

Pleural Effusion And Hydronephrosis In A Newborn

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Abstract: While the association of obstructive uropathy with ascites has been known since 1863, and with pleural effusion since 1954, the latter combination remains rare. This case report describes a male newborn with a massive left pleural effusion that was caused by same-sided hydronephrosis from obstructing posterior urethral valves. The effusion disappeared within a few days after adequate urinary drainage was established, and the infant has remained well since the abnormal urethral valves

were fulgurated cystoscopically. Review of the clinical and experimental literature reveals no consensus about how pleural fluid accumulates in the presence of obstructive uropathy, and this neonate showed a direct inverse relation between the amount of pleural fluid drainage and urinary output via catheter. Hydronephrosis should be considered diagnostically when a newborn has a pleural effusion that is otherwise unexplained. (J Am Bd Fam Pract 1989; 2:55-7.)

A male infant weighing 3000 g was delivered by primary Cesarean section at 38 weeks' gestation 8 hours after spontaneous onset of labor during which there were four episodes of sudden fetal bradycardia followed by tachycardia and poor beat-to-beat variability. The mother was 21 years of age with one previous pregnancy and delivery, which was uncomplicated. The current pregnancy was complicated only by an episode of cystitis during the second trimester.

At delivery, the amniotic fluid was thickened with meconium, but its volume was estimated to be normal. The newborn was suctioned immediately on the mother's abdomen, using the De Lee apparatus, and again on the infant warmer in the delivery room, with clear returns below the vocal cord. Apgar scores were seven at 1 minute and nine at 5 minutes. Initial physical examination of the infant was consistent with gestational age.

In the nursery one-half hour after birth, the newborn was noted to have respiratory distress with shallow respirations at a rate of 100 per minute. He occasionally grunted and displayed flaring nares and moderate substernal retractions. Physical examinations revealed a meconium-stained, pale, irritable neonate with diminished muscle tone. His heart rate was 140 beats per minute and no murmurs were heard. Breath sounds were clear on the right and minimally decreased on the left. No other abnormalities were noted. A chest radiograph showed a completely opacified left

hemithorax with rightward displacement of the heart silhouette and normally aerated right lung. Arterial blood gases drawn with hood oxygen at 68 percent showed the pH was 7.26; pO_2 , 54 mmHg; pCO_2 , 42 mmHg; and oxygen, 88 percent. Transportation to a tertiary-care facility was arranged. The transport neonatal specialist placed a chest tube into the infant's left pleural space with immediate return of approximately 25 mL of straw-colored, blood-streaked fluid. The tertiary center's admitting physician palpated a nondiscrete fullness in the baby's left flank. Radiographs of the abdomen showed no evidence of ascites or masses, but the small intestine was displaced anteriorly on the lateral view. The infant voided a good stream on two occasions during the first 16 hours, but then urine output diminished to infrequent low-volume dribbles during the first night. The chest tube output was more than 250 mL during the first 24 hours.

On day 2, a voiding cystourogram showed a left megaureter and hydronephrosis with blunted calyces. Mild ureteral dilation was observed on the right, and the bulbous urethra was dilated. No spontaneous voiding of the contrast medium occurred. Renal sonography confirmed cystic dilation of the left kidney with a one-half centimeter thickness of cortex remaining inferiorly. The right renal pelvis was considered normal, but fluid was seen in the right perinephric space and the bladder wall was thickened.

The diagnosis of posterior urethral valves was made, and the bladder was catheterized for decompression. The urine and chest tube output equilibrated for the next 2 days, then the chest

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tube output diminished proportionate to an increased urine output. By day 6, the chest tube output was almost nothing with the exception of one brief episode of rapid output that coincided with obstruction of the urinary catheter; however, it ceased very soon after the catheter was replaced.

The chemical composition of the thoracic fluid was initially identical to serum measurements of osmolality, sodium, potassium, chloride, bicarbonate, urea nitrogen, and albumin. On one occasion the laboratory reported results from a specimen probably mislabeled "chest tube drainage," which were consistent with urine electrolytes; however, a simultaneous sample labeled "urine" was similar in composition to previous chest fluid and serum samples. All subsequent chest tube drainage and analyses showed values comparable with serum.

The chest tube was withdrawn on the 10th day with no further accumulation of fluid. On day 15 transperineal fulguration of posterior urethral valves was done under cystoscopic visualization. The infant was discharged on day 20 and has done well since that time.

Discussion

The primary abnormality of posterior urethral valves was clinically recognized and described by Young and co-workers in 1913.¹ This anatomical anomaly consists of membranous "valves," which are actually exaggerated urethral ridges that obstruct urine outflow by means of a wind-sock effect. The obstructive valvular effect is greater at high pressures, so urine commonly dribbles at low pressure. Diagnosis of the condition in utero may be suspected when oligohydramnios is present. Uterine sonography may show a distended fetal bladder and renal system or abdominal ascites.

