

for age 1.8–4.5 mmol/l) and triglyceride concentration 1.48 mmol/l (normal for age 0.37–1.2 mmol/l). The nephrotic syndrome was diagnosed and studies to determine the aetiology were planned. The antimicrobial treatment was changed to phenoxymethyl penicillin (62.5 mg twice daily) and cotrimoxazole (2 mg/kg daily of trimethoprim). Two weeks later she developed tense ascites, pallor, and hypothermia. Culture of the peritoneal fluid showed *S pneumoniae* resistant to penicillin. She improved on treatment with intravenous vancomycin.

Discussion

Patients with the nephrotic syndrome are at an increased risk of developing systemic pneumococcal infections due to perturbations of the normal immune responses.^{1–3} Immunisation with pneumococcal polysaccharide vaccine seems to be the most rational method of preventing pneumococcal disease, but it may not be effective in children younger than 2 years,⁴ or protective in patients with nephrotic syndrome who are resistant to steroid treatment.⁵ For these reasons we have used penicillin prophylaxis in nephrotic children younger than 2 years of age which has the potential disadvantage of encouraging the emergence of penicillin resistant pneumococci.

In a recent survey at this hospital 24% of pneumococcal isolates from blood culture in children were due to penicillin resistant strains that were both hospital and community acquired.⁶ We believe that the two patients reported here became colonised in the nasopharynx with penicillin resistant pneumococci while they were in hospital, and that the use of prophylactic penicillin selected for

penicillin resistant strains subsequently resulted in invasive disease. Our experience suggests that this practice may be hazardous in a hospital or community in which penicillin resistant pneumococci are common. Our current practice is to give pneumococcal vaccination to nephrotic children at 2 years of age. For younger children we no longer give antimicrobial prophylaxis but explain the potential risks of infection to the parents who are advised to seek urgent medical attention for any febrile illness. Further studies are planned to evaluate the role of antimicrobial prophylaxis in preventing pneumococcal infection in nephrotic syndrome.

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Correspondence to Dr L Milner, Department of Paediatrics, Johannesburg Hospital, Parktown, Johannesburg, South Africa.

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Infant feeding and atopy

R E MIDWINTER, A F MORRIS, AND J R T COLLEY

Department of Epidemiology and Community Medicine, University of Bristol

SUMMARY Parents of 457 5 year olds from a previous study of infant feeding and eczema in the first year of life were questioned about subsequent atopy. No association was found with early breast or bottle feeding. Family history was important. Parental recall of first year eczema was often inaccurate.

Between 1979 and 1981 we studied the association

between different types of infant feeding and eczema.¹ Four hundred and seventy five infants born to parents with a family history of atopy were investigated to see if there was an association between feeding in the first weeks of life and the incidence of eczema during the first year. Exclusive breast feeding in the first four weeks postponed the onset of eczema; significantly fewer infants had eczema at three and six months, but by one year the incidence was similar in breast and bottle fed infants. To investigate a possible association with

asthma these infants were followed up on their fifth birthdays.

Subjects and methods

Two months before each child's fifth birthday the parents of the 475 children were contacted by letter. Questionnaires were then sent two weeks before the fifth birthday. Questions were asked about the presence of skin diseases (including eczema since birth) and respiratory problems (including wheeze and asthma) diagnosed by a doctor.

Parents who did not reply were sent a second questionnaire. If this failed they were either visited or telephoned. When parents had moved and their new address was unknown, the help of health visitors, family practitioner committees, and the school health service was enlisted.

The significance of differences between proportions was analysed using the χ^2 test.

Results

Replies were received from 457 (96%) of the 475 parents.

ASTHMA

Ninety one children (20%) were reported to have had a wheezing attack between 1 and 5 years of age, and 45 (10%) were diagnosed by a doctor as having asthma. Of these 45, 28 (62%) were boys and 17 (38%) were girls. Seventeen had been seen in hospital because of their asthma and eight had been treated as inpatients. At the time of the survey 26 were still said to have asthma. Eczema was diagnosed at some time in 23 (50%) of these 45 children, and 10 still had eczema at the age of 5.

Table 1 shows the association between infant feeding and risk of developing asthma: there were no significant differences.

Table 1 Association between feeding during the first four weeks and the incidence of asthma in the first five years

Type of fed	No*	Patients with Asthma No (%)
Breast milk only	217	19 (8.8)
Breast milk/or soya milk, or both	86	11 (12.8)
Cows' milk only	150	15 (10.0)
Total	453	45 (9.9)

*Excludes one breast fed and three fed cows' milk in whom the diagnosis of asthma was unknown

In the earlier study the family history of atopy was defined as either 'mild' (one parent with either eczema or asthma) or 'severe' (either both parents affected, or one parent with both eczema and asthma). Of the 45 children who had been diagnosed as having asthma, 18 (40%) had a family history of 'severe' atopy compared with 81 (20%) of the 408 children who were not diagnosed as having asthma ($p < 0.01$).

