Urinary ascites in a preterm female neonate: a rare case report

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Here we report a rare case of urinary ascites due to spontaneous bladder rupture in a preterm female neonate. The baby presented with respiratory distress, abdominal distension, anuria, and renal insufficiency. The diagnosis of bladder rupture was confirmed by peritoneal fluid aspiration with biochemical analysis and ultrasonography abdomen. The patient was managed successfully by establishing urinary outflow with indwelling Foley's catheterization of the urinary bladder. ***Ann Pediatr Surg*** **13:228–231 © 2017 Annals of Pediatric Surgery.**

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Introduction

Neonatal bladder rupture leading to urinary ascites is extremely rare and very few cases have been reported in preterm female neonates [1]. The underlying etiology may be spontaneous or iatrogenic. The most common cause of bladder rupture in neonates is the umbilical catheterization, accounting for 75% cases [2]. Obstructive uropathy like posterior urethral valve, abdominal trauma, neurogenic bladder, iatrogenic injuries during surgical procedure, birth trauma, difficult obstetric delivery, and forceful urethral catheterization may also lead to bladder perforation [3]. Very rarely, it can occur due to ischemic injury caused by prolonged hypoxia, which may be precipitated by drugs like morphine administration, birth asphyxia, and poor perfusion [4]. Management may be done conservatively with establishment of urinary drainage by catheterization or may require surgery (Table 1).

Case report

A 1500 g preterm female neonate was delivered vaginally to a primigravida mother at 34 weeks' gestation. Antenatal and perinatal period was uneventful. There was no evidence of instrumentation during the birth. The newborn cried immediately after birth, with 1 and 5 min APGAR score 7 and 9/10 and no complication was detected in the immediate postnatal period. The baby was discharged after establishment of feeding on expressed breast milk after a short stay in the postnatal ward. At home, on the 10th day of life, the mother perceived that the baby was not accepting feed properly and had poor activity. The baby had also developed progressive abdominal distension, diarrhea, and vomiting.

At the time of admission the patient was severely dehydrated, and showed a marked respiratory distress with gross abdominal. Urine output was almost nil for the last 12 h. The patient was resuscitated with intravenous fluids, high-flow oxygen, and put on appropriate antibiotics. Due to marked respiratory distress and worsening of shock, the baby was mechanical ventilated. Despite adequate fluid augmentation, urine output remained nil. In investigations hemoglobin was 13 g/dl, total leukocyte count was 14 000/mm^3, platelets were 260 000/mm^3, blood urea was 140 mg/dl, and serum creatinine was 3.4 mg/dl. On chest and abdomen radiographs there was central pooling of gut loops with ground glass opacities in flanks along with right upper-lobe pneumonia (Fig. 1a). On ultrasonography (USG) abdomen, urinary bladder was structurally normal but there was presence of 3+ fluid was in the peritoneal cavity (Fig. 2). Due to marked respiratory distress and massive ascites, about 400 ml of straw-colored peritoneal fluid was aspirated. After peritoneal fluid aspiration, abdominal radiograph was repeated (Fig. 1b). Peritoneal fluid biochemistry analysis confirmed the urinary origin by showing high urea and creatinine. The baby improved hemodynamically rapidly, and was extubated after 24 h and weaned off to continue positive airway pressure. USG abdomen with injected normal saline through urinary catheter confirmed bladder rupture by revealing urinary extravasation in the peritoneal cavity. There was no previous history of accidental trauma, manual decompression before hospitalization, and umbilical catheterization in the baby. After confirming the diagnosis, an indwelling urinary catheter was left *in situ* and urine output started gradually improving. After 14 days, no leakage of urine in the peritoneal cavity was seen on USG abdomen, and thus urinary catheter was removed. The patient had established adequate urine output and renal function tests were normalized, and was hence discharged 5 days.