The usual presentation in neonates is an abdominal mass, or later, low urine output, dribbling, and urinary infection. Hydronephrosis is common with the primary obstructive uropathies. Neonates may occasionally present with accumulated peritoneal "ascites" with or without demonstrable communication from the renal-collecting system.² An early description of the delivery of infants with abdominal ascites secondary to hydronephrosis was written by Hicks in 1863.³ His report included an infant with probable posterior urethral valves who had bladder distension and ascites. The reason for the inability to drain the bladder was not recognized at that time, be-

cause at autopsy, no impediment could be found in the urethra; "A wire passed quite readily from within to without."^{3(p 288)}

A rare complication of hydronephrosis in adults is pleural effusion with, and on occasion without, concomitant retroperitoneal fluid.⁴⁻⁸ In some instances, the pleural fluid has been reported to be the same composition as urine, with urea nitrogen levels greater than in serum.^{4,5,9,10}

Pleural effusions in newborns are unusual and most often are due to spontaneous chylothorax, thought to be secondary to lymphatic trauma at delivery. There is one previous report of a neonatal boy who presented at 3 weeks of age with poor weight gain, dribbling urine, and gradually developing respiratory distress.¹¹ He was found to have fluid in his pleural cavity with a composition comparable with urine. The effusion resolved after adequate bladder drainage.

It is unclear how pleural effusion evolves in conjunction with hydronephrosis. Experimental data confirm formation of pleural fluid by urinary obstruction. Stoerk, et al. found that bilateral ureteral ligation in rats always resulted in peritoneal ascites and usually pleural effusions within 48 hours; however, the composition of the thoracic fluid was not reported.¹²

Corriere, et al. reported no development of thoracic fluid up to 35 days in a similar experiment with dogs in which one ureter was obstructed and either the peritoneal lymphatic drainage or the pleural lymphatic drainage was disrupted.⁷ However, one dog with ureteral and right lymphatic obstruction developed pleural fluid 11 months later. The other dogs had been sacrificed and further confirmation was not possible. The authors postulated that the delay in development of the pleural effusion was possibly due in part to the anatomical difference in the location of canine kidneys in relation to the diaphragm. It is also possible that the lymphatic obstruction rather than the hydronephrosis was the factor influencing thoracic fluid accumulation in that instance.

It is curious that in some instances of hydronephrosis, pleural fluid is found in the absence of peritoneal or retroperitoneal fluid. This has led to speculation that there may be a circulating factor that stimulates the formation of pleural fluid, although none has been identified.

Hydrothorax has also been found accompanying hepatic abdominal ascites and following instillation of fluid in peritoneal dialysis.^{13,14} Cases in which pleural fluid is found in association with

peritoneal or retroperitoneal fluid raise questions about the method by which the thoracic fluid forms. It is possible that the development of pleural effusion is a separate transudative phenomenon in response to an irritative effect on the diaphragm and hence the pleura.

Another possibility is that the abdominal fluid may directly pass into the pleural space. Although often no portal can be seen radiographically or identified at the time of surgery or autopsy, it appears that some extrusion through minuscule membrane rents must occur on occasion. There have been reports of pneumothorax complicating therapeutic pneumoperitoneum in which no observable communication was found.^{15,16} The collected urine or other fluid may then equilibrate with serum as a dialysate. Different rates of accumulation or equilibration may explain why the fluids in the two compartments are not always identical in composition.

Some observers have argued that the abdominal fluid traverses the diaphragm directly via lymphatics. Experimental evidence for this hypothesis was provided more than 50 years ago in a test in which particulate matter appeared on the pleural surface of dog diaphragms within 3 minutes after instillation on the peritoneal side.¹⁷

Summary

Pleural effusion is a very rare presentation of urinary obstruction. The present case is unique in that the newborn declared the anomaly of his urinary tract initially with respiratory difficulty and a large hydrothorax, which was later found to be a complication of urethral obstruction. A small right perinephric fluid collection was noted on sonography of his abdomen, and no communication with the renal system could be shown. The right kidney and ureter were spared most of the dilation suffered by the left renal system, perhaps due to decompression through fluid discharge into the perinephric space as well as that provided by the massive dilation on the left.

The pleural effusion in this case was on the contralateral side to the observed retroperitoneal fluid and was ipsilateral to the hydronephrotic kidney. No fluid was collected from the right perinephric cavity for study and no further investigations were performed on this otherwise healthy infant to discern the source of the pleural fluid.

Although the mechanism for formation of hydrothorax secondary to urinary obstruction is not

certain, the latter appears to have caused the former in this case. The chest tube drainage diminished commensurate with an increased urine output after bladder catheterization. Also on one occasion after the chest tube drainage had stopped, it recommenced simultaneously to ceased urine output due to catheter obstruction and stopped immediately upon reinstitution of bladder drainage.

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