AN ESTIMATION OF IMPAIRED FASTING GLUCOSE PREVALENCE AND RELATED FACTORS: A MIDDLE SCHOOL-BASED STUDY IN CHILDREN AGED 9-16 YEARS

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ABSTRACT

Objective: *Pre-diabetes, an intermediate stage in the progression of type 2 diabetes (T2D), is characterized by impaired fasting glucose (IFG), impaired glucose tolerance (IGT), or both. Here, we estimated IFG prevalence in middle school students in Mamak province of Ankara, Turkey, and we evaluated related factors.*

Material and Method*: We employed a cross-sectional, school-based study, and we studied 805 students aged 9-16 years to determine IFG prevalence according to the American Diabetes Association 2009 criteria. IFG was determined using a fasting plasma glucose measurement. Family history, first-degree family history, parental history, and maternal and paternal history of T2D were taken into consideration, and we measured body mass index (BMI), waist circumference, and blood pressure.*

Results: *The prevalence of IFG was found to be 9.7% in our cohort. This prevalence did not depend on sex, age, family history of T2D, BMI or blood pressure values. However, this prevalence varied according to first-degree family history, maternal history of T2D and waist circumference. Among female students, IFG prevalence varied significantly according to parental history of T2D, especially in girls with only a maternal history of T2D and abdominal obesity.*

Conclusion: *The risk of IFG increased according to a maternal history of T2D and abdominal obesity in our study population; only a maternal history of T2D and abdominal obesity increased the risk of IFG in female students.*

Keywords: *Impaired fasting glucose, pre-diabetes, maternal history of type 2 diabetes, abdominal obesity, middle school students.* ***Nobel Med 2016; 12(2): 45-52***

BOZULMUŞ AÇLIK GLİKOZ PREVALANSININ TAHMİN EDİLMESİ VE İLGİLİ FAKTÖRLER: 9-16 YAŞINDAKİ ÇOCUKLARDA ORTAOKUL TABANLI BİR ÇALIŞMA

ÖZET

Amaç: Prediyabet, tip 2 diyabetin (T2D) gelişiminde ara bir dönem olup, ya bozulmuş açlık glikozu (BAG) ya bozulmuş glikoz toleransı (BGT) veya her ikisinin birlikte bulunması ile karakterizedir. Bu çalışmanın amacı, Ankara'nın Mamak ilçesindeki ortaokul öğrencilerinde BAG prevalansını tahmin etmek ve ilgili faktörleri değerlendirmektir.

Materyal ve Metot: Okul tabanlı kesitsel bir çalışmadır ve 9-16 yaşındaki 805 öğrenci, Amerikan Diyabet Derneği 2009 kriterlerine göre BAG prevalansını değerlendirmek için araştırma kapsamına alınmıştır. BAG, açlık plazma glikoz ölçümüne göre belirlenmiştir. Aile öyküsü, birinci derece akrabalar ile anne ve babadaki T2D öyküsü dikkate alınmış ve beden kitle indeksi (BKİ), bel çevresi ve kan basıncı değerleri ölçülmüştür.

Bulgular: BAG prevalansı % 9,7 olarak bulunmuştur. Prevalans cinsiyete, yaşa, aile öyküsüne, BKİ ve kan basıncı değerlerine göre değişmemiş; ancak, birinci derece akrabalar ile annede T2D öyküsü bulunma durumuna ve bel çevresine göre değişmiştir. BAG prevalansı, anne-baba öyküsü bulunan kız öğrencilerde özellikle de yalnızca annesinde T2D öyküsü bulunan ve abdominal obez olanlarda anlamlı ölçüde değişmiştir.

Sonuç: Çalışma grubundaki BAG riski, annede T2D öyküsü ve abdominal obezite bulunması durumunda artarken, kız öğrencilerdeki riski, yalnızca annede T2D öyküsü bulunma durumu ve abdominal obezite anlamlı ölçüde artırmıştır.

