Rising incidence of type 2 diabetes in children in the United Kingdom

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Objective: To estimate the incidence of type 2 diabetes in children under 17 years of age and its relationship to increasing childhood obesity in the United Kingdom and the Republic of Ireland (ROI).
Design: Active monthly reporting of cases by consultant paediatricians through the framework of the British Paediatric Surveillance Unit, with additional reports from specialist diabetes nurses.
Subjects: All children under the age of 17 years diagnosed by their clinician as having non-type 1 diabetes from 1 October 2004 to 31 October 2005 (inclusive).
Results: 168 confirmed cases of non-type 1 diabetes were reported giving a national incidence (excluding the ROI) of 1.3/100,000/year. Of these 40% were diagnosed with type 2 diabetes giving a minimum incidence of 0.53/100,000/year. Children of ethnic minorities were greatly over-represented with those of Black and South Asian origin (England data only) having an incidence of 3.9 and 1.25/100,000/year respectively compared to 0.35 /100,000/year in those defined as white. Of those diagnosed with type 2 diabetes, 95% were overweight and 83% obese according to IOTF guidelines. 84% had a family history of type 2 diabetes.
Conclusions: Compared with type 1 diabetes, type 2 diabetes in the UK is still uncommon in children. However, compared to previous prevalence data it appears to be increasing in frequency. Incidence amongst ethnic minorities is far higher than whites as previously described in the USA. Increased adiposity is strongly associated with a diagnosis of type 2 diabetes as is a close family history of the condition.

Introduction

The growing prevalence of childhood obesity in the United Kingdom[1] [2] undoubtedly has implications for our children's health. Predictions from the USA imply that obesity driven type 2 diabetes might become the most common form of newly diagnosed diabetes in adolescent youth within 10 years[3]. There is now good evidence suggesting a global spread of type 2 diabetes in childhood although incidence data are uncommon[4]. Various centres in the USA have recorded dramatic increases in the number of children diagnosed with type 2 diabetes. A 10-fold increase was recorded from a centre in New York from 1990-2000 with 50% of all new cases of diabetes having type 2[5] with similar increases being reported elsewhere [6] . In Japan, researchers have documented a rise in annual incidence from 1.73/100,000 to 2.76/100,000 over twenty years[7] whilst evidence is emerging of an increase in urban South-Asian[8] children. Data from Europe are scarce[9]: a population based study in Austria established an incidence of 0.25/100.000 children[10], whilst a report from France indicated relatively low but increasing numbers of children presenting with type 2 diabetes[11]. In the UK, Ehtisham et al estimated a crude prevalence of type 2 diabetes under 16 years of 0.21/100,000[12] whilst a recent report reviewing first hospital admissions with a diagnosis of type 2 diabetes in patients under 18 years indicated a significant rise between 1996-7 to 2003-4[13].

The emergence of type 2 diabetes in adolescence has important implications for both the health of the individual and health service resources. Treatment compliance[14] and psychological health[15] is often poor in childhood type 2 diabetes. Various studies imply an accelerated risk of nephropathy[16] [17] and retinopathy[18] compared to young people with type 1 diabetes, whilst recent data indicate early signs of cardiovascular disease in youth with type 2 diabetes[19]. The only currently available longitudinal data makes for worrying reading: of 79 children re-contacted up to 15 years after the diagnosis of type 2 diabetes, 9% had died and 6% were on dialysis[20].

The growing number of anecdotal reports of increasing type 2 diabetes in British children and a need to establish clinical guidelines and frameworks for their treatment, prompted us to initiate a prospective, population based surveillance study to establish baseline incidence rates for non-type 1 diabetes and more specifically type 2 diabetes in childhood.

Methods

A prospective monthly surveillance of 2665 consultant paediatricians in the UK and the Republic of Ireland (ROI) through the British Paediatric Surveillance Unit (BPSU) of the Royal College of Paediatrics and Child Health was undertaken to identify cases of non-type 1 diabetes in 0-16 year olds. The study ran from October 2004 until October 2005 (inclusive). The study received ethical approval from the South West Multi Research Ethics Committee (04/MREC06/39) and was also given approval not to seek patient or parent consent by the Patient Information Advisory Group (PIAG/BPSU2-10(b)/2005).

