Urinary Ascites and Transient Intestinal Obstruction in a Preterm Infant: An Interesting Case of Posterior Urethral Valve

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Abstract

Posterior urethral valve (PUV) is the most common congenital cause of bladder outflow obstruction in male neonates. We report a preterm neonate with PUV who presented as nonimmune fetal hydrops with intestinal obstruction in the antenatal period. The mother of our patient is a 33-year-old woman who started her prenatal care at our hospital at 30 weeks' gestation. Her sonogram done at 32 weeks in our hospital revealed fetal hydrops. It showed polyhydramnios, mild pyelectasis of right kidney, normal left kidney, and fetal ascites. Amniocentesis revealed bile stained amniotic fluid. Ultrasound during the procedure showed dilated fetal bowel loops with increased echoes. Following delivery at 32 weeks postnatal exam showed ascites with absence of skin edema, pleural, or pericardial effusion. The abdominal sonogram showed distended urinary bladder and bilateral hydroureteronephrosis. Bladder catheterization was done which relieved the bladder outlet obstruction. Voiding cystourethrogram was done later which confirmed PUV and bilateral grade 5 vesicoureteral reflux. The formation of urinary ascites in PUV serves as a pop-off mechanism to relieve the intravesical and intrarenal pressure. When this happens by mechanisms other than bladder rupture, it can lead on to transient intestinal obstruction and hepatic synthetic defects.

Keywords

► posterior urethral valve
► urinary ascites
► preterm infant
► hypoalbuminemia
► nonimmune hydrops fetalis
► anemia
► intestinal obstruction

Posterior urethral valve (PUV) is the most common congenital cause of bladder outflow obstruction in male neonates. More than 50% of the PUVs are diagnosed prenatally.[1] Prenatal ultrasound findings include thick-walled bladder, the keyhole sign, unilateral or bilateral hydroureteronephrosis, echo bright kidneys, and oligohydramnios.[2] Patients who are not detected prenatally usually are diagnosed as neonates or young infants. About half will present with urinary tract infections.[1] Newborns will present with respiratory distress due to lung hypoplasia. Lung hypoplasia develops because of oligohydramnios. Some may present with abdominal distension due to enlarged overdistended bladder or urinary ascites, difficulty with voiding, or a poor urinary stream.[3]

We present a 32-week preterm neonate with postnatally diagnosed PUV who presented as nonimmune fetal hydrops with intestinal obstruction in the antenatal period.

Case Report

Prenatal Course

The mother of our patient is a 33-year-old woman P1011 who had her prenatal care in St. Lucia which was uneventful except for sonogram done at 28 weeks' gestation revealed full fetal urinary bladder, and she was scheduled for follow-up. Her blood group was "A" positive and Coombs' test was negative. Her comorbidities included previous cesarean

section, obesity (body mass index 41.6), and sickle cell trait. Patient started prenatal care at our hospital at 30 weeks' gestation. Routine prenatal laboratories were done during the initial visit. She was admitted to the labor and delivery at 32 weeks' gestation for preterm labor. She received magnesium sulfate and antenatal steroids. The sonogram done at 32 weeks at our hospital revealed fetal hydrops. It showed an amniotic fluid index 44 (polyhydramnios), biophysical profile 4/8, expected fetal weight 3,424 g (>97%), mild pyelectasis of right kidney, normal left kidney, fetal ascites, umbilical arterial Doppler, and middle cerebral artery (MCA) Doppler values were within normal limits. In view of polyhydramnios, therapeutic amniocentesis was performed under ultrasound guidance around 1,800 mL green colored fluid was removed from the amniotic cavity. Fluid was sent for cultures, glucose, and karyotyping. They were later reported as normal. There were no complications. Ultrasound during the procedure revealed dilated loops of fetal bowel, echogenic bowel, and small hepatic calcifications. Postprocedure amniotic fluid index was 14. Following the amniocentesis, the preterm labor subsided. Fetal echocardiogram was done by the pediatric cardiologist. The fetus was noted to have sinus tachycardia with normal intracardiac anatomy.

