
CASE REPORT

Neonatal Urinary Ascites due to Kinking of the Right Ureter

by

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Abstract

A case of neonatal urinary ascites in a female newborn infant due to kinking of the right ureter is presented. The diagnosis was based on the following findings: clinically, the presence of ascites which was confirmed radiologically, and by abdominal paracentesis; by I.V.P. and operatively. After operative relief of the obstruction, the ascites disappeared and on the 8th day after operation she was discharged in a good condition.

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Introduction

Neonatal urinary ascites or leakage of urine from the urinary tract into the intraperitoneal space is a rare component of urinary tract obstruction in neonates (Mann et al., 1974). Three centuries ago Mauriceau vividly described his difficulties in delivering an infant whose abdomen was distended by fluid (Bryan, 1975). In 1864, Hicks was the first who described neonatal ascites associated with obstruction of the urinary tract. Later on in 1894, Fordyce reviewed autopsy findings and collected 63 cases of fetal ascites, 17 of which had urinary tract dilatation. He drew no conclusion from these findings and it remained until James and Davis (1952) showed a cause and effect relationship between urinary tract obstruction and urinary ascites and to report the first survivor with this disorder. Shortly thereafter in 1953, in a comprehensive review of the subject, Lord proposed neonatal urinary ascites to be secondary to transudation of urine into the peritoneal cavity from dilated upper urinary tract (Linde, 1966; Krane and Retik, 1974).

This paper is a report of a successfully treated female newborn infant with urinary ascites due to kinking of the right ureter. The diagnosis, management, and treatment will be discussed.

Case report

A fullterm female infant was born on July 29, 1975, by Caesarean section due to a dermoid cyst in the cavum Douglas

in the Obstetric Department, Dr. Cipto Mangunkusumo General Hospital, Jakarta. She was the 7th child of a 30-year-old Indonesian woman on whom no antenatal care was performed. The antenatal history was uneventful. At birth the infant was mildly asphyxiated, the Apgar score was 5 at 1 minute and 8 at 5 minutes after birth. At the age of 15 minutes physical examination revealed a female infant with a body weight of 3,650 gm and a body length of 50 cm. She was dyspnoeic with a respiration rate of 50/minute; cyanosis, pallor, and jaundice were absent. The heart rate was 138/minute. The heart and lungs were within normal limits. The abdomen was distended; shifting dullness and fluid thrill were observed. The liver and spleen could not be evaluated due to the tense abdominal wall. No abdominal organ nor mass was palpable at that time. The head circumference was 34 cm., the chest 32 cm., and the abdominal circumference 41 cm. Catheterization of the bladder yielded 40 ml. of yellow urine.

The peripheral blood examinations were within normal limits; blood glucose concentration examined by way of dextrostix was normal. A plain abdominal X-ray, made 1 hour after birth, showed homogenous opacification with small air shadows at the middle region.

At the age of 18 hours, abdominal paracentesis was performed and 220 ml. of yellow ascitic fluid were aspirated. The specific gravity of the aspirated fluid was 1.030 and the urea content was 63 mg.%, while the blood urea content was 41 mg.%. The abdominal girth

decreased by 3.5 cm. and the respiratory distress was markedly relieved.

An intravenous pyelogram (I.V.P.) on the first day of life showed delayed excretion of the right urogram.

A second plain abdominal X-ray film on the 3rd day of life showed that ascites signs were still present and micturation cystourethrogram (M.C.U.) examination revealed that the bladder was within normal limits; reflux posterior urethral valves were not observed.

Exploratory laparotomy was performed at the age of 8 days which revealed the presence of kinking at 3 places at the proximal portion of the right ureter causing obstruction. Besides, a slight dilatation of the right kidney was observed. These kinkings were abolished by relieving the adhesion around the ureter which resulted in a flow of urine from the right kidney. Three days after the operation there were no signs of ascites. On the 8th post-operative day the infant was discharged in a good condition.