A parallel reporting system was set up with diabetes specialist nurses to identify adolescents directly referred to adult services. From a list of diabetes specialist nurses provided by Diabetes UK, an individual was identified and

invited to participate from each acute hospital. The 157 nurses who agreed, were sent reporting cards every two months throughout the study.

Clinicians (paediatricians and nurses) were asked to report any new case of non-type 1 diabetes either suspected or confirmed seen in the previous month. Cases of non-type 1 diabetes were to be reported as:

- Type 2 diabetes (with evidence of insulin resistance: acanthosis nigricans, raised fasting insulin or C-peptide)
- Maturity Onset Diabetes of the Young (MODY; characterised by known MODY gene mutation)
- Diabetes as part of a recognised syndrome
- Diabetes as part of a suspected or unrecognised syndrome
- Diabetes secondary to another condition (eg cystic fibrosis)

With the rising overall prevalence of childhood obesity, children presenting with types 1 or 2 diabetes may be obese, so obesity itself is not a useful discriminatory marker. Additionally, monogenic forms of diabetes such as Maturity Onset Diabetes of the Young (MODY) are recognised to have clinical overlap with type 2 diabetes. Consequently we planned a survey of non-type 1 diabetes, using a broad definition, to maximise the capture of type 2 cases. We excluded obesity from our notification criteria in order to analyse the anthropometric characteristics of the survey children and because obesity itself is a poor discriminator as children with type 1 diabetes relatively frequently have excess adiposity as general prevalence levels of obesity increase[21]. Because of the difficulties of distinguishing type 1 from non-type 1, reports of cases previously diagnosed as being diabetic but newly recognised as atypical for type 1 were sought (i.e. children initially diagnosed with type 1 diabetes, but not behaving as if they had classical "insulin dependent" diabetes: eg diabetic but low or no insulin requirement; excellent control without regular monitoring, few hyperglycaemic episodes, absence of ketonuria and out of the honeymoon period).

Clinicians reporting a case of non-type 1 diabetes were sent a questionnaire to collect the physician diagnosis, basic demographic details, presenting symptoms, confirmatory diagnostic tests, date of diagnosis, height and weight at diagnosis and family history of non-type 1. Duplicate case reports were identified using hospital number and month and year of birth. When duplicates were identified, data from both questionnaires were collated and recorded. A letter and subsequent phone call reminded clinicians not returning the questionnaire within one month, with a final reminder letter at the end of the study period. To maximise ascertainment, at the end of the reporting period all clinicians reporting cases were sent a list of cases they had notified to confirm that all eligible individuals had been identified.

Completed clinical questionnaires were scrutinised to confirm that the cases met the study criteria (case of non-type 1 diabetes diagnosed before the 17th birthday between 1st October 2004 and 31st October 2005) and were diabetic according to the ADA criteria[22]. The physician diagnoses were reviewed by the study clinicians (JPHS, TGB) in the light of the information provided on the questionnaire and were re-classified as necessary according to the following case definitions:

- **Definite type 2.** A case where the clinical questionnaire confirmed the

presence of raised insulin (>132 pmols/litre or equivalent) or C peptide levels (>600 pmols/litre) and/or the absence of autoimmune antibodies found in type 1 diabetes.

- **MODY.** A case reported as MODY with a confirmed gene mutation.
- **Secondary.** A case where the diabetes was secondary to another condition.
- **Syndromic.** Cases in which there were extra-pancreatic clinical features such as retinal dystrophy or sensorineural deafness that suggest a syndrome such as DIDMOAD.
- **Unclassified.** Cases where insufficient clinical information was supplied to make a diagnosis on the above criteria.
- **Type 1.** Cases in which there were positive Glutamate decarboxylase (GAD) antibodies or high titres of Islet Cell Antibodies (ICA).

Paediatricians reporting a confirmed case of non-type 1 diabetes were sent a follow-up questionnaire one year after the initial case report, in order to clarify diabetes status and to clarify insulin/C peptide and autoimmunity status.

Incidence rates were calculated using population denominators available from the Office of National Statistics.

