

Type 2 diabetes mellitus among children and adolescents in Al-Ain: a case series

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حدوث الإصابة بالسكري، من النمط الثاني، لدى الأطفال في «العين»: سلسلة من الحالات
جون بونوس، موكيش منشا أقروال، سالم بن عثمان

الخلاصة: لتحديد خصائص الإصابة بالسكري من النمط الثاني، بين الأطفال والمراهقين في منطقة «العين»، تم إجراء فحص استعادي لملفات جميع الأطفال المصابين بالسكري، ممن يراجعون المستشفى التعليمي في المدينة، وذلك خلال الفترة من كانون الثاني/يناير 1990 حتى كانون الأول/ديسمبر 2001. ومن بين 96 فتى، شخّصت حالاتهم مؤخراً بالإصابة بالسكري، تم استعراض 11 منهم على أنهم مصابون بالسكري، من النمط الثاني. وكانت الخصائص السريرية لديهم هي بداية ظهور الداء مع البلوغ، والرُجحان أثوي، والبدانة، والتاريخ العائلي الحافل للإصابة بالسكري من النمط الثاني، وارتفاع مستوى الغلوكوز في البلازما عند القدوم إلى المستشفى، ووجود احتياطي ملائم من الخلايا B، وسلبية أضداد خلايا جزيرات البنكرياس في المصل. وتُضَافُ هذه السلسلة من الحالات إلى البينات التي تدلُّ على حدوث السكري من النمط الثاني، لدى الأطفال في هذه المنطقة.

ABSTRACT To characterize the features of type 2 diabetes mellitus among children and adolescents in Al-Ain, the records of every child with diabetes attending a teaching hospital in the city from January 1990 to December 2001 were retrospectively examined. Of 96 young people newly diagnosed with diabetes mellitus, 11 were identified as type 2. The clinical characteristics were: pubertal onset, female preponderance, obesity, strong family history of type 2 diabetes mellitus, high plasma glucose at presentation, adequate β cell reserve and serum pancreatic islet cell antibody negativity. This case series adds to the evidence that type 2 diabetes mellitus is emerging among children in our region.

Diabète de type 2 chez les enfants et les adolescents à Al Ain : série de cas

RÉSUMÉ Afin d'établir les spécificités du diabète de type 2 chez les enfants et les adolescents à Al Ain, les dossiers de chaque enfant diabétique ayant fréquenté un hôpital universitaire de la ville entre janvier 1990 et décembre 2001 ont été examinés rétrospectivement. Sur 96 jeunes pour lesquels le diagnostic de diabète sucré a été récemment établi, 11 ont été identifiés comme souffrant d'un diabète de type 2. Les caractéristiques cliniques étaient les suivantes : apparition à la puberté, prépondérance féminine, obésité, antécédents familiaux importants de diabète de type 2, forte glycémie lors de la présentation, réserve de cellules β adéquate et négativité des anticorps contre les cellules de îlots pancréatiques dans le sérum. Cette série de cas confirme que le diabète de type 2 augmente chez les enfants dans cette région.

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Introduction

Type 2 diabetes mellitus (DM2) is generally understood to be a disease of adults. However, over the last 2 decades, DM2 has been emerging as a public health problem in children and adolescents [1–3]. This increase has been linked to the rising problem of obesity and physical inactivity. The other risk factors are a family history of DM2, origin from certain ethnic groups, female sex and the onset of puberty [4]. Many of these children have other clinical signs of insulin resistance, such as acanthosis nigricans or polycystic ovary disease (PCOD). DM2 in children is distinct from maturity onset diabetes of the young, a rare form of diabetes associated with genetic defects in beta cell function [5].

Several epidemiological studies have shown a high prevalence of DM2 among the adult Arab population in the Gulf countries [6,7]. In the United Arab Emirates (UAE), it is estimated to be about 20% of the adult population [8]. However, the prevalence of DM2 in the paediatric population is unknown. In an earlier analysis of childhood and adolescent diabetes mellitus in the Al-Ain city, UAE, we observed an emergence of DM2 in our paediatric population [9]. This report is an attempt to characterize the features of DM2 in children and adolescents attending a 500-bed teaching hospital in Al-Ain city over a decade.

