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Management and one year outcome for UK children with type 2 diabetes

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Key words: Type 2 diabetes, obesity, treatment

Objective: To report the one year outcome for newly diagnosed children with type 2 diabetes in the United Kingdom (UK).

Design: Follow up study of a UK national cohort.

Subjects: All children under the age of 17 years diagnosed with type 2 diabetes from 1 October 2004 to 31 October 2005 (inclusive).

Results: Follow up data was available for 73 of the 76 cases. The mean age at follow-up was 14.5 yrs, with mean duration of diabetes 1 year. The revised incidence of type 2 diabetes in the UK in children under 17 years is 0.6/100,000/year. The mean BMI SDS at diagnosis was 2.89 and mean change at one year was -0.11 (range: -1.53 to +1.37). At one year only 58% achieved the ADA/EASD recommended treatment target ($HbA_{1c} \leq 7.0\%$). There was no relation between improvement in BMI and improvement in HbA_{1c} . There was wide variation in choice of therapies and regimes. Hypertension is a common co-morbidity (34%) whilst early nephropathy appears rare (4%). Evidence of polycystic ovarian disease was common in females (26%). 22% of children had not been screened for nephropathy or retinopathy during the first year after diagnosis.

Conclusions: The 3.8% mean reduction in BMI SDS in the first year after diagnosis indicates that many children find it hard to make the necessary lifestyle changes needed to positively impact upon metabolic health .

Physicians are using a wide variety of treatment regimens which are relatively effective in achieving glycaemic targets but systematic screening for complications is incomplete. There is an urgent need to develop an evidence base for effective treatment and management protocols to reduce the risks for long term micro- and macro-vascular complications.

Introduction: The prevalence of childhood obesity is increasing rapidly in many countries and the complication of type 2 diabetes now accounts for up to 45% of newly diagnosed diabetes cases in children in some centres in the USA¹. Some co-morbidities such as nephropathy, are as common in children as in adult onset type 2 diabetes^{2,3}. The development of early onset type 2 diabetes will have lifelong implications both for the individual child and future health care budgets. Outside of ethnic minority groups there are sparse prospective data on outcome in paediatric cohorts with type 2 diabetes.

From October 2004 to October 2005 we conducted a prospective study to estimate the incidence of type 2 diabetes in UK children under 17 years using the active monthly reporting system of the British Paediatric Surveillance Unit⁴. This included contacting the referring physician one year after initial notification to confirm diagnosis and review short term treatment and outcome. The purpose of this report is to describe the effectiveness of current treatment in terms of weight change and glycaemic control one year after diagnosis.

Methods: A prospective monthly surveillance of 2665 consultant paediatricians in the UK and the ROI through the BPSU was undertaken to identify cases of non-type 1 (non-autoimmune) diabetes in 0-16 year olds⁴. In summary, clinicians reporting a case of non-type 1 diabetes were sent a questionnaire shortly after notification 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. A second questionnaire was sent approximately 12 months after the initial case report. This sought the physicians' current diagnosis: a review of insulin, 'C' peptide and auto-antibody measures; height and weight; blood pressure, latest glycated haemoglobin and known secondary co-morbidities.

For the purposes of this study, only those cases with an initial diagnosis of type 2 diabetes and those who were initially unclassified due to lack of supportive data were reviewed. All completed clinical questionnaires were scrutinised and the physician diagnoses at one year were reviewed by the study clinicians (JPHS, TGB) in the light of the information provided. The diagnostic criteria for type 2 diabetes were the same as those used in the initial study with a supplementary criterion .

- **Definite type 2.** A case where the clinical investigations confirmed the presence of raised fasting insulin (>132 pmols/litre (20µIU/ml) or equivalent) or fasting C peptide levels (>600 pmols/litre)⁵ and/or the absence of autoimmune antibodies found in type 1 diabetes (Glutamic acid decarboxylase (GAD), Islet cell antibodies (ICA), Insulin Autoantibodies (IAA)) with no insulin requirement one year after diagnosis.

OR

A case not meeting the above criteria but in whom there had been no insulin requirement for the year after diagnosis.

Analysis: Body mass index "z-scores" or standard deviation scores (BMI SDS) at diagnosis and one year were calculated from weight and height using the 1990 UK growth standards⁶ . Blood pressure was analysed in relation to the latest available centiles for Britain⁷. Statistical analysis was performed using SPSS for Windows (version 14)⁸.