Results

363 cases were reported over the 13 months; 250 (69%) through the BPSU and 113 (31%) by diabetes specialist nurses. The mean monthly return of BPSU cards over the study period was 93%, and 91% for the nurse cards.

Completed clinical questionnaires were received for 94% of reported cases (341/363). We excluded 173 (48%) reports because they were duplicates or they included children: aged older than 17 years at diagnosis; diagnosed outside the study period; or whose diagnosis was not non-type 1 diabetes.

Scrutiny of the questionnaires confirmed that 168 met the study criteria and were cases of non-type 1 diabetes; 126 (75%) were reported by paediatricians and 42 (25%) by nurses. Of the 42 cases reported by nurses 39 were being cared for by a paediatrician, 20 of which were also reported by the paediatrician concerned, whereas 3 were only being cared for by an adult endocrinologist/diabetologist. The 19 cases reported solely by a nurse but under the care of a paediatrician may reflect a degree of under-reporting by paediatricians. However, in most cases it probably reflects a degree of negative independence of paediatrician and nurse reporting by which one professional, aware of the other's report does not duplicate it. This process was well documented in a previous diabetes survey through the BPSU[23].

All non-type 1 cases

The 168 cases of physician-diagnosed non-type 1 diabetes included 67 cases of type 2, 17 MODY, 37 secondary to another condition and 18 cases of diabetes associated with a recognised or unrecognised syndrome (Figure 1). There were 34 cases of non-type 1 diabetes that could not be classified as they did not satisfy our criteria for type 2 but did not fit any other category of non-type 1.

Of the 168 non-type 1 cases 93 were girls and 75 boys (1.24: 1, girls: boys) and the mean age at diagnosis was 12.3 years for girls and 12.7 years for boys (1.2 months – 16.8 years).

In 22% cases (37/168) the child was originally diagnosed as having type 1

diabetes but the clinical course had prompted a diagnostic revision. 14 cases were revised from type 1 to type 2, 8 were revised to MODY, 5 to syndromic diabetes and 1 case was revised to secondary diabetes. 9 cases were revised to an unclassified category of diabetes.

Type 2 diabetes

There were 67 cases of Type 2 diabetes representing 40% (67/168) of all non-type 1 cases reported. 57% (38/67) of cases were girls and the mean age at diagnosis was 13.3 ± 1.7 for girls and 14.1 ± 2.0 for boys (range 8.3 to 16.8 years). 57% of cases were in white children and 43% belonged to an ethnic minority; predominantly South Asians (35%) and Black/Black British (45%).

57% (38/67) cases of type 2 had acanthosis nigricans, most commonly in the neck and axillae. This was present in only 42% of whites compared to 76% of those classified as Black/South Asian or of Mixed race (Continuity corrected χ^2 test, $p = 0.012$).

Out of the 67 type 2 cases, 45 had measures of insulin and/or C-peptide levels ; raised C-peptide levels (>600 pmol/l) were reported in 12 cases, raised insulin levels (>132 pmol/l) in 15 cases and both raised insulin and C-peptide levels in 18 cases.

In 79% of type 2 cases (53/67) antibody testing was carried out. Antibodies tested included glutamic acid decarboxylase-65 (GAD) (35 tests: all negative), the islet cell (ICA) (37 tests: 36 negative, one weakly positive), and Anti Insulin Autoantibodies (IAA) (7 tests: all negative). The one case testing weakly positive for ICA was negative for GAD-65, suggesting continued type 2 classification.

In those cases in whom details were recorded (39/67), 28% had ketosis at diagnosis whilst 7% (5/67) of all the type 2 cases were reported as presenting in ketoacidosis. 7 female patients with type 2 diabetes (18% of girls) had additional biochemical evidence of polycystic ovarian syndrome.

Body mass index "z-scores" or standard deviation scores (BMI SDS) at diagnosis was calculated from weight and height. Overweight and obesity being defined as ≥ 1.30 and ≥ 2.37 respectively for males and ≥ 1.19 and ≥ 2.25 for females[24]. 95% of children were overweight and 83% obese according to the International Obesity Task Force guidelines. The mean BMI SDS, estimated using the 1990 UK growth standards[25] was 3.04 for girls and 2.45 for boys.

