Cystinosis and vitamin D

Sir,

Katzir et al reported a case of nephrogenic diabetes insipidus, cystinosis, and an abnormality of vitamin D metabolism. The idea that nephrogenic diabetes insipidus might herald cystinosis is interesting. Their analysis of the serum vitamin D metabolite concentrations on the patient, however, should not be regarded as a feature of the described association but rather part of the natural course of cystinosis. In 1983 Steinherz et al reported on the circulating vitamin D metabolites in nephropathic cystinosis. In this study, 10 cystinotic patients with various degrees of functional renal impairment were screened for their vitamin D metabolites. The mean (SD) concentrations of 24,25 dihydroxyvitamin D3 (24,25(OH)2D3) were reduced in those patients treated with low dose of ergocalciferol (<25 μg) but within the normal range in the patients who received above 625 μg vitamin D2: 0.75 (0.5) and 6.5 (2.8) nmol/l respectively, while normal concentrations were 4-3 (1-3). These reduced 24,25(OH)2D3 concentrations have previously been found in children with uraemia. (Among these patients there were three cases of cystinosis.)

Serum 1,25 dihydroxy vitamin D3 (1,25(OH)2D3) concentration depended upon the therapeutic agent used in patients. On either low or high vitamin D supplementation, 1,25(OH)2D3 was below the normal concentrations (25(20) and 9(20) compared with 108(30)pmol/l). Treatment with calcitriol and dihydrocalcitriol significantly increased 1,25(OH)2D3 above normal concentrations: 190(40) and 900(513) compared with 108(30)pmol/l.

The conclusion of our report2 on cystinotic patients was in accord with that of Chesney et al3 that circulating values of 24,25(OH)2D3 were reduced in relation to the renal parenchymal damage. The low 1,25(OH)2D3 concentrations could reflect (as also stated by Katzir et al) a renal phosphate leak with impairment of synthesis of 1,25(OH)2D3 besides its correlation with renal insufficiency associated with intrarenal cystine accumulation.

References

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Atopic eczema and preterm birth

Sir,

We noted with great interest the report stating that preterm infants are at decreased risk of suffering from atopic eczema, and attempted to confirm this finding using data from a well known data source, the Collaborative Perinatal Project. This project was a prospective study of pregnancy and child development that from 1959 to 1966 enrolled approximately 55,000 pregnancies at 12 centres in the United States. The recruitment and follow up procedures have been described.2 When the subjects were 1 year old, study physicians completed a diagnostic summary form that included a code for eczema. Infant gestational age was determined from the date of the mother’s last menstrual period. Children whose birth weight was grossly incompatible with their gestational age were eliminated.

There were 44,793 children who survived the first year and for whom the presence or absence of eczema was known. Four thousand and eighty nine (9.1%) of these children were born after less than 37 completed weeks' gestation. Eczema was slightly less common among preterm infants: 1.5% of preterms had eczema compared with
1·8% of term infants. The odds ratio for a preterm infant to have eczema was 0·86, and the 95% confidence interval was (0·66 to 1·11). This indicates that preterm infants are at slightly decreased risk of eczema, but that this difference was minimal and not significant. The 95% confidence interval in this analysis excludes 0·56, which is the estimated odds ratio reported by David and Ewing. Adjustment for maternal race and education (a proxy for social class) did not change the odds ratio substantially.

In summary, this analysis of data from the Collaborative Perinatal Project failed to confirm the hypothesis posed by David and Ewing of a decreased risk of atopic eczema among preterm infants followed to 1 year of age.

References

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Silastic catheters for antibiotics in cystic fibrosis

Sir,

We read with interest the paper by Dr Williams and her colleagues, comparing percutaneous silastic catheters with conventional intravenous cannulas for giving antibiotics to patients with cystic fibrosis. We agree and have reported that these catheters are favoured by the patients, last longer, and have fewer complications.

Since 1985 we have been using a modified version of the catheter described by Dr Williams, which does not require threading onto a 25 gauge butterfly needle (Vygon epicutaneous catheter designed for neonatal use). The manufacturers changed the design of the catheter in 1985 after an incident when a line was accidentally cut by the butterfly needle and apparently disappeared into the child (without ill effect). The new catheter is also marked every 5 cm and is radio-opaque so that it is easy to ensure that the whole catheter has been removed at the end of treatment.

Local anaesthetic cream is applied to the site at least one hour before the procedure. The catheter is primed with heparinised saline and is flushed one hour after insertion with 2 ml heparinised saline (100 U/ml). We have not found it necessary to administer a continuous infusion of heparinised saline, but merely flush the catheter every four hours. The catheter is covered with a transparent plastic film and a bandage, allowing the patient freedom to play or even go swimming.

The cost of each catheter (£1·84) is negligible in comparison with the total cost of a two week course of antibiotics. Many patients have been able to complete their treatment at home, without the inconvenience of returning to the hospital for a conventional cannula to be resited. The technique of inserting the catheter has been taught to the house physicians caring for adult cystic fibrosis patients as well as those caring for paediatric patients at this hospital.

References

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