Anahtar kelimeler: Bozulmuş açlık glikozu, prediyabet, annede tip 2 diyabet öyküsü, abdominal obezite, ortaokul öğrencileri. **Nobel Med 2016; 12(2): 45-52**

INTRODUCTION

In parallel with the recent worldwide epidemic of obesity, the prevalence of childhood obesity has increased dramatically over the past three decades.[1-9] Concomitant with this rise, recent data reveal a corresponding increase in the incidence of type 2 diabetes (T2D) in children and adolescents in both developed and developing countries, and pre-diabetes is becoming more common in obese children and adolescents.[2,5,8,10-12]

The progression from normal glucose tolerance to overt T2D involves an intermediate stage, referred to as prediabetes, characterized by isolated impaired fasting glucose (IFG), isolated impaired glucose tolerance (IGT), or both IFG and IGT.[3,13] IFG is associated with an enhanced risk for developing T2D.[8,12,14-19] This state has been studied extensively in adults, but corresponding studies in children and adolescents have been rare.[3] Obesity is associated with an increased likelihood for having either IFG or IGT, but the data are limited in terms of defining the significance of pre-diabetes in children and adolescents.[8,14]

In Turkey, there was a recent representative study "The Turkish Epidemiology Survey of Diabetes, Hypertension, Obesity and Endocrine Disease (TURDEP) II" reporting the prevalence of IFG, IGT, and T2D according to the American Diabetes Association (ADA) criteria in a population older than 20. According to this cross-sectional, population-based survey, the age-standardized diabetes prevalence was 13.7%, the crude prevalence of pre-diabetes was 30.8%, the isolated IFG prevalence was 14.7%, the isolated IGT prevalence was 7.9%, and the combined IFG and IGT prevalence was 8.2%.[20] Compared with the TURDEP I findings from 1997-1998, the prevalence of diabetes increased by 90%.[21] Although there has been a constant increase in the rates of obesity in Turkish children and adolescents, limited data are available pertaining to the risk of pre-diabetes and T2D in this group.[11,22] According to a cross-sectional, population-based study by Uckun-Kitapci *et al.* in school children aged 12-18 years, the prevalence of IFG, defined as fasting plasma glucose (FPG) levels ≥110 mg/dL, was 1.97%.[4]

Here, we estimate the prevalence of IFG as one of the situations of pre-diabetes in middle school students in Mamak province of Ankara, Turkey, and we evaluate the related factors. We employ a school-based study of a representative sample of middle school students in this region. To the best of our knowledge, this study is the only one to investigate pre-diabetes via IFG in this age group.

MATERIAL AND METHOD

We conducted a cross-sectional study of 805 middle school (6^{th}-8^{th} grade) students in Mamak province of Ankara, Turkey. Forty-seven middle schools are located in this province, and a total of 13,965 students attend

these schools. Our study sample was determined via the cluster sampling method, and we calculated our sample by taking into consideration the nearest IFG prevalence (11.4%) in adults aged 20-24 years in the TURDEP II study.[20] We estimated that our sample size would be able to detect a prevalence of IFG with a 0.20 deviation in the prevalence noted above, based on an error level of 5%, a statistical power of 0.80, and a confidence interval of 95%. We recovered a minimum sample size of 708 students that satisfied these criteria.

This study was approved by the Research Ethics Committee of the Faculty of Medicine, Ankara University, Turkey (Decision Number: 06-235-13; Date of Decision: April 8, 2013).

Study Protocol

Students were required to meet the following inclusion criteria:1) no prior diagnoses of diabetes, coagulation defects, or mental and physical disorders according to a self report;2) willingness to participate in the study; and 3) written, informed consent from their parents and agreement from the students themselves to arrive after an overnight fast (≥10 h). The status of fasting and whether the inclusion criteria were satisfied or not were verified via a questionnaire, and 805 eligible students were included in the study.

We used a glucometer (Accu-CHEK Performa Nano; Roche Diagnostics, Germany) to measure FPG. Although a glucometer measures whole blood glucose concentration, the instrument yields values calibrated for plasma glucose. We opted to use a glucometer to estimate the IFG prevalence in this study because the device was easy to use, less invasive than venous testing and it was free of charge. IFG was defined according to the 2009 ADA criteria as a FPG ≥ 100–125 mg/dL.[13] The sensitivity of the glucometer was 96% when the plasma glucose level was ≥ 100 mg/dL.[23]

We measured the height and weight of the participants using a SECA portable stadiometer (SECA 213, SECA, Germany) and TEFAL (Premio PP1051, Groupe SEB, France) portable electronic scale, and the measurements were recorded to the nearest 0.1 cm and 0.1 kg, respectively.