Discussion

Urinary bladder perforation leading to ascites in neonates is very rarely reported. Umbilical catheterization, urethral catheterization, congenital anatomical malformations, and rare spontaneous rupture of bladder are the common observed etiological factors (Table 2). Spontaneous rupture of bladder in female neonates with absence of genitourinary abnormality is even still rare [15]. Although exact etiology is not known but it had been documented that prolonged exposure to hypoxia and sepsis may act as predisposing factors for idiopathic bladder rupture. In the index case the baby was passing urine adequately for ~6–7 times in 24 h initially after discharge. As the baby was on expressed breast milk feeds, faulty feeding techniques might have caused aspiration pneumonia. Aspiration pneumonia along with late onset sepsis had

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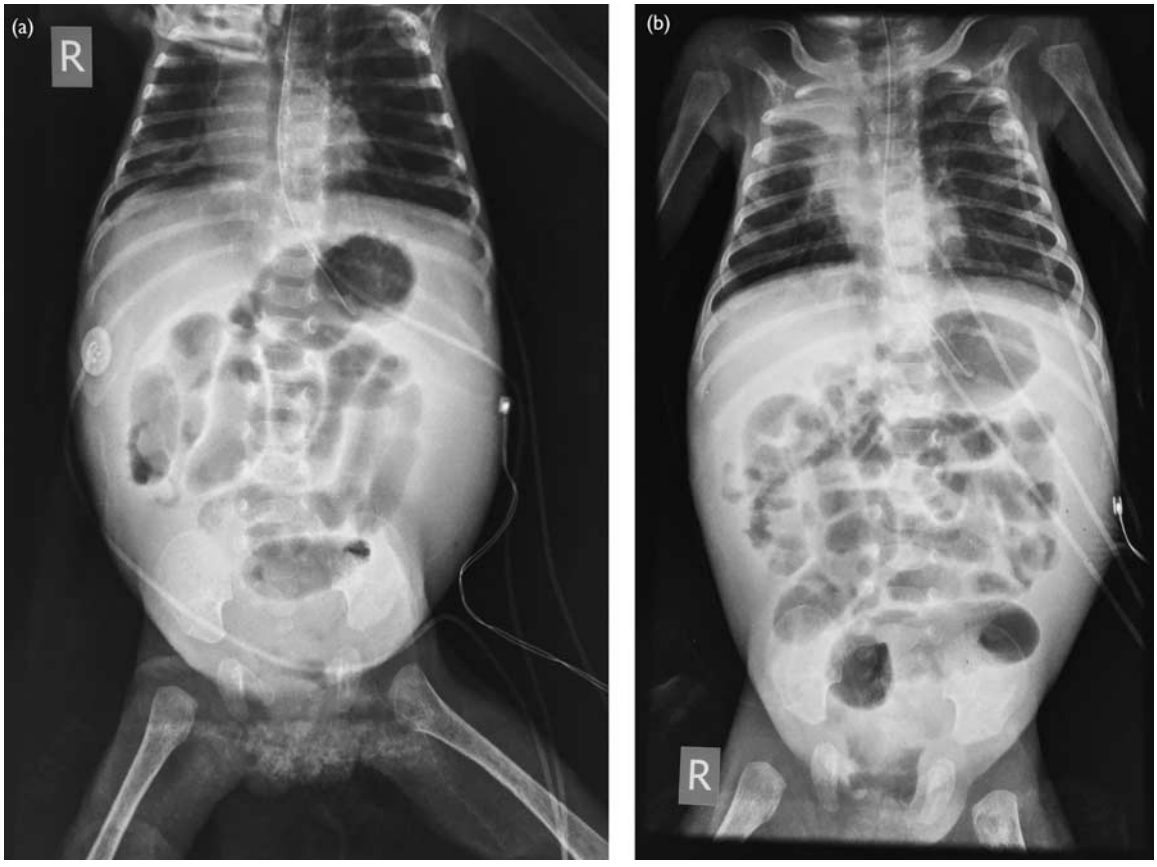
Table 1 Documented cases of bladder rupture with urinary ascitis and modalities of management

