrostomy led to a better outcome for our patient. A definitive procedure was performed 6 months after the recovery. Early diagnosis and prompt surgical treatment will decrease the morbidity associated with this condition.

Conclusively, beyond symptoms and signs, suspicion of the physician to bladder rupture may be an important factor for rapid diagnosis of bladder rupture.

Pathophysiology of Bladder Rupture (8,9)

Sustained pressure from the foetal head against the bladder during forceful uterine contractions

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Compression on full bladder

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Pressure necrosis of the bladder wall

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Urinary retention

↓

Gross bladder distention

↓

Subsequent spontaneous Bladder rupture

↓

Urinary ascites

↓

Peritoneal Irritation due to urine

↓

Peritonitis

↓

Sepsis

↓

Shock

Conclusion

It is the most important thing to remember that bladder rupture is a preventable condition provided the obstetrician empties the bladder during the second stage of labor. Retention of urine leads to overdistention and thus bladder rupture. One should be vigilant in the postpartum period regarding urine output and encourage patients to empty their bladder. The high degree of suspicion remains the cornerstone of management. Early operative intervention with the closure of bladder rent along with prolonged bladder drainage can reduce the morbidity and mortality caused by bladder rupture.

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