Methods

Patients and clinical features

Al-Ain Hospital is in the eastern region of the UAE. It caters to a multi-ethnic population of over 300 000 people comprising indigenous UAE Arabs, Arabs from other Middle East countries and migrants from the Indian subcontinent. The medical records of all patients in the 0–18 year age group with a diagnosis of diabetes mellitus

between 1 January 1990 and 31 December 2001 were analysed for features of DM2.

The age of onset, family history, presenting symptoms, response to treatment and presence of other co-morbid conditions (such as hypertension, acanthosis nigricans and PCOD) were documented. Blood pressure readings were obtained using a mercury sphygmomanometer with an adult-sized cuff. The blood pressure centiles were plotted according to data of the Task Force on Blood Pressure Control in Children [10]. Hypertension was diagnosed when the average systolic and/or diastolic blood pressure was \geq 95th percentile for age and sex. Body mass index (BMI) was expressed as standard deviation score using the 1990 UK growth standards [11].

Diabetes mellitus diagnostic criteria

The diagnosis of diabetes mellitus was made according to the American Diabetes Association 1997 criteria, i.e. fasting plasma glucose \geq 7 mmol/L or random plasma glucose \geq 11.1 mmol/L [12]. The classification of diabetes mellitus in these children was also made according to the scheme of the American Diabetes Association [4]; the determining factors being the presence of obesity, serum pancreatic auto-antibodies and fasting serum C peptide and insulin levels. Accordingly, a diagnosis of DM2 was made if any 2 of the following criteria were satisfied: (a) BMI $>$ 85th percentile for age and sex; (b) serum pancreatic islet cell antibodies (PICA) negative at the time of diagnosis; (c) fasting serum C peptide \geq 0.2 nmol/L [13].

Laboratory work-up and data analysis

The plasma glucose was measured by the glucose oxidase method (CX7/CX9 Synchron Systems, Beckman-Coulter Instruments, Brea, California, USA). The hospital

laboratory subscribes to an overseas external quality control from the United Kingdom (UK National External Quality Assessment Scheme), with the laboratory achieving acceptable standards. Serum C peptide and insulin levels were estimated by radioimmunoassay (DPC Diagnostics, USA). Serum PICA levels were estimated by indirect immunofluorescence at the reference Bioscientia Laboratory, Ingelheim, Germany.

The calculation of homeostasis model assessment for insulin resistance (HOMA-IR) was made by the following formula [14]: $HOMA-IR = (\text{fasting plasma insulin } \mu\text{U/mL} \times \text{fasting plasma glucose mmol/L}) / 22.5$

Patients with an HOMA-IR value above 2.7 were considered to have insulin resistance.

Data were logged into a computer database and analysed using SPSS, version 11.0 for Windows statistical analysis program running on a personal computer.

The protocol was approved by the research and ethics committee of the Faculty of Medicine and Health Sciences, UAE University.

Results

Patients

During 1990–2001, of the 96 newly diagnosed diabetic patients in the 0–18 years age group, 17 with probable non-insulin dependent diabetes mellitus were identified. However, only 11 patients fulfilled the criteria for the diagnosis of DM2. The initial investigations and follow-up data of the other 6 patients were inadequate to confirm the diagnosis of DM2. Only 1 patient had the diagnosis of DM2 between 1990–93, 3 patients between 1994–97 and 7 patients during 1998–2001.

Age, sex and ethnicity

All the patients had resided in the UAE for a minimum period of 3 years before diagnosis. The mean age at diagnosis was 14.6 (SD 3.0) years (median 15 years, range 8–18 years). There was marked female preponderance, with 10 of the 11 patients female (M:F ratio 1:10). Nine patients were Arabs from Middle East countries (including UAE), while 2 originated from the Indian subcontinent.