In 84% (56/67) of cases there was a first or second degree family history of diabetes, 71% in first-degree relatives.

Secondary diabetes

37 children (21 girls, 16 boys) had diabetes secondary to another condition. The commonest disease association was cystic fibrosis (57%) followed by iatrogenic causes such as steroid induced diabetes after renal or bone marrow transplantation. All children with secondary diabetes were white with two exceptions (one South Asian, one mixed race).

MODY

There were 17 MODY cases, of which 9 (53%) were female. Mean age at presentation was 11.9 ± 3.65 for girls and 12.5 ± 4.4 for boys. The 17 confirmed molecular diagnosis cases included 7 Glucokinase, 6 HNF-1α, one HNF-1β and 2 HNF-4α gene mutations.

Syndromic diabetes There were 13 cases of syndromic diabetes; 69% (9/13) being cases of neonatal diabetes.

Incidence rates for non-type 1 and type 2 diabetes

No cases of type 2 diabetes and only one syndromic case were reported in the study period from the ROI so incidence figures are given for UK alone. The UK under-17-year population mid-2004 was 12,440,700 (Office for National Statistics). The total number of confirmed cases of children with non-type 1 diabetes aged 0-16 years at diagnosis for the UK in 2004-2005 was 167 (excluding the 1 case from ROI), giving a national incidence of non-type 1 diabetes of 1.3 (95% CI 1.2-1.6)/100,000 children per year. For type 2 diabetes the UK incidence was 0.53 (95% CI 0.41-0.68)/100,000 children per year.

The incidence of type 2 diabetes was substantially higher in children from an ethnic minority background; 3.9(95% CI 2.1-6.7)/100,000/year and 1.25 (95% CI 0.6-2.4)/100,000/year for Blacks and South Asians compared with 0.35(95% CI 0.2-0.5)/100,000/year for whites (incidence rates calculated for cases reported in England only as mid-2004 estimates for ethnic groups only available for that country).

Discussion

These data are the first to accurately quantify the UK incidence of type 2 diabetes in childhood. The only previously collected national data were prevalence figures reported in 2004[12]. Given that the incidence figures for our study are around two and a half times higher than those of the prevalence data collected in 2003, this suggests that although ascertainment in the previous collection and our study may have been less than complete, we are witnessing an increase in childhood type 2 diabetes cases in this country. However, the overall incidence of type 2 diabetes in our survey at 0.53/100,000 per year in comparison with the incidence for type 1 diabetes: 15-20/100,000 in children under 15 years[26], indicates that type 2 diabetes is still relatively infrequent in our population. The incidence data for white children at 0.35/100,000 are relatively similar to the only other available data from Europe, that of Austria with an estimate of 0.25/100,000. However, as in the USA some ethnic minorities are greatly over-represented in our figures[3]. The incidence rates for South-Asians and Blacks are more alarming being 3.5 and 11 times more frequent than in whites respectively. In some urban centres in the UK, ethnic minorities make up 25-50% of the resident population and there are indications that race does affect the prevalence of obesity[27], making specific health policies to address the aetiology of type 2 diabetes in youth in these areas a matter of some urgency.

The lack of notified cases from the ROI at first glance might appear surprising. Although there are scant prevalence data for childhood obesity in the Republic, one recent report identified a prevalence of around 25% in a rural setting amongst primary school children[28], a figure comparable to the UK. Under-reporting might be a factor causing this apparent anomaly but equally the racial demographics of the ROI are different from the UK with only 1% of the population being of African or Asian extraction (National Statistics Office. Ireland. 2002 census). Given approximately 900,000 individuals under 17 years of age and a putative incidence similar to UK figures for whites of 0.35/100,000/year the expected number would be just over 3 new cases in the reporting period. This perhaps serves to exemplify the influence of racial heterogeneity on type 2 diabetes national incidence.

