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BRIEF REPORT

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Urinary bladder perforation in a premature infant with Down syndrome

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Abstract Urinary bladder perforation due to bladder catheterization in neonates is a rare iatrogenic complication. It has been reported secondary to various causes and a variety of surgical settings in neonates. A case of urinary bladder perforation due to catheterization in a premature baby with Down syndrome, who presented with progressive renal failure and mild-to-moderate ascites, is reported. Urinary bladder perforation should be considered in a case of neonatal ascites with renal failure, which is unexplainable by other causes. We recommend that bladder catheterization in a baby with Down syndrome, whose urinary bladder may be at an increased risk for perforation as part of their generalized hypotonia, should be performed cautiously. To our knowledge, this is the first case report of bladder perforation due to urinary bladder catheterization in a case of Down syndrome.

Keywords Down syndrome · Urinary bladder perforation · Bladder catheterization · Voiding cystourethrography · Ascites

Introduction

Perforation of the urinary bladder due to catheterization is a rare complication in neonates. Generalized hypotonia is the most striking finding in infants with Down syndrome and the involvement of internal organs such as urinary bladder remains possible. If unrecognized early, traumatic urinary bladder perforation, secondary to hypotonia from the patient's underlying condition, can pose a diagnostic challenge for physicians. We report a case of urinary bladder perforation due to catheterization in a preterm infant with Down syndrome. To our knowledge, this is the first report of bladder perforation due to catheterization in an infant with Down syndrome.

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Case report

A preterm female baby was born in a private hospital at 32 weeks of gestation, by cesarean section to a 40-year-old gravida 5, para 4 mother. The mother had regular antenatal care with an uneventful course, except for being a gestational diabetic, which was well controlled by diet alone. The Apgar scores were 5 and 7 at 1and 5 min, respectively. Her birth weight was 2.4 kg. The clinical

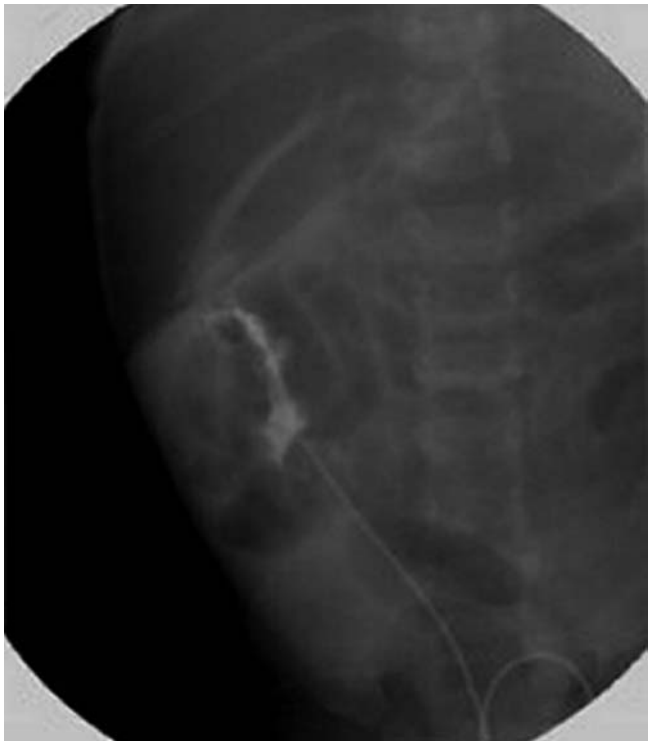

Fig. 1 Voiding cystourethrogram showing leakage of contrast in the peritoneum. The tip of the catheter is seen in the right upper quadrant of the abdomen

