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Case Report

Postpartum spontaneous bladder rupture

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ABSTRACT

Spontaneous bladder rupture after normal vaginal delivery is a rare complication. Patients may present with abdominal distention, fever, haematuria, oliguria and deranged KFT (kidney function test). We are reporting two cases of primigravida with postpartum bladder rupture, one case was diagnosed at laparotomy and the other preoperatively. A patient who presents with retention of urine, haematuria ascites and deranged KFT after uneventful normal vaginal delivery, spontaneous bladder rupture should be suspected. Early diagnosis and management can decrease the morbidity.

Keywords: Post-partum bladder rupture, Retention of urine, Vaginal delivery

INTRODUCTION

Spontaneous bladder rupture is a term applied to those cases in which there is neither a history of antecedent trauma or bladder pathology. It is a very rare complication seen after normal vaginal delivery with an incidence of 1 in 1,26,000.[1] The patient can present varying from post natal day (PND) 3 to PND 20 with complaints of acute abdominal pain, retention of urine, oliguria or anuria.

CASE REPORT

Case Report 1

A 23 years old P1L1, presented to gynae emergency with pain abdomen for 8 days, high grade fever, abdominal distension, vomiting for 4 days, retention of urine for 2 days. She had normal vaginal delivery 12 days back. She was well till day 5 of delivery when she started having pain abdomen and decreased urine output which also foul smelled. On PND 9 she had acute pain abdomen and was unable to pass urine for which she was catheterized and 4 litres of foul smelling urine was drained. On PND10 she had high grade fever, abdominal distention and vomiting for which she took self medications. On PND12 she again developed retention of urine and presented in emergency. On examination she was conscious, oriented, afebrile to touch, blood pressure of 90/48 mmHg, pulse -128/min and respiratory rate-60/min. On catheterization 900 ml of high colored foul smelling urine was drained. All systemic examination was within normal limits. Abdomen was distended, no guarding, no rigidity, bowel sounds absent, clinically no free fluid was elicited and uterus was 16 weeks, subinvoluted. On Speculum examination os was closed, lochia healthy and episiotomy site healthy. Vaginal examination uterus was 16 weeks, mobile, bilateral fornices free and non-tender. She was stabilized on intravenous fluids, inotropic support and Continous Positive Airway Pressure (CPAP). Broad spectrum antibiotics were started. The investigations revealed TLC-18,400/mm^3, blood urea-128mg/dL, serum creatinine-3.6mg/dL, urine microscopy:10-15 pus cells, ABG- severe metabolic acidosis, urine culture and sensitivity- Klebseilla growth sensitive to Colistin.

Ultrasoundm (USG) Abdomen and Pelvis revealed moderate ascites with internal septations. After 24 hours of admission, tachypnoea did not settle and she was unable to maintain her saturation and required intubation. Decision for Exploratory Laparotomy was taken in view of post partum sepsis. Per-operatively loculated clear fluid of 1.5lts was drained; bladder walls were edematous covering the fundus of uterus. The uterus ≠16weeks, intact, covered with exudates. A rent of 8x5cms with necrotic walls was identified in middle of anterior wall of bladder, bulb of Foleys catheter was seen and bladder limits were defined. Both ureteric orifices were away from the ruptured site, clear urine spurt present. Necrotic margins of the bladder defect were excised, reconstruction of bladder done and reinforced with omental flap (Figure 1).

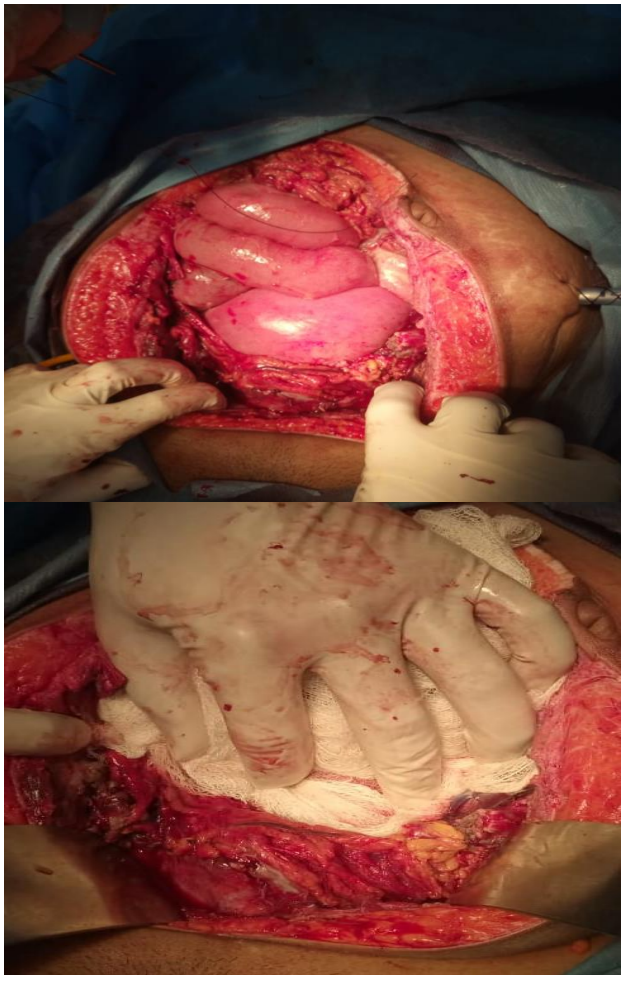

Figure 1: The repaired bladder with omental flap.