References	Gestation (sex)	Age (days)	Etiology	Management
Morrell *et al.* [5]	Preterm (female)	24	Ischemia	Conservative
	Term (male)	After birth	Ischemia	Conservative
Basha *et al.* [6]	Preterm (female)	28	Catheterization	Surgery
Limas *et al.* [7]	Preterm (female)	2	Idiopathic	Conservative
Tran *et al.* [8]	Fullterm (female)	27	Urinary tract infection	Surgery
Vasdev *et al.* [9]	Preterm (male)	9	Idiopathic	Surgery
	Preterm (male)	1	Idiopathic	Conservative
	Preterm (female)	24	Idiopathic	Conservative
	Preterm (male)	1	Ischemic	Conservative
Upadhayaya *et al.* [10]	Term (male)	2	Idiopathic	Surgery
Trigui *et al.* [11]	Preterm (female)	2	Manual decompression	Surgery
Solarin *et al.* [12]	Preterm (male)	At birth	Sepsis	Surgery
	Preterm (male)	At birth	PUV	Ablation of PUV
	Preterm (male)	3	Sepsis	Surgery
Arya *et al.* [13]	Fullterm (female)	5	Idiopathic	Surgery
Bakal *et al.* [14]	Preterm (female)	Not known	Catheterization	Surgery
Bakal *et al.* [14]	Term (female)	Not known	Catheterization	Surgery

PUV, posterior urethral valve.