Neonatal Course

A male neonate was delivered by elective cesarean section at 32 with Apgar score of 7 at 1 minute and 9 at 5 minutes. Physical examination of the neonate showed tense distended abdomen with no organomegaly or skin edema. Initial postnatal radiograph did not show any evidence of pleural or pericardial effusion. The neonate was mechanically ventilated, received surfactant, and was started on total parental nutrition and enteral feeding was withheld. He was administered ampicillin and cefotaxime for sepsis cover pending blood culture.

In the absence of postnatal evidence of hydrops, evaluation for isolated neonatal ascites was initiated. The umbilical cord blood rapid plasma reagin test for syphilis was negative. The initial blood work showed a hematocrit of 43.2 with no fetomaternal blood group incompatibility. The neonate's blood group was A positive and Coombs' test was negative. Kleihauer–Betke's test was done to rule out fetomaternal hemorrhage and was negative. Blood chemistry done in the first few days showed hypoalbuminemia, increasing trend of blood urea nitrogen and creatinine, hyponatremia, and hyperbilirubinemia (►Table 1). Total serum immunoglobulin M, serum lipase, and cytomegalovirus DNA polymerase chain reaction were negative.

The abdominal sonogram showed moderate amount of perihepatic ascites, tiny gall stones, patent main portal vein, full urinary bladder, and bilateral hydroureteronephrosis. In view of suspicion of urinary ascites, diagnostic abdominal paracentesis was done which showed the presence of urea and creatinine. Nephrology and urology were consulted.

In view of distended urinary bladder and deranged renal parameters, provisional diagnosis of bladder outlet obstruction was made. Bladder catheterization was done with a 5-Fr

feeding tube which promptly relieved the bladder obstruction and around 120 mL of urine was drained. The electrolyte abnormalities were corrected with appropriate fluid management (►Table 1). In view of PUV being the most common cause of bladder outlet obstruction in a male neonate, urology consult was obtained. Continuous bladder drainage was continued, and renal parameters gradually normalized. The abdominal distension gradually decreased and resolved. Hypoalbuminemia was corrected with albumin infusion. After the clinical status of the neonate improved, voiding cystourethrogram was done which confirmed PUV and bilateral grade 5 vesicoureteral reflux (VUR). Percutaneous vesicostomy was done. The neonate started prophylactic amoxicillin therapy for grade 5 VUR. In view of anemia with a downward trend of hematocrit, the neonate started iron and erythropoietin therapy to which the neonate responded.

In the absence of dysmorphology and clinical evidence of any other congenital anomaly, genetic evaluation was withheld. The neonate failed the hearing screen in both the ears which was done before discharge. Audiology follow-up was scheduled. The neonate was discharged home with urology and nephrology follow-up.

Discussion

Urinary ascites in PUV can occur because of rupture of calyceal fornices or transudation across the intact upper tract. It can also occur following the rupture of bladder wall. In obstructive uropathy, the upper tracts are subjected to high pressures in the intrauterine life. This affects the development of the kidneys and cystic renal dysplasia ensues. However, protective mechanisms do exist to prevent this irreversible damage to the kidneys. These protective mechanisms include VUR, bladder diverticula, and urine extravasation.[4] Extravasation at the level of the fornices may result in the formation of perinephric urinoma which may remain contained or communicate freely with the peritoneal cavity, leading to urinary ascites. We consider this to be the mechanism behind the formation of urinary ascites in our patient.

Although oligohydramnios is the common presentation of severe PUV, polyhydramnios does occur due to unclear mechanisms.[1,4,5] The amniocentesis done in our case showed bile stained amniotic fluid. We postulate that urinoma and full urinary bladder associated with the PUV led to transient bowel obstruction due to mass effect and that could explain the bile stained amniotic fluid. It could also be a possible mechanism for polyhydramnios.