Dual drainage with supra pubic catheter (SPC) and per uretheral catheter (PUC) was done. On postoperative day (POD) 8, clear discharge from the main wound soaked the dressing. Urinary leak was suspected and stitch line was opened. Urologist confirmed the leak and advised wound management with daily dressings. She continued to have high grade fever, cultures were sent, higher antibiotics were given according to cultures. Patient responded to the antibiotics. She was extubated on POD 22. Wound management was done. She continued to leak and bilateral PCN was done on POD36 (Figure 2). Patient was discharged on POD 50 with the plan to remove PCN at 6-8 weeks. The main wound healed with secondary intention (Figure 3). The patient is doing well and is on follow up with us. The excised wall of bladder which was sent for histopathology showed necrotizing cystitis.

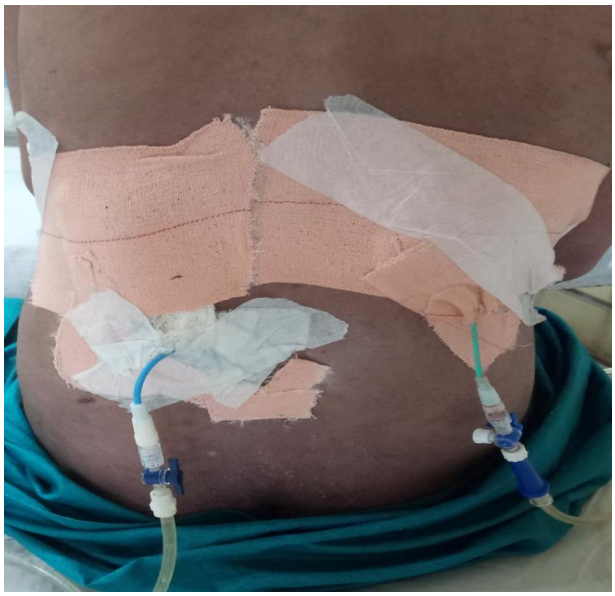

Figure 2: Bilateral PCN.

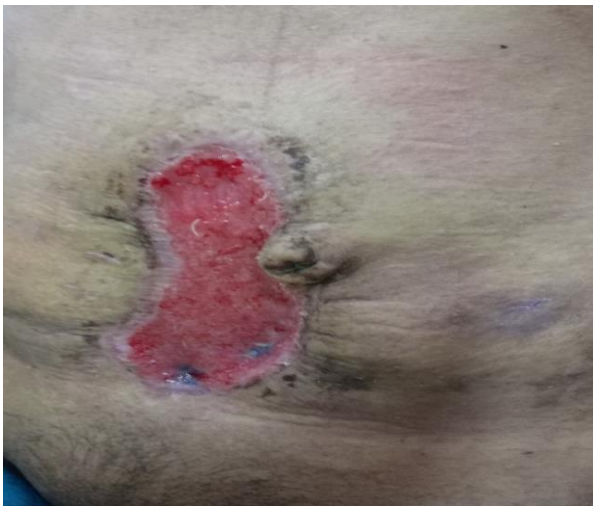

Figure 3: Wound healed with secondary intention.

Case Report 2

A 24 years old P1L1, presented on PND one of full term normal vaginal delivery (FTNVD) of a 3.5kg stillborn baby with chief complaints of abdominal distension, haematuria and fever. Patient was conscious, oriented, BP- 116/76 mmHg, PR-96/min and had 101º F fever. Abdomen was distended, mild tenderness was present and bowel sounds were absent. On catheterization 1500 ml of haematuric urine was drained. Speculum examination cervix, episotomy wound and lochia was

healthy. Vaginal examination uterus was 18 weeks and well contracted. The baseline investigations were normal. USG abdomen and pelvis showed a collection of 6x8 cm in left iliac fossa. The patient was managed conservatively for abdominal distention which got relieved, bowel sounds appeared but haematuria persisted. In view of persistent haematuria, Computed Tomography (CT) abdomen and pelvis done revealed two rents in uterus one at anterior surface and the other at anterolateral wall each of 1cm. The defect on anterior surface of uterus was communicating with defect in urinary bladder wall and other on left anterolateral wall was communicating with left broad ligament. There was a collection of 10x7.6x15.6cm. Since the patient was having adequate urine output and was accepting orally, urologist were of the opinion that patient should be managed conservatively. On PND 7 she started having fever which was not getting controlled despite antibiotics. In view of persistent fever not responding to higher antibiotics and collection on USG decision for exploratory laparotomy was taken. Per-operatively findings similar to CT were found. The margins of uterine rent were necrosed and highly friable and could not be repaired and decision for hysterectomy was taken. Primary closure of bladder rent along with dual drainage with SPC and PUC was done. The patient was discharged in satisfactory condition.

