Table Enterovirus neutralising antibody titres of immunoglobulin batch (GG 219) used

Virus (100 TCD 50)	Titre
Echovirus type 6	1/64
type 11	1/32
type 20	1/8

We wish to thank the many members of the medical, nursing, and laboratory staff who helped with this case.

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Neonatal urinary ascites

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SUMMARY Two neonates with spontaneous rupture of the bladder and an otherwise normal genitourinary tract are described. Conservative management resulted in complete resolution of the lesion in one but the other child died from a coliform septicaemia. Necropsy showed a discrete ischaemic lesion in the fundus of the bladder.

Rupture of the bladder in the newborn and secondary urinary ascites is a well recognised, if uncommon, complication of obstructive lesions of the genitourinary tract.¹ Spontaneous rupture of the bladder in an otherwise normal infant is rare,²⁻⁵ and previous reports have not suggested a possible mechanism. We describe two further cases presenting with urinary ascites and an otherwise normal genitourinary system, and suggest that the bladder lesion was ischaemic in origin.

Case reports

Case 1. A girl (the second twin) weighing 1.46 kg was delivered by Kielland's forceps after the spontaneous onset of labour at 30 weeks' gestation. The pregnancy had been otherwise uneventful. She developed severe hyaline membrane disease requiring mechanical ventilation from birth for 19 days. This was complicated by a right sided pneumothorax

on day three, responding to chest drainage. On day 10 she became unwell with hypotension, hypoxia, and abdominal distension. There was clinical evidence of a patent ductus arteriosus, with a systolic murmur and bounding pulses. Blood cultures subsequently grew *Staphylococcus albus*. After treatment with antibiotics and intravenous indomethacin, there was rapid clinical improvement and the murmur resolved. The abdominal distension was thought to be caused by intestinal obstruction with hard faeces, and it resolved after stools were passed.

At age 24 days she developed abdominal distension due to ascites and became oliguric. Biochemical analysis of the ascitic fluid compared with plasma and urine suggested that the ascitic fluid was urinary in origin. (Table). An intravenous urogram was normal, but a micturating cystogram showed leakage of urine from the fundus of the bladder. A small diverticulum of the bladder was also detected but this was not associated with the point of rupture.

Table Biochemical values for plasma, ascitic fluid, and urine taken simultaneously in case 1 on day 25

	Plasma	Ascitic fluid	Urine
Sodium (mmol/l)	124	119	109
Potassium (mmol/l)	4.0	8.8	19
Urea (mmol/l)	9.5	20	35
Creatinine (μmol/l)	245	882	1400
Protein (g/l)	53	7	5

Treatment was conservative with paracentesis and continuous bladder drainage via a urethral catheter. The rupture healed both clinically and radiologically after two weeks. Although initial urine culture was sterile she subsequently developed colonisation of the urine with *Staph albus* and *Escherichia coli* but this cleared with antibiotic treatment. At the age of 6 months renal function was normal biochemically and radiologically. There was no evidence of any diverticulum and there has been no further urine infection.

Case 2. A boy weighing 2.83 kg was delivered by Kielland's forceps with a grossly distended abdomen containing 400 ml of sterile, acellular ascitic fluid after spontaneous onset of labour at 33 weeks' gestation. Pregnancy had been complicated by abdominal pain and reduced fetal movement at 27 weeks' and at 31 weeks' gestation, although a cardiotocograph trace at 32 weeks' was unremarkable. Oligohydramnios and a retroplacental clot were noted at birth. Serial biochemical analyses showed that the ascitic fluid was renal in origin and ultrasound scans of the renal tract were normal. He remained, however, oliguric, fluid continued to accumulate in the abdomen, and the plasma creatinine concentration continued to rise until a urethral catheter was passed 56 hours after birth. Thereafter his urinary output and creatinine clearance returned to normal and no further fluid accumulated in the abdomen. Ventilation was necessary for six days from birth because of severe hyaline membrane disease, but progress was uneventful until the ninth day when he collapsed with fulminating coliform septicaemia and died very rapidly.

A micturating cystogram after death was normal and necropsy confirmed that the urinary tract and the kidneys were essentially normal. There was a very small vestigial valve-like flap on one side of the posterior urethra that was non-obstructive. The bladder wall was not trabeculated but there was a discrete focal lesion in the muscular wall of the fundus of the bladder about 2 mm in diameter. Histology of this necrotic area showed a lightly calcified healing area of ischaemic muscle that had probably allowed urine to leak into the peritoneal cavity. There was no overt rupture of the bladder architecture, and no sign of inflammatory change other than a thick coat of fibrin on the peritoneal surface of the bladder and other intraperitoneal organs consistent with acute peritonitis.