Results:

Incidence: There were a total of 101 cases eligible for review: 67 cases considered to be type 2 diabetes from the initial case report, 34 cases where the type of diabetes was originally unclassifiable. The response from the referring clinicians for the 101 cases at one year after diagnosis was 96%. Of the original 67 cases classified as having type 2 diabetes at onset, 66 were still considered to have this diagnosis at follow up which was confirmed by the study review team. In one case the referring physician re-classified the diagnosis to diabetes secondary to an unclassified, syndromic disorder. Of the 34 cases originally unclassified due to a lack of supportive data, ten were considered to meet the criteria for type 2 diabetes: in three cases because of raised fasting 'C' peptide and/or insulin measures supplied at follow up, whilst seven cases did not require treatment with insulin in the year after diagnosis. In total we concluded that there were 76 cases of type 2 diabetes in the original study period giving an overall incidence for the United Kingdom of 0.6/100,000/year (CI 0.48-0.75) a value slightly higher than that originally quoted in our incidence paper (0.55/100,000/year)⁴.

Patient demographics: Follow up data were available from 73 (96%) of the 76 cases. The mean age at follow up was 14.5 years (range 10.8-17.8) with 45% being male. 57% were white, 18% South-Asian, 17% black and 8% of mixed or Chinese ethnic origin.

Outcomes: Over the year, the mean change in weight was + 3.1Kg (maximum weight gained 22.8 Kg: maximum weight lost 11.1Kg). Females were marginally more obese than males with a mean BMI SDS at follow up of 3.0 (SD 0.8) compared to 2.7 (SD 0.9) (NS). Overall there was a mean change in BMI SDS over the year of -0.11 (males -0.18, females -0.06: range -1.53 to +1.37. NS in either gender). Although 67% of cases attained some reduction in BMI SDS at 12 months only 15% (n=11) of cases were able to reduce by 0.5 or more. At follow up, 96% of children were overweight and 78% obese according to the International Obesity Task Force guidelines⁹ compared to baseline when 96% were overweight and 80% obese.

The mean glycated haemoglobin (HbA1c) for the cohort at follow up (median time from diagnosis 343 days) was 7.5% (range 4.1-15%: median 6.5%). This information was not collected at diagnosis. An HbA1c of $\leq 7.0\%$ was achieved by 58% whilst 38% had a level greater than 8%. There was a trend for a lower HbA1c in those with an improved BMI SDS compared to those in whom it increased (7.4% (SD 2.1) vs 7.9% (SD 2.4) respectively) but this did not reach statistical significance. There was also a trend towards poorer glycaemic control in ethnic minority groups (whites 7.3% (SD 2.0): Asians 8% (SD 2.4): Blacks 8.2% (SD 2.9) but again this did not reach statistical significance. Gender did not statistically influence HbA1c (males 7.2% (SD 2.1) vs females 7.7% (SD 2.3)).

Initial and subsequent treatment: The most common treatment at initial diagnosis of type 2 diabetes was metformin: 34/72 (47%) commenced treatment with metformin, 18 (25%) started on insulin therapy alone, diet and lifestyle changes were the only initial treatment for 12 (17%) children and 4 (5.5%) commenced insulin and metformin in combination. Four children (5.5%) were given sulphonylureas as first line therapy and in four cases no data on initial treatment was recorded. There were 10 children who presented with evidence of ketosis at diagnosis, nine of whom were initiated on insulin therapy.

At one year, only 6 children (8%) of the cohort remained on diet alone or no therapy (2 coming off insulin, 2 off metformin during the course of the first year) whilst those receiving metformin alone, had increased from 34 to 44 (61%). In total, 77% (n=56) of patients received metformin as monotherapy or in combination with insulin or thiazolidinediones. Those patients initiated on diet and lifestyle changes alone (n=12) and who subsequently required drug therapy (n=10) had no significant improvement in their BMI SDS scores over the year, mean change of 0.09 (range +0.04 to -0.28).

Of the 56 children receiving metformin alone or in combination with other pharmacotherapy, the median daily dose was 1 gm (range 500mg-2.55 gm). In five children (7%), thiazolidinediones were added as adjunctive therapy to cases receiving metformin (n=3), a sulphonylurea or insulin/metformin combined. Two children were given orlistat as an adjunctive therapy for weight loss whilst one 17 year old with a BMI of 52, on insulin and metformin had been listed for gastric banding surgery.

