copper is also grossly raised. We were able to re-examine the necropsy histology of this child, and found it indistinguishable from the changes seen in ICC.

The boy’s copper toxicity was attributed to the ingestion of water which had a high copper concentration. Although the family’s well water contained little copper, it had a pH of 3.8-4.8, and dissolved copper from the domestic plumbing. Three explanations may be offered for the fact that his parents and his 4-year-old brother were unaffected. Firstly, he may also have had Wilson’s disease, as suggested by a plasma caeruloplasmin concentration of 150 mg/l. However, this may be significant in the presence of a serum albumin concentration of 21 g/l. Plasma caeruloplasmin concentration is normal in patients with ICC. Secondly, since the neonate has a modestly increased hepatic copper content, there may be greater susceptibility to copper intoxication in infancy. Thirdly, he had been fed on cows’ milk diluted with water, and it is known that the availability for absorption of copper in milk is greater than in other foods.

This case report therefore shows that a disease state strikingly similar to ICC may be produced by excessive copper ingestion, but leaves unresolved the question—Can this occur in normal infants or only in those genetically predisposed?

We thank Dr Pat Bale, Royal Alexandra Hospital for Children, Camperdown, New South Wales, for providing the necropsy specimens.

References


Pneumopericardium in a neonate not artificially ventilated

Sir,

Because the incidence of pneumopericardium in neonates has been increasingly reported since the advent of positive pressure-assisted ventilation in the treatment of hyaline membrane disease, we report a case of pneumopericardium in a neonate in the absence of any form of assisted ventilation.

A 1766-g baby boy was the first of twins born to a 24-year-old gravida 3, para 2 female whose pregnancy was complicated by spotting and a urinary tract infection in the first trimester. The infant was delivered vaginally without difficulty. The Apgar scores were 7 at one minute and 7 at five minutes. On admission to a hospital at another hospital, the infant appeared to be cyanotic and plethoric and had grunting respirations. He was given oxygen by hood to relieve cyanosis. Haemocrit was 80% assumed to be high on the basis of fetofetal transfusion. He was phlebotomised 15 ml and this volume was replaced with Plasmanate (Cutter Laboratories) resulting in a haemocrit of 69%. He was then transferred to the Children’s Mercy Hospital where the initial examination showed a plethoric infant of about 35 weeks’ gestation. He was in mild respiratory distress with audible grunting and intercostal retraction. The initial chest x-ray at age 8 hours showed a fine reticulogranular pattern in both lungs with slight diffuse atelectasis (Fig. 1). Arterial blood-gases and pH on 40% oxygen by hood were pH 7-28, Po2 48 mmHg (6.4 kPa), and Po2 90 mmHg

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Fig. 1 Chest x-ray at 8 hours showing fine reticulogranular pattern of diffuse atelectasis.
At 32 hours a repeat chest x-ray showed more diffuse lung disease with absence of interstitial emphysema. At 41 hours the infant began to cry, became deeply cyanotic, and remained so on 100% oxygen. Heart rate and respiratory rate increased from 140 to 160 beats/min and from 60/80 breaths per min. Breath sounds were clear and equal bilaterally and the peripheral pulses were absent. A chest x-ray showed a large pneumopericardium without pneumothorax or pneumomediastinum (Fig. 2).

A No. 22 Jelco (Jelco Laboratories, Raritan, NJ) catheter was placed in the pericardial sac and approximately 5 cm air aspirated. The infant responded quickly with improvement of colour, return of pulses, and a decrease in heart and respiratory rates. The catheter was connected to a water seal and intermittent aspiration was required to maintain patency. Within 2 hours the infant again deteriorated secondarily to a massive right pneumothorax which responded to placement of a chest-tube. The pericardial catheter was removed after 48 hours without recurrence of the pneumopericardium. The infant was breathing room air by day 6 and, after removal of his chest tube, had no further problems.

There has been only one other case report of a pneumopericardium in infancy in the absence of resuscitative procedures or assisted ventilation. Our patient had a fairly typical clinical presentation with hypotension, absent pulse pressure, increased respiratory distress, and cyanosis. His tachycardia was an unusual finding, bradycardia being more commonly described. Intense cyanosis and agitation with hypotension, poor peripheral perfusion, muffled heart tones, and normal breath sounds are the key elements of clinical diagnosis in pneumopericardium alone, but some infants are asymptomatic and the diagnosis is made incidentally on routine roentgenological examination. Infants without evidence of cardiac tamponade may be managed conservatively but in those who deteriorate rapidly, pericardiocentesis is imperative.

References


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Perinatal listeriosis

Sir,

We agree with Robertson et al. (Archives, 1979, 54, 549) that perinatal listeriosis may be under reported (or even underdiagnosed) in Britain. This belief is supported by two further cases which must have occurred at about the same time in an adjacent health district to that of Robertson et al.

Case 1

A 29-year-old music teacher had a severe cough in late pregnancy. Two weeks later in the 37th week of pregnancy she went into spontaneous labour, which was accompanied by fever and generalised aches. Liquor stained with meconium was drained and emergency caesarean section resulted in the birth of a baby girl weighing 3·49 kg. Her Apgar score was only 4 at one minute. She had increasing respiratory difficulty, unresponsive to treatment of hypoglycaemia. X-ray of chest showed diffuse coarse mottling, and Listeria monocytogenes was cultured from the infant's blood, eye swab, umbilicus, and nose, as well as from the mother's vaginal swab. CSF was sterile on culture, but two bacteria were seen on microscopical examination. IV chloramphenicol (50 mg/kg per day) and ampicillin (400 mg/kg per day) were given for 2 weeks. For several days she had diarrhoea, abdominal distension, enlarged liver, irritability, and fits, with intractable hypocalcaemia, although slowly her condition improved. Now at almost one year she is developmentally and physically normal. Three months before this child's birth the family cat had been ill with cat flu treated with chloramphenicol. Feline stool culture at the time of the child's illness failed to isolate Listeria.

Case 2

After an uneventful term pregnancy a Pakistani woman underwent elective caesarean section to be delivered of a...