We determined waist circumference, blood pressure and body mass index (BMI) values of the students. BMI values by age and sex were evaluated according to the International Obesity Task Force (IOTF) criteria as underweight, normal, overweight and obese.[24] We measured waist circumferences at the level of the iliac crest on the midaxillary line at minimal respiration to the nearest 0.1 cm with a SECA circumference measuring tape (SECA 203). The values of waist circumference below the 75th percentile were evaluated normal; values above the 75th percentile were evaluated as abdominal obesity.[25,26]

We measured blood pressure using an aneroid sphygmomanometer with appropriate cuffs (ERKATEST perfect aneroid, ERKA, Germany) to the size of the student's upper right arm while he or she was sitting quietly for 10 minutes and seated with his or her back supported, feet on the floor, and right arm supported. The evaluation of blood pressure was performed according to the IOTF criteria; it has been determined that Turkish children's blood pressure percentiles are in accordance with the values reported in "The Fourth Report on the Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescents."[27,28] Normal blood pressure was defined as systolic and diastolic blood pressure levels that were below the 90th percentile; prehypertension was defined as systolic or diastolic blood pressure between the 90th and 95th percentiles, and hypertension was defined as systolic or diastolic blood pressure above the 95th percentile by sex, age and height.[29]

A student was defined as having a family history of T2D if at least one family member such as natural mother, father, sibling, grandmother, grandfather, aunt, uncle and/or cousin was reported to have T2D. If the respondent indicated that his/her natural mother, natural father or any full sister or brother had T2D, the student was defined as having a first-degree family history of T2D.

The parental history of T2D was classified into four groups:1) only a maternal history;2) only a paternal history of T2D;3) both a maternal and paternal history of T2D; and 4) no parental history of T2D.

The students whose mothers were reported to have T2D-irrespective of whether their fathers were reported to have the disease or not-were evaluated as having a maternal history of T2D, and the students whose fathers were reported to have the disease-irrespective of whether their mothers were reported to have T2D or not-were evaluated as having a paternal history of T2D.

Statistical Analysis

Descriptive statistics were expressed as mean ± standard deviation, frequencies, and percentages. The associations between dichotomous variables and IFG were analyzed using a chi-squared test and Fisher's exact test. We assessed the risk of IFG using multiple logistic regression analysis. The inclusion (exclusion) criteria of independent variables were 10% (20%).

Table 1. Sociodemographic characteristics, family history of T2D, blood pressure, BMI, waist circumference and glucose measurements

	Number	%
Sex		
Female	354	44.0
Male	451	56.0
Age (years)		
9	1	0.1
10	(-)	0.0
11	92	11.4
12	293	36.4
13	258	32.1
14	145	18.0
15	15	1.9
16	1	0.1
Grade		
6th	240	29.8
7th	308	38.3
8th	257	31.9
Family history T2D		
Yes	384	47.7
No	421	52.3
First-degree family history of T2D		
Yes	98	12.2
No	707	87.8
Parental history of T2D		
Only maternal	55	6.8
Only paternal	33	4.1
Parental	8	1.0
Neither parent	709	88.1
Maternal history of T2D		
Yes	61	7.6
No	744	92.4
Paternal history of T2D		
Yes	43	5.3
No	762	94.7
BMI		
Underweight	72	8.9
Normal	548	68.1
Overweight	145	18.0
Obese	40	5.0
Waist circumference		
Normal	531	66.0
Abdominal obese	274	34.0
Blood pressure		
Normotensive	610	75.8
Prehypertensive	112	13.9
Hypertensive	83	10.3
FPG		
Normal	726	90.2
IFG	78	9.7
Diabetes	1	0.1
Total	**805**	**100.0**

IFG: Impaired fasting glucose, **T2D:** type 2 diabetes, **BMI:** body mass index, **FPG:** fasting plasma glucose.

We performed all analyses using SPSS (Statistical Package for Social Sciences, SPSS Inc., Chicago, IL, USA) version 11.5 for Windows. We considered results with a two-tailed $p<0.05$ to be statistically significant.