ECZEMA

Between their first and fifth birthdays, 39 children who had not had eczema in the first year of life were reported to have had eczema diagnosed by a doctor. In the previous study 181 children were identified who had eczema in the first year of life; of these, 174 appeared in the five year follow up. Thus 213 children either had had eczema diagnosed by the survey team in the first year of life or were reported to have developed eczema subsequently; 113 (53%) were male and 100 (47%) female. Forty children (9% of 457) still had eczema at the age of 5.

The association between early feeding and eczema during the first 5 years is shown in Table 2: there were no significant differences. Of the 213 children reported to have had eczema, 59 (28%) had a family history of 'severe' atopy, compared with 40 (16%) of the 244 children not reported to have had eczema. The association with family history was significant ($p < 0.01$).

This follow up was used to test the accuracy of parental recall of their child's eczema. Parents were asked when the child was 5 years old whether eczema had been diagnosed in the first year. Four hundred and fifty five replied: 360 said 'no', and of these, 109 (30%) had documented evidence of eczema under the age of 1 year. Ninety five said 'yes'; of these, 15 (16%) had no documented evidence of eczema.

Table 2 Association between feeding during the first four weeks and the incidence of eczema in the first five years

Type of feed	No*	Patients with eczema No (%)
Breast milk only	217	103 (47.5)
Breast milk/or soya milk, or both	86	38 (44.2)
Cows' milk only	152	72 (47.4)
Total	455	213 (46.8)

*Excludes one breast fed and one fed cows' milk in whom the diagnosis of eczema was unknown

Discussion

The recording of infant feeding patterns in the previous study, which included a dietary diary, was considered to be reliable.¹ Eczema in the first year was also carefully defined and clinical assessment made by one doctor. In the follow up there was no reason to think that biases had arisen due, for example, to differential recall of asthma or eczema between 1 and 5 years of age by parents of children who received breast or bottle feeds in the first 4 weeks. The high response rate made it unlikely that biases could be the result of differential drop out rates.

No evidence was found for an association between type of feeding and development of asthma in the first 5 years. There is, however, a significant association between family history of atopy and risk of the child developing asthma by the age of 5.

Studies of the aetiology of eczema tend to lack

precision in the definition of eczema and accurate recording of the infant's diet. It is worrying that in this motivated and cooperative group of parents with a low drop out rate, recall at five years of eczema during the first year was relatively poor, even though the eczema was diagnosed and discussed with them by a doctor whom they saw regularly. This must have implications for studies relying exclusively on parental recall for evidence of eczema in infancy.

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Correspondence to Dr R E Midwinter, Department of Epidemiology and Community Medicine, University of Bristol, Canynge Hall, Whiteladies Road, Bristol BS8 2PR.

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Type V hyperlipoproteinaemia in neonates

G N THOMPSON, A J KNIGHT, I HAMILTON CRAIG, AND J L BRESSON

Department of Chemical Pathology, Adelaide Children's Hospital, North Adelaide, South Australia, and Paediatric Department, Necker Hospital for Sick Children, Paris, France

SUMMARY A boy investigated for neonatal jaundice was noted to have lipaemic serum and was subsequently shown to have type V hyperlipoproteinaemia. Dietary treatment was maintained for five years and he followed a typical clinical course. Circumstantial evidence suggested an autosomal recessive inheritance pattern.

Type V hyperlipoproteinaemia is rare in childhood and has not to our knowledge been described in a neonate. It is often difficult to distinguish between type I and type V hyperlipoproteinaemia, and differentiation usually relies on measuring plasma post-heparin lipoprotein lipase (PHLPL) activity which is deficient in type I hyperlipoproteinaemia and usually normal in type V.¹ Until 1974 there was no accurate method of measuring plasma post-heparin lipoprotein lipase. Since then three children have been reported with definite type V hyperlipoproteinaemia but normal PHLPL activity,²⁻³ and there have been several descriptions of children with features suggesting type V hyperlipoproteinaemia⁴⁻⁶ The youngest reported was 2½ years old.⁴

Case report

A 5 year old boy who had been investigated for neonatal jaundice was found to have lactescent serum when 6 days old. Xanthelasma and eruptive xanthomata (buttocks) subsequently developed. The liver and spleen were not enlarged. Breast feeding was stopped and a feed containing a medium chain triglyceride formula (Pregestimil) was begun. The first plasma lipid profile was done when he was 2 months old, when a low fat diet started (table 1). The subsequent clinical course was uncomplicated and at 2 years of age, he began a normal diet. A brief recurrence of eruptive xanthomata, however, necessitated a return to restricted long chain triglyceride feeding.

The boy migrated to Australia with his family. When he was 2 years and 7 months old he developed acute pancreatitis. The parents, who controlled his diet carefully, reported no dietary indiscretion before the episode. Fasting serum lipid concentrations subsequently remained under control (table 1). Triglyceride concentrations, although never within the normal range, did not exceed 6.4 mmol/l. High density lipoprotein cholesterol (mean 0.41