We think that the urinoma was initially communicating freely with the peritoneal cavity which became contained later in the course. This resulted in the mass effect on the adjacent bowel loops. Postnatal renal parameters returned to normal soon after the bladder outlet obstruction was relieved. This showed that the kidneys were not subjected to high pressures for a long time.[6]

Fetal anemia accounts for 10 to 27% of hydrops.[7] To evaluate the risk of fetal anemia, Doppler measurement of

Table 1 Trend of Comprehensive Metabolic Panel of the infant in the first week of life

Laboratory parameter (reference range)	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 9
Blood urea nitrogen (3–25 mg/dL)	9.0	18.0	28.0	26.0	14.0	15.0	35.0	34.0	32.0
Serum creatinine (0.3–1.0 mg/dL)	1.00	1.30	1.80	1.90	0.90	0.60	1.20	0.80	0.60
Serum sodium (130–145 mEq/L)	131	125	126	140	135	145	154	146	136
Serum potassium (3–6 mEq/L)	4.8	–	6.0	6.2	3.5	–	5.5	5.3	4.5
Serum chloride (97–108 mEq/L)	98	92	94	105	101	108	119	110	103
Serum bicarbonate (17–24 mEq/L)	25	25	24	25	23	22	20	19	19
Serum calcium, total (6.2–11 mg/dL)	8.9	6.9	8.0	7.9	8.0	10.5	12.0	12.1	11.2
Serum protein, total (3.6–6 g/dL)	3.7	–	4.0	–	–	7.1	6.6	7.2	–
Serum albumin (3.0–3.9 g/dL)	1.8	–	1.9	–	–	3.9	3.6	4.2	–
Serum alanine aminotransferase (13–45 U/L)	22	–	30	–	–	26	14	<6	–
Serum aspartate aminotransferase (47–150 U/L)	59	–	57	–	–	112	53	85	–
Serum bilirubin, total (< 8 mg/dL)	3.2	–	10.9	13.5	13.1	16.0	12.8	11.7	7.6
Serum bilirubin (conjugated) (< 0.6 mg/dL)	0.0	–	0.0	0.3	0.6	1.5	2.9	3.0	2.0

the MCA peak systolic velocity should be performed in all hydropic fetuses after 16 weeks of gestation. This is an accurate noninvasive tool for predicting fetal anemia of any etiology.[8,9]

In case of suspected fetal anemia, fetal blood sampling is obtained by umbilical vein sampling, and the fetal hemoglobin level should be determined to exclude anemia as a cause of hydrops.[10] In all reported cases with anemia and non-immune hydrops fetalis (NIHF), hemoglobin values are less than 5 g/dL.[8] The mechanism for hydrops is thought to be high output cardiac failure.

In our patient, we had an abnormal MCA Doppler study. We did not do fetal cord blood sampling. The postnatal hemoglobin was 12 g/dL. Therefore, it is unlikely to be involved in the pathogenesis of hydrops fetalis in our case. Our patient did not have ABO incompatibility and direct Coombs' test was negative. There was no evidence of feto-maternal hemorrhage as well. We consider mild intrauterine hemolysis had occurred as evidenced by high reticulocyte count that led to the neonatal anemia. This can explain the gallstones observed in the antenatal sonograms.

Fetal renal regulation of fluid excretion is still unknown. Although both renal function impairment and elevated angiotensin levels may play a significant role in the etiology and pathogenesis of NIHF, hydrops can also occur without any significant renal damage and with normal urine production.[11] Hypoproteinemia with decreased colloid osmotic pressure is frequently proposed as one of the causes of hydrops fetalis. Although it has been reported that low serum albumin levels occur in severely anemic neonates with hydrops, studies have shown that most fetuses with immune hydrops have an albumin concentration within the normal range; it suggests that hypoalbuminemia is unlikely to cause the initial development of nonimmune hydrops.[12,13] Hypoal-buminemia thus seems to occur as a secondary effect in the cascade of hydrops (e.g., because of a reduced re-uptake of albumin from the interstitial compartment).

Hypoalbuminemia in our patient is likely due to the reduced hepatic synthesis of albumin secondary to compromised blood supply that could have resulted from the mass effect of the urinoma. We consider that the proteinuria seen in the postnatal life was probably due to the mild/transient kidney damage secondary to bladder outlet obstruction. This could be a contributing factor in hypoalbuminemia.

Conclusion

The formation of urinary ascites in PUV serves as a pop-off mechanism to relieve the intravesical and intrarenal pressures. When this happens by mechanisms other than bladder

rupture, it can lead on to transient intestinal obstruction and hepatic synthetic defects.

Conflict of Interest
None.

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