Clinical features

The clinical characteristics of these 11 paediatric DM2 patients at presentation are summarized in Table 1. Seven patients (64%) were detected during investigation for unrelated medical problems; 3 patients had glycaemic symptoms and 1 patient was investigated for dizziness. A positive parental history for DM2 was obtained in 8 patients with 2 of them also having second-degree relatives with DM2. One patient (no. 11) was the product of a gestational diabetes pregnancy which was managed in the obstetric unit of this hospital. In the other 4 patients whose mothers had DM2, intrauterine exposure to diabetes could not be confirmed.

Nine (82%) patients were obese, with BMI above the 85th centile. The mean BMI of this group was 30.1 (SD 9.0) kg/m² (median 27.3 kg/m², range 18.8–46.3 kg/m²). In the 2 non-obese patients (no. 1, no. 3), the diagnosis of DM2 was suggested by a negative serum PICA level, adequate serum C peptide level and a good therapeutic response to diet control and glibenclamide.

All but 1 patient had signs of puberty at the time of diagnosis. One patient (no. 7) with prepubertal onset of diabetes also developed secondary sexual characters within 6 months of diagnosis. Seven children had clinical stigmata of insulin resistance,

Table 1 Clinical characteristics at presentation of 11 children and adolescents with type 2 diabetes mellitus (DM2)

Patient	Age ^a (years); sex	Nationality	BMI (kg/m ²) Mean	SD	Presenting symptoms	DM2 family history	Treatment and follow-up	Associations
1	15; F	UAE	18.8	-0.5	Dizziness	Father	Glibenclamide 1.5 y	None
2	15; F	Omani	44.7	4.33	Incidental	Father	Metformin 3 y	Acanthosis, hypertension
3	18; F	Sudanese	23.2	0.58	Incidental	Father, grandfather, uncle, aunt	Diet alone 2 y	None
4	17; F	Omani	34.3	3.12	Glycaemic	Mother, grandmother	Glibenclamide 2 months, insulin 0.5 U/kg during pregnancy, later diet for 6 months	Acanthosis, hypertension
5	17; F	Pakistani	32.1	2.77	Glycaemic	Father, mother	Glicizide + metformin 3 y, insulin 0.3 U/kg during surgery	Cholelithiasis
6	18; F	Jordanian	46.3	4.3	Incidental	Negative	Metformin 1 y	Acanthosis, PCOD
7	8; F	Bangladeshi	21	2.07	Incidental	Negative	Insulin 1 U/kg 1 y, 2 U/kg during puberty, later glibenclamide 6 months	Acanthosis
8	14; F	UAE	32.5	3.14	Incidental	Negative	Glibenclamide 2 y, later insulin 0.5 U/kg 2 y	PCOD, hypertension
9	12; F	Palestinian	27.3	2.62	Incidental	Mother	Glibenclamide + metformin 3.5 y, later added insulin 0.15 U/kg 1 y	Acanthosis
10	14; M	Jordanian	26.3	2.63	Incidental	Mother, uncle, grandfather	Insulin for 2 weeks, later diet alone for 2 y	Hypertension
11	13; F	UAE	25	1.84	Glycaemic	Mother	Glibenclamide + metformin 3 y	None

UAE = United Arab Emirates.

BMI = body mass index. SD = standard deviation.

PCOD = polycystic ovary disease.

^aAge at diagnosis.

i.e. acanthosis nigricans, PCOD or hypertension. Patient no. 6 was found to be diabetic during investigation for hirsutism and menstrual irregularity. Patient no. 10 developed hyperandrogenaemic features 2 years after diagnosis of DM. In both these patients, the serum hormone profile was suggestive of PCOD (high serum testosterone, androstenedione and leutinizing hormone/follicle stimulating hormone ratio with normal oestradiol). Acanthosis nigricans, a cutaneous marker of insulin resistance, was observed in 5 patients. Hypertension was present in 5 patients. Patient no. 2 was found to have DM during investigation for hypertension and she required enalapril for controlling her blood pressure. None of

these children had any evidence of retinopathy (on fundus assessment by an experienced ophthalmologist) or nephropathy. One patient (no. 5) underwent laparoscopic cholecystectomy for symptomatic gallstone disease.