Co-Morbidity: Although the reporting of co-morbidities was incomplete, nine females (26%) out of 34 had clinical features of polycystic ovarian syndrome with hirsutism and menstrual disturbance, in the majority supported by biochemical evidence of low sex-hormone binding globulin and luteinising hormone predominance or ultrasonography. Of these, all were receiving metformin at one year after the diagnosis of diabetes. For fourteen of the seventy-three cases (19%) no blood pressure measures were provided. Of 24 males with a recorded blood pressure, one had systolic hypertension, 6 (25%) diastolic hypertension with a blood pressure over the 98th percentile and one was normotensive on ACE inhibitor therapy. Of the 35 females with blood pressure measurements, 66% (n=23) were normotensive whilst 4 (11%) had both systolic and diastolic hypertension, 3 (9%) systolic and 5 (14%) diastolic hypertension alone; none was being prescribed anti-hypertensive agents. Fifty-five cases had screening for retinopathy and nephropathy reported. Two cases (4%) had nephropathy and none retinopathy. In sixteen cases (22%), these questions were recorded as unknown/not answered suggesting no screening had been undertaken in the year after diagnosis.

Discussion: We have confirmed an incidence rate for childhood type 2 diabetes in the UK of 0.6/100,000/year. In addition, we have shown that body mass indices do not improve as much as would be desired with current therapy although this study was unable to examine which lifestyle interventions were utilised from the data provided. Furthermore, a significant minority of patients are not achieving glycaemic targets. In addition, there are a wide variety of treatment options used at diagnosis and an absence of systematic screening for complications and co-morbidities. This indicates that type 2 diabetes care for UK children does not currently offer a fully developed and effective package.

The overall cohort change in BMI SDS (mean -0.11) is disappointing as lifestyle modification to improve body composition is central to type 2 diabetes management^{10 11}. Only 15% improved BMI SDS by more than 0.5, a level at which clinicians can be relatively confident of improved percentage fat mass¹² and improved insulin sensitivity¹³. Recent reports from studies in adult populations indicate that the year after diagnosis is often associated with significant weight loss that cannot be attributed to very poor metabolic control. In one study the rate quoted for BMI reduction was 0.4-0.6Kg/m²/year¹⁴ whilst in another after 5 years, average weight reduction was 3.3Kg¹⁵. For our population the mean weight change over one year was a gain of 3Kg indicating a failure to address lifestyle and behaviour modification adequately. In the UK, paediatric diabetes clinics are almost exclusively geared towards the treatment of type 1 diabetes and whilst dietetic support is almost universal, the emphasis is on healthy eating and insulin adjustment rather than weight loss and increased physical activity. Given the increasing prevalence of type 2 diabetes in paediatric practice, these poor weight management figures and evidence of poor metabolic control, indicate an urgent need to develop specific strategies to deal with this relatively new patient group with culturally sensitive, lifestyle and behaviour changes as the cornerstone of therapy¹⁶.

In terms of adjunctive weight loss therapy it is notable that only two cases were prescribed Orlistat, a gastro-intestinal tract, lipase inhibitor with data indicating some efficacy in adolescent obesity¹⁷ whilst bariatric surgery was only being considered in one case when evidence is beginning to emerge of reasonable efficacy in obese, post-pubertal adolescents with secondary morbidities^{18 19}.

58% of the cohort at one year were reported as having a glycated haemoglobin $\leq 7\%$ which is defined as the desired treatment goal¹⁰. Considering the previously reported issues with poor compliance in this condition²⁰, these data taken together with a mean glycated haemoglobin of 7.5% (median 6.5%), could be viewed as promising especially when compared to the 8.3% reported in a 2002 UK audit of children with all forms of diabetes in the first year of diagnosis²¹. The seemingly better glycated haemoglobin of children with type 2 diabetes compared to equivalent aged, type 1 diabetics has also been reported in a retrospective study from Australia^{5 22}. Furthermore the median glycated hemoglobin at one year of 6.5% in our cohort is very similar to that reported in adults with newly diagnosed type 2 diabetes²³ indicating that in some ways the clinical course of paediatric type 2 diabetes is similar to that in

adults. However the range in our cohort was wide and 40% of cases had a glycosylated haemoglobin above 7.5%, an identical prevalence to that described in a study of 331 children with type 2 diabetes in the western Pacific region²⁴. Furthermore 37% had a level greater than 8.0% which is associated with a substantially increased risk of complications^{25 26}.