RESULTS

The middle school students were aged between 9 and 16 years, and the mean age of the cohort was 12.6±0.9 years. Of the 805 students, 56.0% were male, and 29.8%, 31.9% and 47.7% were 6th, 7th and 8th grade, respectively. Roughly 12.2% of students had a first-degree family history of T2D. According to parental history of T2D, 6.8% of students had only a maternal history, 4.1% had only a paternal history, 1.0% had a parental history, and 88.1% had no parental history of T2D. 7.6% of students had maternal, and 5.3% had paternal history. The mean BMI of the cohort was 19.8±3.6 kg/m^2 (range: 12.9-33.9 kg/m^2), the mean waist circumference was 66.4±9.5 cm (range: 46.0-112.0 cm), the mean systolic blood pressure was 101.1±10.6 mm Hg (range: 70.0-160.0 mm Hg) and mean diastolic blood pressure was 65.9±10.6 mm Hg (range: 40.0-100.0 mm Hg). Of all the students, 8.9% were underweight, 68.1% were of normal weight, 18.0% overweight and 5.0% were obese; 34.0% were abdominal obese; 75.8% were normotensive, 13.9% were prehypertensive and 10.3% were hypertensive. The mean FPG was 90.4±7.6 mg/dL, and the prevalence of IFG was 9.7% (95% CI:7.7-11.7%), and one student's FPG was 143 mg/dL (Table 1).

The prevalence of IFG did not vary according to sex, age, or family history of T2D, but it varied significantly with first-degree family history of T2D. The percentage of students with IFG doubled from 8.8% to 16.5% for students with a first-degree family history of T2D. A family history of T2D did not influence the IFG prevalence in female or male students ($p=0.377$ and $p=0.094$, respectively). In female students with a first-degree family history of T2D, the prevalence of IFG (20.8%) was higher than in students without a first-degree family history of T2D (8.5%); the difference was statistically significant ($p=0.006$). However, a similar association was not observed in male students ($p=0.499$). The prevalence of IFG varied according to parental history of T2D: Students with only a maternal history of T2D had the highest prevalence of IFG (22.2%) compared with students with a parental (12.5%), only paternal (9.1%) or no parental (8.7%) history of T2D ($p=0.015$). Female students with only a maternal history of T2D exhibited the highest prevalence of IFG ($p=0.002$); male students did not exhibit any significant difference ($p=0.392$).

Maternal history of T2D influenced the overall prevalence of IFG, and the prevalence was significantly higher in students with a maternal history of T2D (21.7%) than in students without a maternal history of T2D (8.7%; $p=0.001$). On the other hand, when we analyzed female and male students separately, we

observed that female students with a maternal history of T2D exhibited a higher prevalence of IFG than students without a maternal history (p=0.005); the prevalence of IFG did not vary significantly among male students according to maternal history of T2D (p=0.147). A paternal history of T2D did not influence the prevalence of IFG in the study population in either female or male students (p=0.593, p=0.634, and p=0.673, respectively).

There was no significant association between the prevalence of IFG and BMI (p=0.124), but the prevalence varied significantly with waist circumference (p=0.008). Among abdominal obese students, the prevalence of IFG was approximately two-fold (13.6%) higher than the prevalence of IFG among students with a normal waist circumference (7.7%). The prevalence of IFG also varied with abdominal obesity in female students (p=0.012), but it did not vary in male students (p=0.259).

Based on our analysis of IFG prevalence according to blood pressure levels, prehypertensive students were combined with normotensive ones, and the prevalence of IFG among students with normal (hypertensive) blood pressure levels was 9.4% (12.0%). This difference, however, was not statistically significant (p=0.446) (Table 2).

Among the 383 students with a family history of T2D, 39 (10.2%) had IFG; of these, 27 (69.2%), 10 (25.6%), and 2 (5.1%) were under/normal-weight, overweight, and obese, respectively. The prevalence of IFG did not vary significantly according to BMI (p=0.595). A similar association was also observed in students without a family history of T2D according to BMI values (p=0.098).

When the variables likely to have significant association with IFG prevalence were used in a multiple logistic regression analysis, we found that a maternal history of T2D and abdominal obesity remained significant contributors to the risk of IFG in our study population. The risk of IFG was 2.7 times higher in students with a maternal history of T2D than in students without such a history (95% CI: 1.4–5.3), and abdominal obesity was also found to increase the risk of IFG by 1.8-fold (95% CI: 1.1–2.9) compared with students without a maternal history of T2D (Table 3).