The comparison between the UK and USA in terms of risk factors is surprisingly consistent. Overweight and

obesity undoubtedly are major factors in type 2 diabetes aetiology and virtually all our cases were either obese or overweight as previously described in the USA[29]. Another major association in our type 2 cases was a family history of type 2 diabetes. The frequency of a history of type 2 diabetes in first or second degree relatives has ranged between 74 and 100% in the USA[3] and our findings of 84% (71% in first degree relatives) mirror these findings almost exactly. The mean age at presentation again reflects the experience in the USA[29] with a peak around the age of 13-14 years corresponding to late puberty in most adolescents with females presenting about a year earlier than males on average.

Acanthosis nigricans was identified in just over half our cases with type 2 diabetes, a lower figure than that quoted for the USA where as many as 90% of individuals have this cutaneous manifestation of insulin resistance[30]. The feature is said to be commoner and more easily recognised in darker skinned individuals and this was certainly the case in our sample.

The UK is currently struggling to identify ways to reduce the year on year increase in obesity prevalence. Recent figures from a number of sources, indicate that these levels continue to increase in the UK[2]. Strategies for the prevention and treatment of childhood obesity need urgent evaluation and should possibly, given limited healthcare resources, be more specifically focussed on cities with large ethnic minority groups. A further effective strategy to prevent childhood onset type 2 diabetes might be to target obesity management schemes at those obese children with a strong family history of the condition.

Our study has limitations, largely based on the framework within which the BPSU has to function. Although response rates for this study were high at 94%, data collection was reliant on notification by paediatricians, some of whom may not have recognised the possibility of type 2 diabetes in their patients. This probably has led to an under-estimate of the real incidence of type 2 diabetes. In an attempt to alert reporters to the possibility of non-type 1 diabetes occurring in childhood, and especially to consider the possibility of type 2, an information sheet was sent to all participating reporters at the start of the reporting period detailing the clinical features suggestive of non-type 1 diabetes and alerting reporters to those clinical presentations, such as antibody positivity, which although traditionally typical of type 1 may also occur in type 2 cases. To maximise case ascertainment, all clinicians reporting cases were sent a list of cases they had notified at the end of the reporting period and asked to check whether any eligible cases had been overlooked Furthermore, the structure and ethical requirements of such epidemiological studies precludes the direct requesting of samples from all children for antibody or fasting insulin/ 'C' peptide analysis meaning that we had incomplete data on a number of children preventing us from accurately classifying them. Moreover, our classification criteria might also have led to an under-estimation of type 2 diabetes incidence due to our decision to exclude any cases presenting with ketosis at diagnosis if there was no direct evidence of hyperinsulinaemia/absence of autoimmunity. It is now well documented that ketosis at diagnosis is a fairly common occurrence, especially in some racial groups such as black African-Americans [31 32] and indeed 28% of our "definite cases" in whom measures were made, had evidence of ketosis at diagnosis. It is of course also possible that some of our unclassified cases (due to limited information provided) might indeed have had type 2

diabetes. Not withstanding these issues, we now have baseline data on which to build up a future picture of the epidemiology of type 2 diabetes in childhood with a repeat survey planned in five years. The incidence data is liable to be an under-estimate of the true figures given the constraints and consequent limitations of our survey as documented above.

However, we present evidence that type 2 diabetes although rare, is becoming increasingly prevalent in childhood in the UK. Risk factors are similar to those in the USA with increasing adiposity, racial origin and heredity all increasing risk. Children with type 2 diabetes tend to have other associated features such as visceral obesity, dyslipidaemia and hypertension termed the "metabolic syndrome"[33]. In all probability this increases the risk of medium to long-term complications compared to children developing type 1 diabetes.

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Authorship: LH had the original idea for the study. JPHS, TGB and LH formulated the research plan and obtained funds from Diabetes UK. KCW, RL and LH developed the data collection instruments for the study and KCW co-ordinated the data collection. All authors contributed to the clinical and data evaluation meetings held regularly in London. KCW and LH conducted the data cleaning. JPHS and KCW wrote the first draft with significant contribution from all authors in developing the final draft of the paper.

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Table 1: Classification of reported non-type 1 diabetes cases

Type of diabetes	Number of Cases	Percentage of all cases (%)
Type 2	67	40
MODY	17	10
Secondary	37	22
Recognised Syndrome	13	8
Unclassified	34	20