Fig. 1

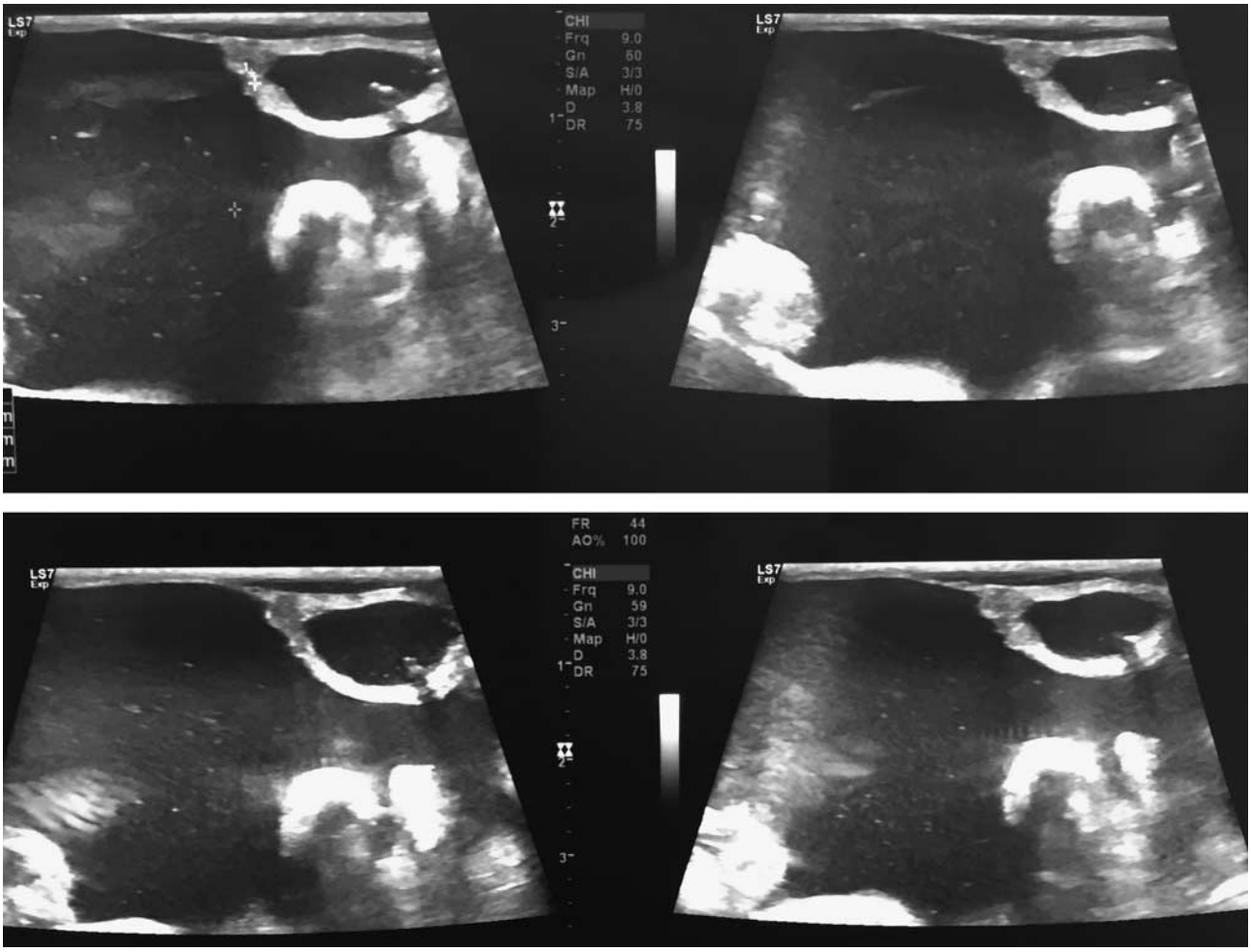


(a) Abdomen radiography (before peritoneal aspiration) showing central pooling of gut loops and ground glass opacity in flanks due to ascitic fluid. (b) Abdomen radiography (after peritoneal aspiration) showing central pooling of gut loops and ground glass opacity in flanks due to ascitic fluid.

lead to development of septic shock in the baby. There was no hospitalization in past and the baby was thriving well at home. As other common etiologies for bladder rupture were ruled out in the index case, we presumed that ischemic injury caused by hypoxia and sepsis was the underlying mechanism of bladder perforation.

Neonatal bladder rupture presents with progressively worsening ascites, oliguria/anuria, deranged renal functions, and marked respiratory distress. Time of presentation has been reported to vary from first day of life to as long as 24th day of life [15]. Neonatal bladder perforation should be always considered when a baby presents with abdominal distension, anuria, and unexplained renal failure [6]. Due to 'autodialysis' occurring at peritoneal membrane, there will be peculiar changes in serum biochemistry that may be life threatening [9]. These findings are helpful in differentiating urinary ascites from lymphatic ascites, which

Fig. 2



Ultrasound abdomen showing massive ascites with internal echogenicity due to interaperitoneal extravasation of urine.

Table 2 Etiology of neonatal bladder rupture

Anatomical malformations
Posterior urethral valve
Congenital diverticulum of the bladder
Pelvic mass
Umbilical arterial catheterization
Profound hypoxia
Birth asphyxia
Sepsis and septic shock
Drugs administration, e.g. morphine administration
Idiopathic (may be associated with)
Obstructive uropathy
Abdominal trauma
Neurogenic bladder
Injuries during endoscopic or open surgical procedures
Birth trauma, difficult obstetric delivery
Urinary tract infections

have more protein content. Serum electrolytes will show hyponatremia and hyperkalemia, and blood urea nitrogen levels will be increased [5].

Diagnosis of urinary ascites can be made by abdominal radiograph and USG abdomen. Abdominal radiograph will show central pooling of gut loops in abdomen and ground glass opacity in flanks. USG abdomen can be confirmative and also help in ruling out other associated congenital anomalies like posterior urethral valve. Peritoneal tapping is necessary to establish the nature of ascitic fluid and to relieve abdominal distension. In case of urinary ascites, as in the index case, aspirated fluid will be transudate. Voiding cystourethrography is the gold standard technique for picking up bladder perforation and other associated anomalies [13].

Conservative management with continuous bladder drainage using an indwelling catheter is preferred initially [15]. Usually when there is a small-sized defect, complete healing takes place by the establishment of normal urinary output. Surgical interventions are preferred in cases where conservative treatment fails and when the size of the defect is large [13]. Surgical interventions commonly used include exploration of inner aspect of bladder and closure of defect with placement of cystostomy. The primary aim of management is establishment of urine drainage away from the peritoneal cavity. Although bladder perforation following umbilical catheterization has a mortality as high as 18%, idiopathic rupture of urinary bladder generally has good prognosis [2]. In our case, we carried out conservative treatment by establishing urine outflow with indwelling catheter, which resulted in complete healing of defect.

Conclusion

Healing of a lesion in urinary bladder rupture can be achieved by conservative management if the size of the lesion is small. Although urinary ascites is very rare, it require prompt resuscitation and management to have a good outcome.

Conflicts of interest

There are no conflicts of interest.

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