Laboratory data

The laboratory work-up of our patients at the time of diagnosis is summarized in Table 2. The mean random plasma glucose at presentation was 18.2 (SD 6.8) mmol/L (median 13.9 mmol/L, range 11.0–28.7 mmol/L). In patient no. 3 with inconclusive random plasma glucose at presentation, DM2 diagnosis was confirmed by the oral glucose tolerance test. All but 1 patient (no.

Table 2 Results of investigations at diagnosis for 11 children and adolescents with type 2 diabetes mellitus (DM2)

Patient	RPG (mmol/L)	Urine ketone	PICA	FPG (mmol/L)	C peptide (nmol/L)	Insulin (mU/L)	HOMA- IR	Chol. (mmol/L)	TGL (mmol/L)
1	13.1	Neg.	Neg.	10.2	1.13	17.7	8.0	n/a	n/a
2	13.9	Neg.	n/a	7.9	2.93	83.2	29.2	5.90	1.56
3	11.0 ^a	Neg.	Neg.	7.1	0.53	4.0	1.3	3.93	0.67
4	20.1	Neg.	Neg.	9.7	0.53	24.0	10.4	n/a	n/a
5	12.8	Neg.	Neg.	12.3	0.90	17.0	9.3	4.39	0.59
6	11.4	Neg.	n/a	11.3	1.30	13.8	6.9	n/a	n/a
7	27.8	Neg.	Neg.	14.9	1.50	40.8	27.0	4.11	1.22
8	13.7	Neg.	Neg.	16.3	n/a	38.0	27.5	n/a	n/a
9	25.0	Neg.	Neg.	21.1	1.33	25.6	24.0	n/a	n/a
10	28.7	Weak pos.	Neg.	7.2	0.76	9.6	3.1	2.95	0.54
11	22.2	Strong pos.	Neg.	19.7	0.43	3.5	3.1	n/a	n/a
Reference range				< 7	0.2 ^b	4–16	< 2.7	< 5.2	< 1.8

Neg. = negative; Weak pos. = weakly positive; Strong pos. = strongly positive; n/a = not available.

RPG = random plasma glucose at presentation; PICA = pancreatic islet cell antibody; FPG = fasting plasma glucose (sampled along with insulin and C peptide); HOMA-IR = homeostasis model assessment for insulin resistance; Chol. = total cholesterol; TGL = triglycerides.

^aConfirmed at 15.6 mmol/L 2 hours after 75 g glucose load; ^bCut-off > 0.2 nmol/L for DM2 diagnosis.

3), had glucosuria on presentation. Although 2 patients had ketonuria, none had biochemical evidence of metabolic acidosis. In 10 patients, fasting serum C peptide was above the cut-off value for DM2 diagnosis and the data were not available in 1 patient. In this patient (no. 8) the adequacy of β -cell function was confirmed by high fasting insulin level. The mean fasting serum C peptide of our patients was 1.13 (SD 0.73) nmol/L (median 1.02 nmol/L, range 0.43–2.93 nmol/L), while the mean fasting insulin level was 25.2 (SD 22.8) mU/L (median 17.7 mU/L, range 3.5–83.2 mU/L). HOMA-IR analysis established insulin resistance in all but 1 patient: mean 13.6 (SD 11.0) (median 17.7, range 1.3–29.2).

Serum PICA negativity at the time of diagnosis was established in 9 patients. In 2 patients (no. 2, no. 6), in whom serum PICA was not done, the DM2 diagnosis was supported by morbid obesity (BMI standard deviation score > 4 kg/m²) and adequate serum C peptide levels. Both of them had acanthosis nigricans and were responsive to biguanide therapy. Of the 5 children whose fasting lipids were measured, 1 patient had an elevated total cholesterol. Serum triglycerides were within the reference range in all 5 patients.

Treatment

One year after the onset of diabetes, none of these children showed a tendency towards ketosis.

All patients were initially managed with dietary measures, with variable response. Three patients (no. 3, no. 4, no. 10) obtained long-term metabolic control with dietary modification alone: 2 (no. 3, no. 4) achieved glycaemic control without significant weight loss, while 1 patient (no. 10) suffered a 10% weight loss.

