PostScript

LETTERS

Heterogeneity of diabetes in south Asian patients in Bradford, West Yorkshire

The incidence of type 1 diabetes in children in India and Pakistan is remarkably low.¹ However, south Asian children resident in the UK have a rising incidence of type 1 diabetes,² approaching that of the indigenous population.³ There is particular concern about the emergence of type 2 diabetes in children, and the consequent morbidity and mortality.⁴⁻⁵ South Asian children are particularly at risk of type 2 diabetes.

A total of 160 children aged 0-16 attend the diabetic clinic in Bradford, of which 58 (38%) are of south Asian origin. In comparison, south Asian children comprise 27% and 32% of the primary and secondary school populations, respectively. Most have type 1 diabetes, but some have more unusual conditions not seen in the indigenous children.

Case 1

A girl aged 11 years presented with diabetes without ketonuria, a body mass index of 25, and negative autoantibodies. Type 2 diabetes was diagnosed; she was treated with an oral agent, but four years later transferred to insulin because of poor glycaemic control.

Cases 2-4: DIDMOAD syndrome

One boy had a ventricular septal defect repair in infancy; he developed diabetes at the age of 3 and was started on insulin therapy. At the age of 10 he developed optic atrophy, then diabetes insipidus, chronic renal impairment, and a neuropathic bladder.

Another boy—one of three brothers with insulin treated diabetes—presented at the age of 3. He later developed optic atrophy. His two brothers have no other features of DIDMOAD syndrome (diabetes insipidus, diabetes mellitus, optic atrophy, deafness).

The third case is a girl who developed insulin dependent diabetes at the age of 3 years; she developed bilateral lens opacities two years later, treated by cataract extractions and bilateral lens implantation. Recently her visual acuity has deteriorated due to bilateral optic atrophy. She has no other features of DIDMOAD at present.

Case 5

An 11 year old boy comes from a family with a probable mitochondrial depletion syndrome. Aged 7 months, he developed diabetes which was treated with insulin. He is the fourth child from a highly consanguineous pedigree to develop diabetes in infancy. These children have a tendency to develop acute liver impairment during intercurrent illnesses. A sibling died from acute liver failure aged 22 months. Their twin cousins died aged 3½ from acute liver failure. The surviving child has intermittent deranged liver function but has never had liver failure and remains insulin dependent with poor growth and some renal impairment.

Case 6

A male infant presented at birth with insulin requiring diabetes, remaining insulin dependent thereafter. He was growth retarded at birth but had no dysmorphic features and a syndromic diagnosis was excluded. Presently aged 18 months, his growth and development are normal.

Case 7

This child was born at 34 weeks to a woman with gestational diabetes. He had hypertrophic cardiomyopathy post-delivery but this subsequently resolved. He now has visual impairment due to a retinal cone dystrophy, which is mirrored in his father and two uncles. Latterly, he developed significant obesity and acanthosis nigricans, and was diagnosed with Alström syndrome. He became hyperglycaemic without autoantibodies and is being treated successfully with metformin.

Discussion

Our clinic population includes a number of children who have diabetes in association with other unusual conditions, for whom their diabetes was not easily classified and may have been a feature of their intrinsic syndrome. The high prevalence of syndromic diabetes in this population may emanate from the high consanguinity rate in the Bradford Asian population. We suggest it is vital for children of South Asian origin presenting with new onset diabetes to be carefully assessed, with the aid of a good family history, due to the high incidence of unusual forms of diabetes in this group of children.

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Advice for new mothers

My first-born arrived just after an SHO paediatrics job in Oxford and passing my DCH and DCCH exams. I was confident in handling small patients and in fielding parents' questions but I was not confident with my own new baby. My scientific clinical self started having arguments with my unconfident maternal self, and even the slightest suspicion of criticism of my maternal skills sent me into neurotic introspection.

I went to a community clinic for my baby's six week check and the CMO asked, "Any worries?"

"No, none really", I replied. "I know in my head that he's fine. He sleeps well and he's growing right along the 50 percentile. My only problem is in confidence. Whenever anyone suggests I should be doing things differently I plunge into a turmoil of guilt and worry."

The doctor examined my baby and on palpating his abdomen said, "He's full of gas. The position you are feeding him in can't be quite right. He must be sucking in air as he feeds."

As doctors I am sure we often say too much and give too much advice, but it wasn't until I left the room that I thought what an unhelpful comment this colleague had made to me. It certainly undermined my confidence—again.

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NHS Direct

The establishment of training sites for consultation skills is a worthwhile development in the service.¹ What we also now need is a fundamental change in our attitude to the educational needs of nursing staff in our daily encounters on ward rounds and in the outpatient clinics. To replace the stereotype of the nurse being merely a passive recipient of the doctor's clinical wisdom, consultants (and other medical staff) should utilise each and every clinical episode to engage the attending nurse in a clinical problem solving exercise aimed at highlighting the diagnostic issues from the nurse's perspective. To take the process one step further, nurses should be encouraged to attend the "problem solving exercise of the week", which has become a tradition in postgraduate centres up and down the country. Each and every nurse is