ate level, bearing in mind social class and education. It seems inadequate merely to ask whether parents are coping well with the diet since there was a curious disparity between their perception of the diet and their ability to ensure compliance. While the proportion of non-compliers has been relatively large in this survey, it is important to emphasise the comparatively disadvantaged nature of the social group. Our findings, therefore, may not be readily applicable in other communities where families may be more affluent and parents better informed. Nonetheless, it is salutary to reflect upon the proportion of patients who, in spite of regular dietetic counselling, seem unable to comply with the diet, the reasons for which seem largely social and educational.

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Prevention of spread of echovirus 6 in a special care baby unit

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SUMMARY A case of fatal neonatal infection with enteric cytopathogenic human orphan virus (echovirus) type 6 is presented. The measures taken to prevent further spread of infection with special reference to the use of human normal immunoglobulin are described.

Since the 1960s enteric cytopathogenic human orphan viruses (echoviruses) have been associated with a spectrum of illness in neonates, ranging from milder disease through respiratory infections, meningitis, 'sepsis', to fulminant fatal disease. Although type 11 virus¹ predominates, others, including type 6, have been implicated.^{2–4}

Outbreaks of infection have occurred in special care baby units where the virus has been introduced by an infected mother.^{1,5} She or other family members may^{3,5} or may not have symptoms.

We report a fatal echovirus type 6 infection in a neonate and describe measures taken to prevent spread in the special care unit including the use of human normal immunoglobulin prophylaxis.

Case report

A girl weighing 2150 g, born by normal delivery at

35 weeks' gestation to a healthy mother, developed transient respiratory distress which resolved by the third day. Then, fully bottle fed, she was transferred to the transitional care ward to be with her mother.

On day five she became unwell, with suspected septicaemia. She was admitted again to the special care unit and was treated with broad spectrum antibiotics. She continued to deteriorate, required ventilation, and developed disseminated intravascular coagulation. She was treated with infusions of platelets, plasma, and fresh blood, with little response.

By day seven she was still being ventilated and had developed hepatomegaly and ascites. On day nine she deteriorated, becoming anuric, severely acidotic, and hypotonic. Cranial ultrasound showed bilateral intraventricular haemorrhage with ventricular dilatation and parenchymal extension. She died the next day.

Pathology. At necropsy the findings were those of massive haemorrhagic necrosis of liver and adrenals, bilateral renal medullary haemorrhage, and bilateral intraventricular haemorrhage with extension into the brain substance on the right side. Multiple fibrin thrombi were present in small pulmonary vessels but there was no associated pulmonary haemorrhage or infarction.

A urine specimen collected three days before death and post mortem viral cultures from lungs, liver, spleen, kidneys, myocardium, and peritoneal fluid all yielded echovirus type 6. No virus was isolated from stool specimens or brain tissue.

Echovirus type 6 was isolated in faeces collected from the mother, although neither she nor other family members complained of recent illness. No maternal blood was available for antibody testing.

Control of infection. When first admitted to the special care baby unit the infant shared a room with three other babies. On becoming ill, she was transferred to an isolation cubicle on the unit and barrier nursed. The other contact infants remained well. When echovirus was isolated action was taken to contain a possible outbreak.

Measures taken.

Other infants

Stool specimens were taken immediately from all babies on the unit and the transitional care ward. These were repeated weekly until one month after the last positive specimen.

All contact babies of the infant or mother, received a single, intramuscular dose of pooled human immunoglobulin (250 mg protein in 1.7 ml; Blood Products Laboratory, Elstree) as did all babies admitted to the special care unit for one month after the last positive specimen.

The unit was closed to outside referrals and only four babies were admitted during the two weeks following the infant's death.

No babies were transferred to other hospitals, although asymptomatic babies were allowed home.

Mothers. All booked high risk pregnancies were transferred to another hospital, so that the only admissions to the unit were from unforeseen neonatal problems arising within the hospital.

Nursing and medical staff. Interchange of staff with other units was stopped. Viral culture was not done on asymptomatic staff, those becoming ill were sent home until they were asymptomatic and current enterovirus infection had been excluded.

Results

Forty six infants were investigated for enterovirus excretion. No echovirus type 6 was recovered, nor did any infant develop illness attributable to enterovirus infection.

Echovirus type 20 was isolated from stool specimens of healthy preterm twins. None of their family

had been unwell nor was the virus isolated from their mother. No other enteroviruses were isolated from any other babies in the special care baby unit.

Echo virus type 6 was isolated in a stool specimen from a nurse who developed an influenza-like illness 10 days after nursing the baby. A further specimen taken three weeks later was negative, and she returned to work. A physician and three nurses had short illnesses with fever and diarrhoea, stool samples were negative. The ward remained closed to outside admissions for two weeks, only reopening when no further cases occurred and no asymptomatic excretors were found.

Discussion

Enterovirus infection was first suspected and barrier nursing was immediately instituted because of the time of onset and severity of the illness. The clinical course and necropsy findings¹⁻³ supported this diagnosis, although virological confirmation was not obtained until the day of the infant's death and serotyping for another three days. The early findings made us suspect an echovirus type 11 infection, but although we had previously isolated this virus from clinical specimens the prevalent enterovirus in Bristol before this infant's death had been type 6, which has² more rarely been implicated in identical disease. Recently there were two cases of echovirus type 7 in Bristol, causing similar fatal illness.

In view of the experience of others^{4,6} with outbreaks of echovirus type 11 within a special care baby unit, we felt that normal human immunoglobulin should be given to all contact infants. On identification of a type 6 virus we still felt that extreme caution should be exercised to prevent an outbreak in the unit.

Over the past two years we have seen that the echovirus most prevalent in our community (1983—echovirus type 6; 1984—type 7) has caused fatal neonatal infections. Since absence of maternal neutralising antibody is related to severe infection in babies and since with echovirus type 11 this may be prevented by injections of antibody, present in normal immunoglobulin,⁶ the use of normal immunoglobulin prophylaxis in other infants in the special care unit seems justified when preliminary laboratory results, or even strong clinical and epidemiological factors suggest enterovirus infection.

The batch of immunoglobulin used contained satisfactory levels of antibody against echovirus type 6 (Table) and it is recommended that new batches are checked for antibody against enteroviruses present in the community that season, particularly if these serotypes are known to cause severe neonatal disease.

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