When we performed multiple logistic regression analysis on the female and male students separately, we found that the risk of IFG was 6.5-fold higher (95% CI: 2.3-18.3) in female students with only a maternal history of T2D than in female students with no parental history of T2D. In abdominally obese female students, the risk of IFG was also 2.1 times (95% CI: 1.1-4.1) higher than in those without. However, the risk of IFG did not change in male students according to parental history or abdominal obesity (Table 4).

Table 2. Prevalence of IFG according to sex, age, family characteristics, BMI, waist circumference and blood pressure

	Normal	IFG	p
Sex (%)			
Female	406 (90.2)	44 (9.8)	0.934
Male	320 (90.4)	34 (9.6)	
Age (years) (%)			
9	(-) (0.0)	1 (100.0)	
10	(-) (0.0)	- (0.0)	
11	85 (92.4)	7 (7.6)	
12	263 (89.8)	30 (10.2)	0,102
13	234 (91.1)	23 (8.9)	
14	129 (89.0)	16 (11.0)	
15	14 (93.3)	1 (6.7)	
16	1 (100.0)	(-) (0.0)	
Family history of T2D (%)			
Yes	344 (89.8)	39 (10.2)	0.660
No	382 (90.7)	39 (9.3)	
First-degree family history of T2D (%)			
Yes	81 (83.5)	16 (16.5)	0.016
No	645 (91.2)	62 (8.8)	
Parental history of T2D (%)			
Only maternal	42 (77.8)	12 (22.2)	
Only paternal	30 (90.9)	3 (9.1)	0.015
Parental	7 (87.5)	1 (12.5)	
Neither parent	647 (91.3)	62 (8.7)	
Maternal history of T2D (%)			
Yes	47 (78.3)	13 (21.7)	0.001
No	679 (91.3)	65 (8.7)	
Paternal history of T2D (%)			
Yes	39 (90.7)	4 (9.3)	0.593[a]
No	687 (90.3)	74 (9.7)	
BMI (%)			
Underweight	68 (94.4)	4 (5.6)	
Normal	498 (90.9)	50 (9.1)	0.124
Overweight	123 (85.4)	21 (14.6)	
Obese	37 (92.5)	3 (7.5)	
Waist circumference (%)			
Normal	490 (92.3)	41 (7.7)	0.008
Abdominal obese	236 (86.4)	37 (13.6)	
Blood pressure (%)			
Normotensive/prehypertensive	653 (90.6)	68 (9.4)	0.446
Hypertensive	73 (88.0)	10 (12.0)	
Total[b]	**726 (90.3)**	**78 (9.7)**	

[a]: Fisher's exact test, [b]: one student was excluded because his FPG was 143 mg/dL, **IFG:** impaired fasting glucose, **T2D:** type 2 diabetes, **BMI:** body mass index.

DISCUSSION

The primary purpose of this study was to estimate the prevalence of IFG as one of the states of pre-diabetes in middle school students in Mamak province of Ankara, Turkey and to evaluate the related factors. The students who were eligible to participate in the study were representative of middle school students in this

Table 3. Multiple logistic regression analysis to explore the contribution of maternal history of T2D and waist circumference on IFG in the study population

	Odds ratio	95% CI	*p*
Maternal history of T2D			
No (r)			
Yes	2.690	1.375-5.263	0.004
Waist circumference			
Normal (r)			
Abdominal obese	1.784	1.109-2.869	0.017

T2D: type 2 diabetes, **r:** reference value, **CI:** confidence interval.

Table 4. Factors associated with the risk of IFG in female students

	Odds ratio	95% CI	*p*
Parental history of T2D			
Neither (r)[a]			
Only maternal	6.539	2.338-18.289	0.001
Waist circumference			
Normal (r)[a]			
Abdominal obese	2.113	1.111-4.019	0.023

T2D: type 2 diabetes, **r:** reference value, **CI:** confidence interval.

province. To the best of our knowledge, this study was the first of its kind to investigate pre-diabetes via IFG prevalence in this age group according to ADA criteria in Turkey.

