Short Reports

103

Surgical closure is associated with a very low mortality and the results are curative.

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REFERENCES


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**Urinary Ascites Complicating Spina Bifida**

Urinary ascites is a rare complication of congenital urinary tract obstruction with a high mortality when present at birth. Since Lord (1953) first drew attention to the connexion between fetal ascites and urinary obstruction over 40 cases of urinary ascites have been reported, nearly all males with posterior urethral valves, but the condition has never been described as a complication of myelodysplasia. This report describes a successfully treated female infant born with a lumbosacral meningomyelocele and urinary ascites, and discusses the pathogenesis of the ascites and its treatment.

**Case Report**

A full-term female infant weighing 3 kg with a lumbosacral meningomyelocele, hydrocephalus, and severe abdominal distension was admitted a few hours after birth. The distension had been present at birth and had caused respiratory distress after birth. She was slightly cyanosed and obviously hydrocephalic, with an occipito-frontal head circumference of 35 cm. There was a large lumbosacral meningomyelocele with almost total paralysis of both lower limbs and a lax anal sphincter. The abdomen was grossly distended and shifting dullness and a fluid thrill were present. Plain x-ray of the abdomen showed obvious free fluid in the flanks and no distension of the bowel. At abdominal paracentesis 220 ml yellow ascitic fluid with a urea content of 65 mg/100 ml was aspirated, and since the plasma urea was 35 mg/100 ml it was concluded that this fluid contained urine.

At operation on the day of admission the meningomyelocele was closed and a further 140 ml fluid aspirated from the peritoneal cavity. A urethral catheter was passed but failed to drain urine from the bladder and the ascitic fluid reaccumulated. An intravenous pyelogram on the second day of life showed a normal upper urinary tract, but a cystogram showed a leak of contrast medium from the fundus of the bladder into the peritoneal cavity (Fig. 1). The site of the urinary leak could not be found when the bladder was explored and a suprapubic catheter was inserted and drained urine satisfactorily. Ventriculo-atrial drainage using a Holter valve was established on the fourth day of life to control the hydrocephalus. A repeat cystogram on the 20th day showed no leak from a bladder with the typical neurogenic features of trabeculation, a wide bladder neck, and funnelled urethra (Fig. 2), and after urethral dilatation to reduce outflow resistance the suprapubic catheter was removed. She now empties her bladder adequately without expression and at the age of 6 months has no evidence of renal impairment.

**Discussion**

Urinary obstruction in almost all recorded cases of urinary ascites has been at the bladder outlet and has given rise to upper urinary tract dilatation. The presence of urine in the ascitic fluid may be suspected from its urea content, which is greater than that of the plasma and confirmed by a micraturating cystogram and intravenous pyelogram which, besides demonstrating the nature of the obstruction and its effect on the upper urinary tract, may reveal the site of the leak.
Posterior urethral valves, which were present in 13 (probably 14) of the 19 cases reviewed by Cywes, Wynne, and Louw (1968), are the commonest cause of urinary ascites; other causes described are absence of muscle from the posterior bladder wall (France and Back, 1954), urethral atresia (Easton, 1960), a ureterocele producing a ball-valve obstruction of the urethra (North, Eldredge, and Talpey, 1966), and ureteric stenosis (Linde, 1966). The bladder had been the source of leakage in only 3 published cases.

Back-pressure effects due to bladder outflow obstruction are common in the neurogenic bladder of myelodysplasia, and since the neural defect is present prenatally these effects are frequently evident in the neonate, Harlowe et al. (1965) finding bladder trabeculation in 35 of their 55 neonatal spina bifida patients and upper urinary tract dilatation in 18. It is therefore surprising that spina bifida has not been described as a cause of urinary ascites.

In the infant described, the absence of back-pressure effects on the upper urinary tract indicates that the leak probably occurred a considerable time before birth.

When urinary ascites is present at birth the mortality is extremely high (87.5% in the collected series of Cywes et al., 1968) due partly to respiratory embarrassment caused by abdominal distention and partly to impaired renal function. Prompt relief of this distension and effective drainage of the urinary tract is essential. The method of draining the urinary tract depends on the site of the leak which must first be established by intravenous pyelography and cystourethrography. When this is in the upper urinary tract or is uncertain, bilateral nephrostomies or cutaneous ureterostomies should be performed, but if leakage has occurred from the bladder supra-pubic cystostomy should provide adequate drainage; drainage of the bladder by an indwelling urethral catheter is unreliable in the neonate, as the urethra will accept only a small catheter which is very liable to blockage. If the leak does not close spontaneously, repair can be undertaken and the
cause of the urinary obstruction treated when the child is fit.

Summary
A case of urinary ascites associated with myelodysplasia is described. The leakage of urine had occurred from a neurogenic bladder.

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REFERENCES

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Peripheral Gangrene in a Newborn

Peripheral gangrene in the newborn, unassociated with dehydration, is rare. The condition was first described in 1828 by Martini (as quoted by Gross, 1945). Since then about 65 cases have been reported (Smith et al., 1965; Okojie, 1967; Vittoria, Cotrufo, and Spampinato, 1965; Gilbert et al., 1970).

Case Report
This infant is the fifth child of healthy, white parents. At 28 weeks' gestation the mother's membranes ruptured. A circumferential suture was placed in the cervix. Within 3 days a purulent vaginal discharge developed and the suture was removed. She immediately went into labour and the baby was born precipitately and by the breech. When seen by the general practitioner, he found evidence of respiratory distress and gangrene, and hospital transfer was arranged.

On admission the patient was an ill, male, premature infant weighing 1.29 kg. He was cyanosed, breathing rapidly (90/min), with retraction of the chest wall, diminished breath sounds, and crepitations over both lung fields. The heart sounds were normal, no murmurs were heard and all peripheral pulses were palpable (including both posterior and anterior tibial arteries). There was complete gangrene of all the toes of the left foot (Fig. 1), but apart from oedema and cyanosis there was no abnormality of the other limbs.

A diagnosis was made of respiratory distress due to aspiration or congenital pneumonia, and peripheral gangrene of unknown origin. Treatment was with oxygen, penicillin, and kanamycin. No local treatment was given for the gangrene.

Investigations. Results of investigations are set out in the Table.

<table>
<thead>
<tr>
<th>Day</th>
<th>Platelet Count mm$^2$</th>
<th>Haematocrit (venous) %</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>32,000</td>
<td>63</td>
</tr>
<tr>
<td>3</td>
<td>124,000</td>
<td>60</td>
</tr>
<tr>
<td>4</td>
<td>140,000</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>142,000</td>
<td>52</td>
</tr>
</tbody>
</table>

VDRL test for syphilis: negative.
Skin swabs and blood culture yielded no bacterial growth.

Clinical course. His response to treatment was satisfactory, and by the fourth day he maintained a good colour without added oxygen. On day 6, penicillin and kanamycin were stopped and chloramphenicol was started, because of clinical evidence of a recrudescence of inflammation of the gangrenous foot, despite therapy. The chloramphenicol was stopped after 5 days.

During his first 3 weeks in hospital he had 4 brief episodes of vomiting, constipation, and abdominal distension, all of which responded to the oral administra-
Urinary ascites complicating spina bifida.

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