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. However, south Asian children resident in the UK have a rising incidence of type 1 diabetes,1 approaching that of the indigenous population. There is particular concern about the emergence of type 2 diabetes in children, and the consequent morbidity and mortality.2,3 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 ventilricular 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 extraction 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 3 years. A sibling died from acute liver impairment during 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 3 years. A sibling died from acute liver impairment during infancy; he developed diabetes at the age of 10 he developed optic atrophy, then diabetes insipidus, chronic renal impairment, and a neuropathic bladder.

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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NHS Direct
The establishment of training sites for consultation skills is a worthwhile development in the service. 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 week”, which has become a tradition in postgraduate centres up and down the country. Each and every nurse is
A potential future employee of NHS Direct. By optimising their educational opportunities we are making a future investment in the service.

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References

Cause and effect?
I read Feltblower and colleagues' article with great interest, having for some time wondered whether there might be an epidemiological association between childhood leukaemia and diabetes. The observations that the authors make regarding regional distribution of disease within a country certainly warrant further investigation.

Coincidentally, Mohn and colleagues have recently shown clinically significant impairment of pancreatic beta cell function in children who have been treated for acute lymphoblastic leukaemia (ALL). Although the numbers will certainly be small, is it possible that some of the observed correlation betwixt these two conditions could stem from "iatrogenic" diabetes?

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References

BOOK REVIEWS
Nutrition in pediatrics: basic science and clinical applications, 3rd edition

In large Victorian households, feeding the children in the nursery was traditionally left to the youngest and most inexperienced householdmaid. Recognition of the importance of nutrition in children in hospital and in the community has been equally slow. Medical undergraduates continue to receive little teaching on nutrition, even though over and under-nutrition continue to be seemingly intractable problems; a substantial proportion, maybe a third, of patients in children’s hospitals exhibit evidence of malnourishment. Moreover, we are now recognising that fetal and infant nutrition are major determinants of long term health.

One reason for the glacial speed in the development of clinical nutrition has been a view that clinicians already know enough about nutrition. That view is fostered in part by nutritional knowledge being recorded in the literature in disparate sites and in different ways. Much of it is in textbooks of biochemistry and metabolism; only rarely is it easily accessible. There is no obvious course book for the Royal College of Paediatrics and Children Health Diploma Course in Nutrition (www.rcpch.ac.uk).

Having the relevant basic science pulled together in one volume is immensely helpful to specialty developments; failure to do so is enormously damaging. One of the most interesting and exciting innovations in paediatric practice in the past 50 years in the United Kingdom was the concept of community paediatrics. It has foundered, partly because the basic science underpinning the discipline was never collected, written down, and published. Clinical texts are little more than recipe books without the underpinning basic science.

Nutrition in pediatrics addresses many of these problems, and the editors are to be congratulated on a tour de force. My assumption about its scope being limited to hospital based, clinical nutrition has not been confirmed. It is useful to the paediatrician working in the community or in developing countries as well as the tertiary, hospital based specialist. The psychosocial aspects of feeding are well covered and I was pleased to see sections devoted specifically to adolescents. There are no obvious gaps.

The RCPCH is seeking to redress the lack of teaching in nutrition in undergraduate medical courses with its course in paediatric nutrition. This volume makes an ideal course book, and should also be a routine acquisition for all postgraduate and ward libraries. Buy.

I W Booth

Managing children with psychiatric problems, 2nd edition

Emerging evidence, and political pressure have changed Child and Adolescent Mental Health Services a good deal over the past 10 years. It is mystifying enough to be part of these changes. I can only imagine what it might be like to be looking on. This second edition brings a useful book up to date. It is a sort of ambassador for Child and Adolescent Psychiatry and should go a long way towards explaining the discipline to general practitioners, and should be on the strength of its illustrations, one of these important areas.

Having got over this minor irritation, the more I looked into this book, the more I liked it. It is easy to read, is well referenced, and punches above its weight.

A West

Pediatric urology

Paediatric urology has come of age over the past few years, with it now being formally recognised as a distinct subspeciality of both urology and paediatric surgery within the European Union. In parallel with this development there has been a rapid expansion in the number of paediatric urologists practising in the United Kingdom. With increasing specialisation there is a risk of isolation, and developments in the field of urology may go unrecognised in the broader areas of paediatrics, paediatric surgery, and urology. Therefore there is a need for publications to highlight recent developments. The non-specialist rarely reads major reference textbooks of which there were two published in
Tuberous sclerosis complex: from basic science to clinical phenotypes


Tuberous sclerosis has always had the capability to confuse clinicians. Friedrich von Recklinghausen confused it with neurofibromatosis, when on 25 March 1862 he presented a case to the Obstetrical Society of Berlin. The case he described was of a young infant who had died soon after childbirth and who was discovered on postmortem examination to have multiple cardiac myomata in the ventricular walls and a great number of “scleroses” in the brain. It was a French physician, Desire Magloire Bourneville, a pupil of Charcot, who has won the plaudits to demystifying this complex disease.

Curatolo and Seri’s chapter on seizures is also strong. Particularly interesting is the discussion of how epileptogenesis in TSC may be related to an impairment of GABA mediated neuro-inhibition. It is startling how well vigabatrin, a specific inhibitor of gamma amino butyric acid aminotransferase, is supposed to work in the treatment of infantile spasms in tuberous sclerosis. In Hancock and Osborne’s Cochrane Review of the treatment of infantile spasms, they found that there was a 95% response rate in those individuals with TSC who were treated with vigabatrin. The acid test of this therapy, of course, will be to see whether the apparent remarkable success rate in stopping spasms is matched by an improved intellectual outcome in these patients later in life.

The genetics of TSC are fascinating, and Kwiatkowski and colleagues carefully and clearly take the reader through the complexities of the subject. The disease is caused by alterations in two genes, TSC1 and TSC2, on chromosomes 9 and 16 respectively. The tumour suppressor genes and mutations in either can be responsible for all the complications seen in the disease. The gene products of the two genes, hamartin and tuberin, appear to act together in the regulation of cell growth by inhibiting a substance known as mTOR (target of rapamycin). In those individuals with TSC who were treated with rapamycin, a clinically available compound, also selectively inhibits mTOR and therefore there is now the possibility that rapamycin may provide some benefit in reducing the clinical manifestations of TSC. Fittingly, Professor Curatolo’s book finishes with the intriguing suggestion that at last we may be on the threshold of being able to do something to ameliorate this complex and potentially devastating disease.

P S J Malone

CORRECTION

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Toni S, Reali M F, Fasullo A, et al. The use of insulin pump improves the metabolic control in children and adolescents with type 1 diabetes Arch Dis Child 2004;89:796–7. The initial of Dr Festini was incorrectly published as an F. The correct initial is P.
Heterogeneity of diabetes in south Asian patients in Bradford, West Yorkshire

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