We found that the prevalence of IFG was 9.7% in our cohort, and one student was determined to have a FPG of 143 mg/dL. In a study by Uckun-Kitapci *et al.*, the prevalence of IFG in a similar age group was reported to be 1.97%, which differs from our result. These authors used World Health Organizations criteria where IFG was defined according to FPG level: 110-125 mg/dL was taken into consideration.[4] In two other Turkish studies conducted with similar age groups that employed the ADA criteria, the prevalence of IFG was found to be 3.8% and 6.6%, respectively.[6,7] These two prevalences are lower than our prevalence; the study populations of these previous studies did not represent the population of the related age groups. In a study by Moadab *et al.* of a population in Isfahan, Iran, the prevalence of IFG, according to ADA criteria, was 4.6%.[11] Our result is similar to what had been recently reported by Li *et al.* in an American adolescent population (aged 12-19 years) and Rodriguez-Moran *et al.* in a Mexico population (aged 7-15 years). These authors reported IFG prevalences of 12.1% and 12.6%, respectively, according to the ADA criteria.[16,30]

In the present study, IFG prevalence did not change according to sex, a result that is similar with the findings of Moadab *et al.* and Mazur *et al.*[11,31] The prevalence of IFG also did not vary with age, as has been reported in several studies.[4,6,11,32-34]

The IFG prevalence that we recovered was not significantly associated with a family history of T2D, which is similar to the findings of a Turkish study conducted by Uckun-Kitapci *et al.*[4]

The prevalence of IFG was higher among students with a first-degree family history of T2D than in students without a first-degree family history of T2D. A similar association has been also reported in a study by Klein *et al.*[35] Among female students, the prevalence of IFG was significantly higher in students with a first-degree family history of T2D compared with students without a first-degree family history of T2D. However, no such relation was observed among male students. This result is in consistent with the findings of Rodriguez-Moran *et al.*[30]

Some studies have reported that a maternal history of T2D is more effective at transmitting the disease compared with a paternal history.[35-38] Since IFG is an intermediate stage in the progression of T2D, a significant increase in IFG prevalence among students with only a maternal history of T2D is thought to be important for alerting an individual about the risk of disease. Another point in this study is that female students with only a maternal history of T2D exhibited a higher prevalence of IFG than male students with only a maternal history of T2D. Therefore, this finding is consistent with the results of Karter *et al.* These authors found that excess maternal transmission was reported to be stronger in female siblings than in male siblings when IFG was taken into consideration as one of the states in the development of T2D.[36]

In terms of IFG prevalence according to be BMI, we did not find any significant association. This result is similar to the findings of Uckun-Kitapci *et al.* and Weigensberg *et al.*[4,32]

We noted that IFG prevalence varied with abdominal obesity; it increased significantly in abdominally obese students compared with students who were not abdominally obese. To the best of our knowledge, there have been no other studies conducted yet that have evaluated waist circumference and IFG. However, there have been many studies assessing waist circumference and risk of T2D. Gautier *et al.* suggested abdominal obesity as a potential value for monitoring waist circumference over time in IFG.[39]

Here, we did not find any relationship between the prevalence of IFG and blood pressure levels, which is consistent with the results of Reinehr *et al.* and Weigensberg *et al.*[5,32]

Based on our multiple logistic regression analysis, we observed that a maternal history of T2D and abdominal

obesity in the study population and only a maternal history of T2D and abdominal obesity in female students were found to be the primary risk factors for IFG. In a study by Mitchell *et al.*, the risk of T2D was 1.59 times higher in women with only a maternal history of T2D than in women with only a paternal history of T2D.[38] IFG is an intermediate stage in the progression to T2D, and we found that students with a maternal history of T2D had a 2.7-fold increased risk for IFG compared with those without a maternal history of T2D. Therefore, it is thought that maternal history may play an important role in the development of pre-diabetes; only a maternal history of T2D seems to play an important role in female students in addition to abdominal obesity.

CONCLUSION

The results of this study suggest that IFG prevalence is positively associated with a maternal history of T2D and abdominal obesity; in female students, the risk of IFG increases significantly only according to a maternal history and abdominal obesity.

Screening for T2D will identify individuals with intermediate hyperglycemia (IFG and/or IGT) who may benefit from interventions to prevent or delay progression to diabetes and cardiovascular disease and other complications.[40] In this context, measuring IFG can be taken into consideration as a screening method for secondary prevention in order to prevent the development of T2D in adolescents. By following up female students with IFG and only a maternal history of T2D and abdominal obesity, we may be able to prevent T2D better.