Discussion

Both the babies described have had an atraumatic rupture of the fundus of the bladder that manifested

itself at a postconceptional age of about 33 weeks. In the first the leak developed after a 'stormy' period during which there were definite episodes of hypoxia and hypotension. No attempt was ever made to catheterise the umbilical arteries, to aspirate the bladder, or to express the bladder manually. In the second patient the obstetric history is consistent with fetal hypoxia and the histological appearance of the bladder suggests an ischaemic insult.

In none of the previous cases has the histological appearance of the lesion been described,²⁻⁵ or a mechanism for the lesion postulated. No information is available regarding the gestational age or weight of two of the six previously published cases but four of the six babies of known gestation were preterm and it may be that the vascular supply of the fundus of the bladder is particularly vulnerable at this time. Nor is much known about the clinical circumstances surrounding pregnancy and delivery in the previously reported cases (although one baby was delivered two weeks after the mother had a laparotomy for an appendix abscess at 30 weeks' gestation).³ Any suggestion that the previous cases were also due to bladder ischaemia is therefore speculative. In three of the four previously reported cases where the site of the lesion was identified it was fundal in origin, as it was in both our patients. The remaining case involved the posterior bladder wall.

The diagnosis of ascites as a cause of abdominal distension in neonates is readily made clinically and by ultrasound. Establishing the cause of the ascites is important for planning further management. Useful information may be gained from an analysis of the ascitic fluid (Table) because only in urinary ascites can the urea and creatinine concentrations of the ascitic fluid exceed those of plasma (although, because of the back diffusion across the peritoneum, these concentrations will be midway between those for urine and plasma). An additional point of differentiation is that with ascitic fluid of other origin the protein concentration is usually much higher. The diagnosis of a ruptured bladder can, if necessary, be confirmed with a micturating cystogram. Conservative management of this condition is the treatment of choice. The bladder should be catheterised to prevent distension and allow healing. Paracentesis will reduce abdominal distension and allow analysis of the ascitic fluid but need not continue once passage of urine through the urinary catheter is established. Great care is required to ensure correct fluid balance.

Spontaneous rupture of the bladder in the newborn carries a good prognosis provided there are no immediate complications and no associated abnormalities.

We are grateful to Dr D Scott for information on the histology in Case 2.

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Passenger safety in cars

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SUMMARY Comparison of child passengers between January 1983 and 1984 showed an increased use of rear safety restraints after the wearing of front seat belts became mandatory. In 1984, however, only 25% of children were restrained, most commonly in a safety seat.

Method

During one week in January 1983 and a corresponding week in 1984, parents arriving at the casualty department with their children completed a questionnaire about travel safety in their family car. Details were obtained about the use of safety restraints relating to: the age and number of children per family, their usual seating arrangements, the use of safety locks on rear car doors, the types of safety restraints possessed, the problems of installing and using them, and any changes following the act enforcing wearing of front seat belts after January 31 1983.

Results

In Sheffield the equivalent of one in four children attend a casualty department each year with medical, surgical, or traumatic problems, and 70% of families possess cars. In the 1983 survey 325 families (709 children) completed the questionnaire compared with 337 families (743 children) in 1984. An increased number of families possessed car safety restraints in 1984 (114 out of 337 (34%)) compared with 1983 (86 out of 325 (27%)) ($P < 0.05$, χ^2).

In 114 families using restraints in 1984, only 76 restrained *all* children—only 201 restraints were available for 237 children. There were 16 redundant

restraints which children had outgrown, for example carrycot straps or rear seat belts inappropriate for the toddler. Thus 185 out of 743 children (25%) were potentially restrained, based on parental reporting. In the remaining 38 families the young children were preferentially restrained.

Ninety seven of the 114 parents (85%) used the restraints regularly—only four expressed difficulties sufficient to abandon using these, for example the toddler undoing the buckle or restriction of his visual field and movement.¹ The commonest restraint in use was the safety seat (age 9 months to 4 years; weight 9 to 18 kg.) (Table 1).

In 1983 child proof rear door safety catches were used by 39 out of 60 families (65%) who had four door cars and children already restrained, compared with only 82 out of 169 families (48%) whose children lacked restraints ($P < 0.05$, χ^2). Fifty five of the 142 (39%) children aged under 5 years were unrestrained in the rear of the car.

In the 1984 survey the larger families used restraints less frequently—44% of 64 single child families were restrained, 28% of 168 two child families, and 11% of 25 families with more than three children used restraints. The older the child the less rear restraints were used (Table 2).

Table 1 *Type of restraint and number potentially in use by 114 families consisting of 237 children (January 1984)*

Type of restraint	No
Carrycot straps	2
Child Safety Seat	65
Child Safety Harness	47
Rear Generation Belt (adjustable for child or adult)	20
Rear Adult Belt and Booster Cushion	13
Rear Adult Belt	38
Total number of restraints	185