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Atopic eczema and preterm birth

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SUMMARY In a group of 443 children with atopic eczema there was a significant lack of subjects born before 37 weeks' gestation. It is possible that preterm birth reduces the chances of the subsequent development of severe atopic disease.

It has been suggested that antigen exposure in the first weeks of life may sensitise the immunologically immature infant, triggering the development of atopic disease.¹ If this were true one might expect an increased incidence of atopic disease in subjects born preterm, and this has been examined in a group of children with atopic eczema.

Subjects and methods

Between January 1982 and August 1987, 445 children with atopic eczema were referred to the department of child health. Eighty were referred by paediatricians, 32 by dermatologists, 312 by general practitioners and 21 by other doctors. The gestational age at birth was recorded as part of a full history taken from the parents at the first visit. Three hundred and ninety two patients (88%) were from the North West Region; 30 (7%) were from the local health district of North Manchester. The rest came from other parts of England and Wales. The median year of birth of the patients was 1981/2. Information about the gestational age of children still alive who were born in 1981 and 1982, to women currently living in the North West Region, was obtained from the North West Regional Health Authority. In these two years there were 87 606 children born who are still alive, and information about gestational age was available in 81 038 (93%).

Results

Information about gestation was unavailable in two patients who were adopted. There were three twins. The gestational age was reported to be 37 weeks or over in 428 (97%) and less than 37 weeks in 15 (3%). The gestational ages of the latter group were 36 weeks in seven children, 35 weeks in four, 34 weeks in three, and 29 weeks in one. The mean (SD) age of onset of the eczema was 6.8 (11.2) months. The social class distribution of the parents of the patients was I and II, 169 (38%); III, 170 (38%); and IV and V, 106 (24%). Fourteen patients were illegitimate, and the fathers of 38 (9%) were unemployed. Of the 81 038 children whose mothers were currently living in the region and where gestational age was known, 4803 (6%) were less than 37 weeks' gestation and 76 235 (94%) were of 37 weeks' gestation or more. The distribution of gestation of the patients was significantly different from that of these 'control' children, $\chi^2=4.66$, $p=0.03$.

Discussion

In a group of 443 children with atopic eczema, there was an unexpected and significant lack of subjects born preterm. As in any hospital based study the patients were highly selected, mainly by severity and poor response to topical treatment, but also by social class. A further drawback is that information about gestation was obtained from parents rather than from maternity records. Nevertheless, although parents may have overestimated or underestimated gestational age these errors should cancel each other out, and there is no reason to suspect that parents of atopic children are especially prone to overestimate

gestational age. There is a modest excess of low birthweight and preterm infants in the lower social classes,² and a small excess of patients in this series from the higher social classes, but this social class effect is insufficient to account for the findings of this study.

The findings of this study are not consistent with the hypothesis that atopic disease is simply the result of sensitisation in genetically predisposed infants who are immunologically immature. It appears that preterm infants are, if anything, less likely to develop severe atopic eczema than those born at or after term. It is known that preterm infants, if compared with term infants, have a decreased production of specific antibodies to cows' milk proteins during the first six months of life.^{3,4} Furthermore, the immunological effects of antigens in the neonatal period may be related not only to the state of maturity of the lymphoid system, but also to that of the neonatal digestive system. It has been shown that the absorption of antigenically intact protein in infants less than 33 weeks' gestation may be up to 100 fold greater than at term,⁵ and it is possible that the absorption of large quantities of antigenic material leads to tolerance rather than sensitisation.⁶

There are other possible explanations for these observations, such as protection from preterm delivery in atopic subjects, a lack of atopic disease among twins, or some difference in the method of

feeding preterm infants. These possibilities need to be examined in a prospective study to discover the incidence of atopic disease in preterm infants. Protection from severe atopic eczema in preterm infants, if confirmed, could provide important clues about the pathogenesis of atopic disease.

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Mycotic intracranial abscesses during induction treatment for acute lymphoblastic leukaemia

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SUMMARY A boy with newly diagnosed acute lymphoblastic leukaemia developed mycotic cerebral abscesses despite treatment with amphotericin. He survived this episode on combination antifungal treatment.

Case report

A 13 year old boy presented with a week's history of bruising and a white cell count of $19.4 \times 10^9/l$ of which 39% were blasts. He was febrile at presen-

tation, with no obvious site of infection, and was started on intravenous cefuroxime and tobramycin with oral nystatin (100 000 units four times a day). An indwelling Hickman line was inserted and UKALL X induction, which included prednisolone 40 mg/m^2 for 28 days, was commenced.

On the fifth day of treatment he developed glycosuria, with a blood glucose of 25 mmol/l , and required insulin. Antibiotics were discontinued from day five, as bacterial cultures were negative, but were restarted on day 11 as he again became febrile and was neutropenic. The only positive culture was *Aspergillus* species on a throat swab; chest radio-