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REFERENCES

1. Lobstein T, Baur L, Uauy R for the IASO International obesity taskForce. Obesity in children and young people: a crisis in public health. Obes Rev 2004; 5: 4-85.
2. Baranowski T, Cooper DM, Harrell J, et al. The STOPP-T2D Prevention Study Group. Presence of diabetes risk factors in a large U.S. Eighth-Grade Cohort. Diabetes Care 2006; 29: 212-217.
3. Weiss R, Dufour S, Taksali SE, et al. Pre-diabetes in obese youth: a syndrome of impaired glucose tolerance, severe insulin resistance, and altered myocellular and abdominal fat partitioning. Lancet 2003; 20: 951-957.
4. Kitapçı-Uçkun A, Teziç T, Fırat S, et al. Obesity and type 2 diabetes mellitus: A population-based study of adolescents. J Pediatr Endocrinol Metab 2004; 17: 1633-1640.
5. Reinehr T, Wabitsch M, Kleber M, et al. Parental diabetes, pubertal stage, and extreme obesity are the main risk factors for pre-diabetes in children and adolescents: a simple risk score to identify children ar risk for pre-diabetes. Pediatr Diabetes 2008; 10: 395-400.
6. Babaoğlu K, Hatun Ş, Arslanoğlu İ, et al. Evaluation of glucose intolerance in adolescents relative to adults with type 2 diabetes mellitus. J Pediatr Endocrinol Metab 2006; 19: 1319-1326.
7. Atabek ME, Pirgon O, Kurtoğlu S. Assessment of abnormal glucose homeostasis and insulin resistance in Turkish obese children and adolescents. Diabetes Obes Metab 2007; 9: 304-310.
8. Gomez-Diaz R, Aguila-Salinas CA, Moran-Villota S, et al. Lack of agreement between the revised criteria of ımpaired fasting glucose and ımpaired glucose tolerance in children with excess body weight. Diabetes Care 2004; 27: 2229-2233.
9. Bundak R, Furman A, Gunoz H, et al. Body mass index references for Turkish Children. Acta Paediatr 2006; 95: 194-198.
10. Kelly LA, Lane CJ, Koebnick C, et al. Parental History and risk of type 2 diabetes in overweight latino adolescents. Diabetes Care 2007; 30: 2700-2705.
11. Moadab MH, Kelishadi R, Hashemipour M, Amini M, Poursafa P. The prevalance of impaired fasting glucose and type 2 diabetes in a population-based sample of overweight/obese children in the Middle East. Pediatr Diabetes 2010; 11: 101-106.
12. D'Adamo E, Caprio S. Type 2 diabetes in Youth: Epidemiology and pathophysiology. Diabetes Care 2011; 34: 161-165.
13. American Diabetes Association. Position statement. diagnosis and classification of diabetes mellitus. Diabetes Care 2010; 33: 62-69.
14. Bacha F, Lee S, Gungor N, Arslanian SA. From pre-diabetes to type 2 diabetes in obese youth. Pathophysiological characteristics along the spectrum of glucose dysregulation. Diabetes Care 2010; 33: 2225-2231.
15. Li C, Ford ES, Zhao G, Mokdad AH. Prevalence of pre-diabetes and its association with clustering of cardiometabolic risk factors and hyperinsulienmia among U.S. adolescents. National health and nutrition examination survey 2005-2006. Diabetes Care 2009; 32: 342-347.
16. Greig F, Hyman S, Wallach E, Hildebrandt T, Rapaport R. Which obese youth are at increased risk for type 2 diabetes? Latent class analysis and comparison with diabetic youth. Pediatr Diabetes 2012; 13: 181-188.
17. American diabetes association. screening for type 2 diabetes. Diabetes Care 2004; 27: 11-14.
18. Cowie CC, Rust KF, Ford ES, et al. Full accounting of diabetes and pre-diabetes in the U.S. Population in 1988-1994 and 2005-2006. Diabetes Care 2009; 32: 287-294.
19. Unwin N, Shaw J, Zimmet P, Alberti KGMM. Writing comittee. Impaired glucose tolerance and impaired fasting glycaemia: the current status on definition and intervention. Diabet Med 2002; 19: 708-723.
20. Satman I, Omer B, Tutuncu Y, et al. Twelve-year trends in the prevalence and risk factors of diabetes and pre-diabetes in Turkish adults. Eur J Epidemiol 2013; 28: 169-180.
21. Satman I, Yılmaz T, Sengul A, et al. The TURDEP Group. Population-based study of diabetes and risk characteristics in Turkey: results of the Turkish diabetes epidemiology study (TURDEP). Diabetes Care 2002; 25: 1551-1556.
22. Cizmecioglu FM, Etiler N, Hamzaoglu O, Hatun S. Prevalence of metabolic syndrome in schoolchildren and adolescents in Turkey: A population-based study. J Pediatr Endocrinol Metab 2009;8:703-714.
23. Freckmann G, Schmid C, Baumstrak A, et al. System accuracy evaluation of 43 blood glucose monitoring systems for self-monitoring of blood glucose according to DIN EN ISO 15197. J Diabetes Sci Technol 2012; 6: 1060-1075.
24. Extended ınternational (IOTF) body mass ındex cut-offs for thinness, overweight and obesity in children. November 11,