Six (6) patients were managed with oral anti-hyperglycaemic agents: metformin (2

patients), sulfonylurea (2 patients) and metformin/sulfonylurea combination (2 patients). Patient no. 9 was responsive to oral anti-hyperglycaemic agents for 3.5 years, but later required the addition of a small dose of insulin. Patient no. 8 had to be managed with insulin 0.5 U/kg due to sulfonylurea failure after 2 years. Many of the patients required insulin therapy initially and during intercurrent medical problems (e.g. surgery and pregnancy). Generally, their insulin requirement was < 0.5 U/kg (Table 1). The high insulin requirement in patient no. 7 coincided with the onset of puberty.

Discussion

DM2 in children and adolescents is emerging as a global epidemic. It was first described in Pima adolescents in 1979 [15]; the disease was subsequently reported among various minority non-Caucasian ethnic groups in USA, Canada, Australia and New Zealand and among children from Japan, Hong Kong, Libyan Arab Jamahiriya and Bangladesh [16]. Over the years, not only is the DM2 prevalence rising among children of the above ethnic groups [4,16], but it is also being reported in Asian and Arab children from the UK [17]. More recently, DM2 has been documented in urban children from south India [18]. In our cases series, the apparent increase in the numbers of children and adolescents diagnosed with DM2 over the decade parallels the apparent rise in prevalence of DM2 in our adult population. Two population studies conducted in the Al-Ain adult population showed a rise in DM2 prevalence from 9% in 1990 [19] to 20% in 1999 [20]. The above reports suggest that in populations with a high prevalence of DM2 in adults, there may be a spillover of the disease into its paediatric population.

An important finding in the present study is that 7 of the 11 patients (64%) were asymptomatic at the time of diagnosis. In DM2 children with similar ethnic background (Asian and Arab immigrants) in the UK, Ehtisham et al. also observed a chance detection in 75% of their patients [17]. This "silent" nature of DM2 among our children points to the importance of screening Arab and Asian paediatric populations and the appreciation of this disease by paediatricians. The American Diabetes Association [4] recommends screening for DM2 in obese children, provided they have 2 of the following risk factors: (a) positive family history of DM2, (b) ethnic group with high prevalence of DM2, (c) signs of insulin resistance or conditions associated with it, i.e. acanthosis nigricans, hypertension, dyslipidaemia and PCOD. As the UAE population is a high-risk group for DM2 [8], these recommendations for DM2 screening of our obese children are applicable.

The marked female preponderance, pubertal onset of the disease and a strong family history of DM2 observed in this cohort are comparable to the pattern of DM2 observed in children of ethnic minority groups in the USA [10] and UK [17]. Nine out of our 11 patients (82%) were obese and all of them had high HOMA-IR, suggesting a major role of insulin resistance in the pathogenesis of DM2. Though earlier reports observed a more than 90% prevalence of obesity in DM2 children [2], in a recent report from India, obesity (above 120% of ideal body weight) was observed in only 50% of their children [18]. The variability in obesity prevalence between different studies is due to the different criteria used for the diagnosis of both obesity and DM2 in these children. However, it is important to recognize that, as in adults, a "non-obese" category of DM2 is present in

the paediatric population. Thus, obesity must no longer be considered as an essential prerequisite for DM2 diagnosis in children. Also, Scott et al. observed diabetic ketoacidosis at onset in 25% of their "non-insulin-dependent" diabetic children [21]. In adults, DM2 is now regarded as a heterogeneous disease with variable defects in insulin sensitivity and insulin secretion [22]. There is evidence to suggest a similar heterogeneity for the paediatric form of the disease. The atypical presentations of DM2 lead to practical difficulties in "typing" DM among children. Unfortunately, further investigations (plasma insulin, C peptide, PICA) proposed in the American Diabetes Association scheme for diabetes mellitus classification [4] are not freely available in many developing countries. It is in these countries that DM2 is increasingly being reported.

