Short reports

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Neonatal ascites from spontaneous rupture of the bladder

Association between neonatal ascites and abnormalities of the genitourinary tract, particularly of the lower urinary tract obstruction in the aetiology of the condition was clearly shown by Lord (1953), but in this group spontaneous rupture of the bladder is extremely rare. Two infants, one male and one female, with spontaneous rupture of the bladder and neonatal ascites are here reported.

Case reports

Case 1. A male infant of 1·9 kg was delivered by vacuum extraction after spontaneous onset of labour at 32 weeks' gestation. There were no immediate problems but on the second day of life he was transferred to this hospital because of progressive abdominal distension and failure to pass urine. Marked ascites was present and abdominal paracentesis produced 50 ml straw-coloured fluid with a urea content of 29·6 mmol/l. The infant's general condition remained good, and apart from a moderate jaundice no complications of prematurity developed. Fluid continued to drain freely from the paracentesis site, and the abdominal distension and anuria persisted.

An intravenous urogram showed satisfactory contrast excretion from two normal kidneys and the bladder outline appeared normal. Micturating cystography clearly showed a leak of contrast from the posterior bladder wall, otherwise the bladder and urethra were normal (Fig. 1). A suprapubic catheter was then passed into the bladder and a drain inserted into the peritoneal cavity. The abdominal distension subsided as fluid drained from the peritoneum, and urine continued to be passed via the suprapubic catheter.

Fig. 1.—Case 1. Micturating cystogram showing leakage of contrast from the posterior wall of the bladder.
The infant made an uninterrupted recovery and after the catheter had been removed 17 days later he passed urine normally through the urethra. He is now aged 7 years, and has normal renal function and a normal pyelogram series.

**Case 2.** A female infant of 2.4 kg was born by assisted breech delivery at an estimated 32 weeks' gestation. The pregnancy had been complicated at 30 weeks' gestation by a laparotomy for drainage of an appendix abscess. There were no perioperative problems. Marked abdominal distension due to massive ascites was immediately evident, but there were no other abnormalities. Despite initial spontaneous respiration, progressive respiratory embarrassment occurred soon after birth and necessitated mechanical ventilation by endotracheal tube.

Abdominal x-ray confirmed the presence of ascites, and 300 ml pale yellow fluid were obtained by paracentesis. Synchronous biochemical examination of ascitic fluid and plasma gave the following results (Table).

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<tr>
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<th>Ascitic fluid</th>
<th>Plasma</th>
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<tbody>
<tr>
<td>Protein (g/l)</td>
<td>9.0</td>
<td>50</td>
</tr>
<tr>
<td>Sodium (mmol/l)</td>
<td>128</td>
<td>127</td>
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<tr>
<td>Creatinine (μmol/l)</td>
<td>247.5</td>
<td>194.5</td>
</tr>
<tr>
<td>Urea (mmol/l)</td>
<td>13.1</td>
<td>11.0</td>
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A urethral catheter was then passed but initially urine was not obtained. An intravenous urogram at the age of 14 hours outlined poorly two apparently normal kidneys. Contrast material injected into the bladder by way of the urethral catheter showed a leakage from the peritoneal cavity.
fundus of the bladder into the peritoneal cavity (Fig. 2). Thereafter urine continued to drain freely from the catheter, and the child was successfully weaned off the ventilator. Ascites accumulated again and on the second day of life a further 12-hour period of assisted ventilation was required. On the fifth day the infant was transferred to this hospital for further assessment.

Repeat abdominal paracentesis was performed and thereafter the abdominal distension gradually lessened and urine drained freely in the urethral catheter. No surgical intervention was thought necessary at that time. Recovery was temporarily slowed by three complications—paralytic ileus, transient renal tubular defect with polyuria and urinary sodium loss, and coliform urinary infection. Each responded satisfactorily to appropriate therapy. The urethral catheter was removed on the 13th day, and she was able to pass urine normally thereafter.

The child is now 7 months old, is well, and has normal renal function. A repeat intravenous urogram has shown completely normal appearances.

Discussion

Most cases of urinary ascites may be grouped into three categories (Weller and Miller, 1973). Firstly, there are the majority of patients who have posterior urethral valves and obstruction. A second group of those with complex caudal anomalies, usually anorectal and urethral atresia. A third group of those with miscellaneous lesions which include bladder outlet flaps, ureterocele, ureteral atresia, myelomeningocele with neurogenic bladder, and extrinsic lesions such as presacral neuroblastoma. Spontaneous rupture of the bladder in the newborn must be extremely rare and our search of published reports showed only 4 reported cases to date (Miller et al., 1960; Gandhi, 1964). Only 2 liveborn females have been described previously with urinary ascites, and both these children had bladder outflow obstruction (Baghdassarian, Koehler, and Schultz, 1961; Howat, 1971).

Bladder rupture in our 2 cases must have occurred in utero or possibly during the birth process, though it is difficult to accept that during this premature labour, there would be sufficiently increased hydrostatic pressure within the urinary tract to cause the bladder to rupture. In the second child it is not likely that the uneventful laparotomy can be implicated as a cause of the bladder rupture. A question must be raised as to whether or not there was an inherent weakness of the bladder wall present in these children.

The overall mortality in the few reported series of neonatal ascites has been high. Infants with massive ascites at birth do less well, only 1 of 8 infants in one series surviving, whereas the prognosis is better if ascites develops in the first few days of extrauterine life (Cywes, Wynne, and Louw, 1968).

Summary

Two premature infants who presented with neonatal ascites due to apparent spontaneous rupture of the bladder are described. In both children the site of bladder rupture was clearly shown, but neither at time of presentation nor at subsequent review at age 7 years and 7 months, respectively, was any other renal tract abnormality detected.

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References


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Congenital midline scalp and skull defect

Congenital midline scalp and skull defects have been reported periodically since 1826 (Greig, 1931) and the published reports have appeared mainly in America. In this communication we record the presence of this congenital anomaly which has occurred in a family over two generations.

Case report

In the first stage of labour, in this mother's third pregnancy at term, the midwife reported that the fetal skull felt abnormal per vaginam and at delivery two abnormal areas were found to be present across the midline at the vertex. The abnormal areas measured

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