2013. http://www.iaso.org/resources/aboutobesity/child-obesity/newchildcutoffs.
25. Anthropometric Reference Data for Children and Adults: United States, 2007-2010. Centers for disease control and prevention. National Center for Health Statistics. Hyattsville, Maryland October 2012. DHHS Publication No. (PHS) 2013-1602. June 10, 2013 http://www.cdc.gov/nchs/about/major/nhanes2005-2006/nhanes05_06.htm.
26. Hatipoğlu N, Öztürk A, Mazıcıoğlu MM, et al. Waist circumference percentiles for 7 to 17 year-old Turkish children and adolescents. European J Pediatrics. 2007; doi: 10.1007/s0043100705023.
27. Tümer N, Yalçınkaya F, İnce E, et al. Blood pressure nomograms for children and adolescents in Turkey. Pediatr Nephrol 1999; 13: 438-443.
28. American academy of pediatrics. national high blood pressure education program working group on high blood pressure in children and adolescents. The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. Pediatrics 2004; 114: 555-576.
29. Rodriguez-Cruz E. Pediatric Hypertension. Medscape December 30, 2013 http://emedicine.medscape.com/article/889877-overview#showall.
30. Rodriguez-Moran M, Guerrero-Romero F, Aradillas-Garcia R, et al. Obesity and family history of diabetes as risk factors of impaired fasting glucose implications for the early detection of pre-diabetes. Pediatr Diabetes 2010; 11: 331-336.
31. Mazur A, Gryzwa M, Malecka-Tendera E, Telega G. Prevalence of glucose intolerance in school age children. Population based cross-sectional study. Acta Paediatr 2007; 96: 1799-1802.
32. Weigensberg MJ, Ball GDC, Shaibi GQ, Cruz M, Goran MI. Decreased B-Cell Function in Overweight Latino Children With Impaired Fasting Glucose. Diabetes Care 2005; 28: 2519-2524.
33. Ranjani H, Sonya J, Anjana RM, Dipdiabetes UK, Moham V. Prevalance of glucose intolerance among children and adolescents in urban south India (ORANGE-2). Diabetes Technol Ther 2013; 15: 13-19.
34. Cizmecioglu FM, Hatun S, Kalaca S. Metabolic syndrome in obese Turkish children and adolescents: comparison of two diagnostic models. Tur J Ped 2008; 50: 359-365.
35. Klein BEK, Klein R, Moss SE, Cruickshanks KJ. Parental history of diabetes in a population-based study. Diabetes Care 1996; 19: 827-830.
36. Karter AJ, Rowell SE, Ackerson LM, et al. The Northern California Kaiser permanente diabetes registry. Excess maternal transmission of type 2 diabetes. Diabetes Care 1999; 22: 938-943.
37. Mitchell BD, Valdez R, Hazuda HP, et al. Differences in the prevalence of diabetes and impaired glucose tolerance according to maternal or paternal history of diabetes. Diabetes Care 1993; 16: 1262-1267.
38. Meigs JB, Cupples LA, Wilson PWF. Parental transmission of type 2 diabetes. the Framingham offspring study. Diabetes 2000; 49: 2201-2207.
39. Gautier A, Roussel R, Ducluzeau P, et al. For the Study DESIR Group. Increases in waist circumference and weight as predictors of type 2 diabetes in individuals with impaired fasting glucose: influence of baseline BMI. Diabetes Care 2010; 33: 1850-1852.
40. "Screening and Diagnosis". International Diabetes Federation, 2012. Clinical Guidelines Task Force. Global Guideline for Type 2 Diabetes. P: 11.