course at the referring hospital included the management of mild respiratory distress syndrome and the administration of total parenteral nutrition. None of the cultures taken at the referral hospital revealed any growth. She was referred at the age of 4 weeks to our hospital with the diagnosis of Down syndrome, congenital heart disease, and elevated levels of serum urea and creatinine, for further evaluation and management. The initial examination at our neonatal intensive care unit revealed mild-to-moderate ascites. The plasma electrolyte profile showed serum sodium 131 mmol/l, chloride 97 mmol/l, potassium 4.2 mmol/l, bicarbonate 12 mmol/l, serum urea 11.7 mmol/l, and creatinine 242 μmol/l. Repeated urea and creatinine measurements showed progressive elevation. Urine output was also decreased. Her bladder had been catheterized in the referring hospital with a 5-Fr nasogastric tube. The initial catheterization was performed at the referring hospital due to initial difficulty in passing urine. The catheter was left in place 3 days prior to the transfer to our institution. Voiding cystourethrography (VCUG) performed at our institution showed contrast material leakage into the peritoneum, and the tip of catheter was in the right upper quadrant of the abdomen (Fig. 1). No other abnormalities in the urinary tract were noted.

The diagnosis of bladder perforation was made. Conservative management with antibiotics was considered unsuitable due to worsening of the clinical status and the rapid increase in blood urea and creatinine. The patient underwent exploratory laparotomy on the same day. A large 1-cm intraperitoneal perforation in the superior aspect of the urinary bladder was found and was repaired. Recovery was uneventful and the serum urea and creatinine became normal within 48 h (urea 5.4 mmol/l, creatinine 30 μmol/l).

Repeat VCUG 1 week later was normal. Renal ultrasonography did not reveal any abnormalities in the urinary tract. Cardiac failure with complete atrioventricular canal defect was well controlled by diuretics and digoxin. After successful management of feeding difficulties, the patient was discharged at 92 days of life.

Discussion

Urinary bladder perforation has been reported secondary to various causes in neonates and children [1, 2, 3, 4]. There are reports of bladder perforation in patients with Ehlers-Danlos syndrome [5, 6], premature babies on ventilators with prolonged sedation [7], umbilical catheterization [8], children using clean intermittent catheterization [9], urinary bladder anomalies, and posterior urethral valves [10]. Milles [11] proposed pressure necrosis as the pathogenic mechanism, as autopsies performed on catheterized patients revealed areas of denuded bladder urothelium, submucosal edema, and leukocyte infiltrates. Infection is another mechanism that can cause bladder rupture. A literature review revealed only isolated cases of catheter-related bladder rupture in neonates. Recently, Salama et al. [7] reported urinary bladder perforation in an extremely low birth weight infant that probably was triggered by prolonged urinary retention secondary to morphine infusion requiring Credé's maneuver on an overdistended bladder and insertion of an umbilical catheter. Jorion and Michel [5] and Schippers and Dittler [6] reported spontaneous rupture of bladder diverticula in association with Ehlers-Danlos syndrome. Sahdev et al. [10] reported a case of congenital bladder perforation and urinary ascites caused by posterior urethral valves. To the best of our knowledge, this is the first reported case of urinary bladder perforation in a preterm neonate with Down syndrome after urinary bladder catheterization.

Intra-abdominal leakage of urine can mimic renal failure and respiratory compromise through the development of urinary ascites [12]. The metabolic derangements associated with urinary ascites include elevation of serum urea and creatinine levels and hyperchloremic metabolic acidosis [13]. Case reports have shown patients presenting with increasing abdominal girth, low urinary output, and progressive respiratory distress [9, 10]. Although the pressure necrosis as a result of urinary retention can be a causative factor for the bladder perforation, inadvertent urinary bladder perforation secondary to catheterization and leakage of urine into the peritoneal cavity seems more likely in our case. This is strengthened by the finding of the tip of the urinary catheter in situ during VCUG, acute renal failure with electrolyte disturbance, acidosis, progressive elevation of urea and creatinine, decreased urine output, and mild-to-moderate ascites. VCUG remains the most valuable technique for demonstrating bladder perforation. Follow-up VCUG 1–2 weeks after postoperative closure is warranted to confirm the healing.

In conclusion, the diagnosis of urinary bladder perforation after catheterization should be highly suspected in the differential diagnosis in neonates and young infants with acute renal failure, ascites, and unexplained electrolyte disturbance.

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