In a case-control study of native Canadian DM2 children, maternal diabetes was found to be the strongest prenatal risk factor for the development of diabetes [23]. There is strong evidence to suggest that intrauterine exposure to diabetes *per se* increases the risk for the development of diabetes and obesity in offspring, in excess of the risk attributable to genetic factors alone [24]. These observations are relevant to the UAE multiethnic community, which has a high prevalence of gestational diabetes, obesity and multiparity [25]. In the present cohort, intrauterine exposure to diabetes was confirmed in 1 child, but in another 4 patients whose mothers had DM2, this possibility could not be excluded. It is possible that the early onset of DM2 among girls in this population causes a hyperglycaemic state during pregnancy, which may further promote pubertal onset of DM2 in their offspring.

Several co-morbid cardiovascular risk factors have been reported in diabetic Pima

Indian children [2]. Similarly, a high prevalence of hypertension [17] and lipid abnormalities [18] have been observed among DM2 children from various ethnic groups. Four (36%) of our children had systemic hypertension, 6 (55%) children had either acanthosis nigricans or PCOD and 1 out of 5 children who had a serum lipid profile (20%) had hypercholesterolaemia. The HOMA-IR analysis demonstrated significant insulin resistance in all of the above patients. These co-morbid conditions substantially increase the risk of premature macrovascular disease in these children.

Being a retrospective cases series, the present analysis has several limitations. Although this report highlights the emergence of DM2 among children residing in the UAE, we were unable to determine its prevalence in our paediatric population. Only a community or school-based survey will clarify this important issue. The rising detection rate of DM2 among children attending our hospital during the study period may reflect the rising prevalence in our community. But it may also be the result of improved health care standards leading to an increased detection rate. Prior to our study period, diabetes among children was always regarded as type 1 and was routinely managed with insulin. At that time, serum PICA, serum C-peptide and insulin levels were not evaluated at the time of diagnosis in our centre. Hence, we do not have any supportive data to make a diagno-

sis of DM2 among our children in the decades preceding our study period. Another limiting factor in this study is that the obesity assessment of our children was made based on UK BMI standards.

The lack of consensus on the selection of oral hypoglycaemic agents for children with DM2 is apparent in the treatment profile of our patients. The safety and efficacy of oral hypoglycaemic agents in children is not established [4] and in the absence of definite guidelines, the choice of the agent reflects the preference of the paediatrician.

In conclusion, the present cases series adds to the evidence that the global epidemic of DM2 in adults is spreading to children and adolescents. Over the years, the age of onset of DM2 has been steadily decreasing in many population groups and the disease can no longer be called "maturity onset" diabetes. This progression seems to be the consequence of rapid modernization with major lifestyle changes resulting in childhood obesity and insulin resistance. Apart from producing glucose intolerance, insulin resistance induces a procoagulant state that may cause premature atherosclerosis. Therefore, the major public health challenge is to design and implement strategies for prevention of the ongoing epidemic of childhood obesity. Then we may be able to delay the onset of insulin resistance syndrome in genetically predisposed individuals to as late in life as possible.

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Intercountry workshop on the Arabic experimental multimedia version of the updated action-oriented school health curriculum for basic education

The World Health Organization, Regional Office for the Eastern Mediterranean, jointly with ISESCO, UNESCO and UNICEF Regional Offices, organized the above-mentioned intercountry workshop on the Arabic experimental multimedia version of the updated action-oriented school health curriculum for basic education in Damascus, Syrian Arab Republic from 3 to 5 May 2005. The objectives of the consultation were:

- to introduce the Arabic experimental multimedia version of the updated action oriented school health curriculum for basic education (CD ROM version);
- to review, share and document success stories in the field of school health education, including health promoting schools in the Region within the framework of FRESH Initiative;
- to select countries that volunteer to experiment the multimedia version of the AOSHC and provide feedback (comments and appraisal) at the end of the experimentation period (June 2006); and
- to increase institutional capacity in Member States to promote health among schoolchildren and their families, with special reference to life skills development.

Participants from Bahrain, Egypt, Iraq, Jordan, Lebanon, Kuwait, Libyan Arab Jamahiriya, Oman, Qatar, United Arab Emirates, Tunisia, Morocco, Palestine, Saudi Arabia, Sudan, Syrian Arab Republic, Yemen, the FIRDOS Syria, Ajjaluna and Islamic Makased Society Lebanon, UNESCO, UNICEF, as well as WHO concerned staff participated in this consultation.