In terms of treatment, it would appear that clinicians are adopting similar strategies to those recommended from the USA¹⁰ although there was a diverse spectrum of therapeutic pathways. Recent consensus guidelines on treatment for type 2 diabetes advocate lifestyle intervention and metformin from diagnosis as most patients fail to achieve metabolic goals with lifestyle changes alone²⁷. This is borne out by this cohort as less than 10% remained on lifestyle intervention alone at one year. It is encouraging that most clinicians chose metformin as primary pharmacotherapy with the mean dosage (1gm/day) in line with current recommendations¹⁰. Nearly all those presenting with ketosis were appropriately prescribed insulin at least initially. Half of these children were able to discontinue insulin in the first year with metformin as a replacement medication in line with current recommendations²⁷. In a few cases the thiazolidinedione class was added in combination with insulin/metformin and/or sulphonylureas within the first year of diagnosis.

Within the cohort, polycystic ovarian disease amongst females was common. An increased prevalence has been recorded in young adult females with type 2 diabetes²⁸ but there are no other data in adolescents. Given the problems of menstrual dysregulation and hirsutism associated with PCOS, adolescent females with type 2 diabetes need specific attention although all were receiving metformin which is a suitable therapy for much of the PCOS symptomatology²⁹. A significant proportion of cases had no recorded blood pressure (19%) whilst 22% had not been screened for nephropathy or retinopathy, contrary to best practice guidelines^{10 11}. This is of concern given the evidence of early complications in this patient group³⁰. It is perhaps unsurprising that no retinopathy was reported in those screened as this is not thought to be an early feature of young onset type 2 diabetes^{3 22}. However it is surprising that only two out of 55 patients screened had evidence of proteinuria; a recent USA study reported a prevalence for an elevated albumin/creatinine ratio, in the first year after diagnosis of 16%³⁰ whilst Eppens et al reported a prevalence of 28% with a median disease duration of 1.3 years²². However, the prevalence of hypertension in our cohort of 34% is similar to that reported from other studies indicating significant levels of co-morbidity^{22 24}.

This study provides relatively reliable incidence figures for overt type 2 diabetes in childhood in the UK, confirming that the condition is far less common than type 1 diabetes. However it is necessary to point out that some obese adolescents will have “silent” type 2 diabetes that is not clinically apparent³¹ whilst other overt cases at the top end of the age spectrum may have been referred directly into adult services and thus not notified to the study. Only 15% of cases achieved a clinically significant improvement in body composition suggesting that we are currently not addressing behaviour modification of lifestyle choices successfully. By designing paediatric clinics to

cater for both type 2 and type 1 diabetes we may be able to significantly improve outcome in terms of weight management and optimal metabolic control, possibly by including experts in the field of exercise and health in multi disciplinary teams. Furthermore the study indicates that most patients require pharmacotherapy at diagnosis with the requirement for increasing treatment over time. There is a need to broaden the repertoire of drugs to optimise metabolic control in childhood diabetes by means of large, randomised trials, to examine the efficacy and safety of agents used in adult medicine such as rosiglitazone which are not easy to prescribe off-licence in childhood³².

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Table 1: Baseline and one year follow up data on the cohort, showing basic demographics and treatments at diagnosis and after one year.

	At Baseline	One year follow up
Demographics		
Age	13.6 (9.9-16.8) years	14.5 (10.8-17.8) years
Sex	Male 33 (45%) / Female 40 (55%)	
Body Mass Index (BMI)	32.5 (18.7-56.2)	32.7 (21.6-55.6)
BMI SDS	2.9 (-0.34-4.5)	2.8 (0.42-4.5)
Glycated haemoglobin		7.5% (4.1-15%)
Treatment modalities		
Diet alone	17% (12/72)	8% (6/72)
Insulin alone	25% (18/72)	7% (5/72)
Metformin +/- other oral medication	47% (34/72)	65% (47/72)
Insulin and Metformin	5.5% (4/71)	13% (9/72)
Sulphonylurea +/- other oral medication	5.5% (4/72)	7% (5/72)

What is already known on this topic

- Type 2 diabetes incidence is increasing in childhood in the UK
- Weight improvement through lifestyle changes is a cornerstone of early management
- Type 2 diabetes carries as much risk of complications as type 1 diabetes

What this study adds

- A significant proportion of children with type 2 diabetes have poor glycaemic control
- Screening for complications which should be mandatory is not universal
- A framework for type 2 diabetes care in childhood needs to be developed urgently