Discussion.

Our case was a newborn female infant who was delivered by Caesarian section. The physical examination at birth revealed a tense abdomen and signs of ascites. This was confirmed by an abdominal X-ray taken one hour after birth.

The differential diagnosis of ascites at birth includes :

1. Urinary ascites due to obstruction of the urinary tract, a more common

cause of ascites in newborns (Behrman, 1973).

2. Meconium peritonitis (North et al., 1966; Behrman, 1973).
3. Peritonitis secondary to ruptured ovarian cyst (North et al., 1966; Behrman, 1973).
4. Chylous ascites (North et al., 1966; Nelson, 1966; Schaffer and Avery, 1971; Behrman, 1973).
5. Ascites caused by syphilis (Behrman, 1973; Bryan, 1975).
6. Hemolytic disease (North et al., 1966; Bryan, 1975).
7. Bile ascites (Schaffer and Avery, 1971; Behrman, 1973).

If ascites is present in a newborn at birth, the examination of the fluid is very important. For this purpose paracentesis intraperitoneally must be done. The presence of urine in the ascites fluid may be suspected from its urea content, which is greater than that of plasma (North et al., 1966; Howat, 1971). Abdominal paracentesis in our case removed 220 ml. of yellow ascitic fluid with an urea content of 63 mg./100 ml.; since the plasma urea was 41 mg./100 ml., it was assumed that this fluid was urine.

Urinary ascites is an uncommon finding in newborn infants; male infants have been more frequently affected than females (North et al., 1966). Mann et al. (1974) reported 31 cases of male infants out of 36 cases since 1952. Posterior urethral valves were the most common underlying cause of urinary tract

obstruction in male infants; Cywes et al. (1968) reported 13 out of 19 cases and Mann et al. (1974) reported 23 out of 36 cases. The remaining causes included a variety of lesions like bladder neck obstruction ureterocele urethral atresia meningomyelocele congenital neuroblastoma ureteric stenosis absence of the posterior bladder wall and hamartoma of the bladder.

If ascites in the newborn is attributed to urinary tract blockade, then rational therapy is necessary. Prompt relief of abdominal distention and effective drainage of the urinary tract is essential (Howat, 1971). Paracentesis in the delivery room relieved respiratory difficulties. Investigation of the cause of obstruction of urinary tract in the newborn or in perinatal period starts with intravenous pyelography and micturation cystourethrography. Urinary ascites is an absolute indication for this study and must be worked out immediately. Urographic signs could support the presence of urinary tract blockade.

In our case IVP and MCU examinations resulted in obstruction of the right ureter. In this case operation was performed to investigate the type of the obstruction. On operation there were 3 kinkings at the proximal portion of the right ureter and a slight dilatation of the right kidney. Since there was no per-

foration of the kidney or the ureter, we can assume that the pathogenesis of this urinary ascites might be a transudation process. Such a condition was first described by Lord in 1953, where dilatation of the urinary tract was secondary to the high hydrostatic pressure. Another possibility may be a rupture of the urinary tract causing a communication of the urinary tract and the peritoneal cavity. The perforation is usually located at the fornix of the renal calices. Mann et al. (1974) reported 23 cases with kidney perforation.

The timing of operation is very important and depends on radiological examination, clinical state, and renal function. Before 1965, most of the reported cases of this disorder concerned stillborns or babies who died shortly after birth (North et al., 1966; Krane and Retik, 1974). Three infants have been previously reported who were born alive with urinary ascites and only one of them had survived (North et al., 1966). In the collected series of Cywes et al. (1968) the mortality rate was 87.5% when urinary ascites was present at birth, and 60% when the diagnosis was made on the first day of life (Mann et al., 1974). Mortality was due to respiratory discomposure and impaired renal function. Since 1952, 33 cases have been reported with 16 survivors (Ravits et al., 1973).

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