Table 1: Cases reported by various authors.

S .no	Year	Author	Presenting day	Type of delivery	Mode of diagnosis	Chief complaint	Site of perforation	Duration of labour	High risk factors
1.	1995	Kibel et al[1]	PND 3	Ventouse	CT Cystogram	-	Posterior wall	10 hours	-
2.	1997	E N Ekuma et al[6] (Saudi Arabia)	PND 7	Normal	Laparotomy	Acute pain, oliguria	Dome	10 hours	-
3.	2004	Julius Wandabwa et al[7] (Africa)	PND 9	Normal	Laparotomy	Acute pain, abdominal distension	Fundus	14 hours	Necrotizing cystitis
4.	2005	Pal A K et al[8] (Kolkata)	PND 3	Normal	CT Cystogram	Ascites, oliguria	Dome	11 hours	-
5.	2008	Aruna Kekre et al[2]	PND 9	Normal	Laparotomy	-	Fundus	1 hour (2nd stage)	-
6.	2008	KS Png et al[4] (Singapore)	PND 5 PND 2	Normal Ventouse	CT Cystogram	Acute pain, abdominal distension	Dome	½ hour (2nd stage)	-
7.	2015	Vidyasagar et al[9] (Mumbai)	PND 7	Forceps	CT Cystogram	Anuria, abdominal distension	Dome	1 hour	Macrosomic baby (4.2 Kgs)
8.	2016	Farahzadi et al[10] (Iran)	PND 20	Normal	Laparotomy	-do-	Dome	N.A	-
9.	2018	Valsa Diana et al[11] (Puducherry)	PND 4	Normal	CT Cystogram	-do-	Dome and posterior wall	12 hours	Macrosomic baby (4.2 Kgs)

DISCUSSION

The spontaneous bladder rupture can be attributed to bladder disease or sudden increase in the intravesical pressure during second stage of labour.[2] The proximity of lower urinary tract and reproductive tract predisposes them to iatrogenic injury. As the trigone of bladder rests on anterior vaginal fornix and base of bladder rests on lower uterine segment and cervix, they are most likely to get injured.[3]

One of the pathogenesis explained for spontaneous bladder rupture is that during pregnancy there is reduced muscle tone in the bladder leading to incomplete evacuation of bladder further causing stretching of bladder walls and damaging the detrusor muscle. Because of this pathogenesis retention may occur as they pass small amounts of urine and retention may go unnoticed leading to gross bladder distention and subsequent spontaneous bladder rupture.[4] The other theories proposed for spontaneous bladder rupture are sustained

pressure from the fetal head against the bladder during forceful uterine contractions which may lead to pressure necrosis of the bladder dome, macrosomic babies and prolonged second stage.[5]

Kibel et al in 1995 reported the first case of spontaneous bladder rupture in puerperium.[1] Since then nine more cases have been reported.[1,2,4,6-11] In all these case reports patient had presented with abdominal pain, retention of urine and oliguria/anuria which is similar to our cases. In our case 1 blood urea and Serum creatinine was elevated. The earlier cases reported in the literature have also shown that urea and creatinine is raised in nearly 100% cases who present 24 hours after bladder rupture.[12] In our case 2 urea and creatinine was normal because she presented within 24 hours of delivery. Heyns et al reported that serum urea and creatinine is elevated in 45% of patients who present within 24 hours of bladder rupture and in rest it is normal.[12] The diagnosis of bladder rupture can be made either preoperatively by CT cystography or Retrograde cystography or Cystoscopy or by laparotomy. In our case 1 the diagnosis was made at laparotomy while in case 2 diagnosis was made preoperatively. As per the cases reported in the literature (Table) 45% of the cases are diagnosed on laparotomy and rest preoperatively.

The risk factors for spontaneous bladder rupture have been reported as macrosomic baby and necrotizing cystitis.[7,9] In case 1 necrotizing cystitis was the reason for rupture. Although this patient was asymptomatic prior to delivery but could have had probably asymptomatic bacteriuria which got further aggravated by retention of urine eventually leading to bladder rupture. Second case had macrosomic baby which probably led to this complication.

It has been reported that in 55% of spontaneous bladder rupture the site is at dome of the bladder (Table), the explaination given for this is during intrapartum period there is incomplete emptying of bladder leading to distention of bladder and there is constant pressure by fetal head leading to pressure necrosis. In 45% cases of spontaneous bladder rupture reported the site was posterior wall of bladder which was similar to our case 2. In our case 1 rupture was on anterior wall of bladder which has not been reported earlier.

CONCLUSION

Patient presenting with abdominal distension, oliguria/anuria, deranged KFT after normal delivery one should think of bladder rupture. In case of foul smelling urine in postpartum period appropriate antibiotics should be prescribed to avoid necrotizing cystitis. As it's a totally preventable condition early diagnosis and prompt surgical treatment is important and decreases the morbidity in these cases. The patients should be encouraged to empty bladder during intrapartum and postpartum period. Counseling at the time of discharge regarding importance of decreased urine output should be done and to ask them to report to the health facility immediately in case she is unable to pass urine.

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