Aswan University Medical Journal, volume 3 / No.2/ December 2023 (129-137) Online ISSN: **2735-3117**

ORIGINAL ARTICLE

Clinical Characters of Egyptian Children with Maturity Onset Diabetes of the Young type three

Ahlam Mohamed Hafez Mohamed[1*], Hanan Mohamed Abd-El Moneim[1], Shereen Abdelghaffar Taha[2], Hanan Ali Ahmed Madany[3] and Marwa Farouk Mira[2]

[1] pediatric department, faculty of medicine, Aswan, University, Egypt
[2]Pediatrics department, faculty of medicine, Cairo University, Egypt
[3] Clinical and Chemical Pathology department, faculty of medicine, Cairo University, Egypt

ABSTRACT

Keywords: Children; Egyptian; MODY3; Maturity Onset Diabetes of the young

*__Corresponding author__: Ahlam Mohamed Hafez Mohamed

mobile: +96567658237

E-mail address: ahlamhafez716@gmail.com

Background: Maturity-onset diabetes of the young (MODY) is a type of monogenic diabetes. MODY3 is more frequent than previously thought. **Aim and objectives** detect clinical characteristics of cases of MODY 3 as regards to; age of disease onset, insulin dose, family history and complications in Egyptian children. **Methods:** This case control study was conducted on 20 cases and 10 controls. All subjects were subjected to full history taking, complete clinical examination and laboratory tests, including; C peptide assays, Glycosylated hemoglobin (HbA1c), Fasting and 2 hours postprandial blood sugar levels, Triglyceride, LDL cholesterol, Measurement of anti-bodies to glutamic acid decarboxylase (anti-GAD) and anti-islets antibodies. **Results:** All cases (100%) had a positive family history of DM. All cases were negative for Anti-Gad and Anti-Islet. The mean age on onset of DM was 11.08 ± 2.77 years, the mean duration of DM was 2.2 ± 0.16 years. Regarding complications; 10% had neuropathy. Regarding treatment; 5% of cases were treated by oral hypoglycemic, 15 % were treated by insulin and oral hypoglycemic drugs, 15% were managed by healthy life style and 65% of cases treated by insulin with a mean dose 0.34 ± 0.19**. Conclusion** we suggest that a thorough clinical evaluation, including a family history, phenotypic features, a response to diabetes-specific autoantibodies, and an assessment of endogenous insulin secretion, may be a more effective first step in identifying diabetic children who require genetic testing for MODY.

INTRODUCTION

Type 1 diabetes, which is a form of autoimmune disease, and type 2 diabetes, are the most recognized types of diabetes mellitus, and Type 2 diabetes is a polygenic illness that is affected by environment as well as genetics. Now that hybrid forms of diabetes are far less common, it is known that there are more than just two forms of diabetes. A kind of monogenic diabetes known as maturity-onset diabetes of the young (MODY) was first identified as a mild and asymptomatic form of the disease that was seen in non-obese children, adolescents, and young adults. (1)**.**

People under the age of 25 are frequently affected by MODY, which results from heterozygous mutations in a number of transcription factors involved in the growth and maturation of pancreatic β-cells. Additionally, early-onset diabetes is caused due to abnormalities in enzymes related to β -cell glucose sensing. (2)

Because MODY is often mistaken as both type 1 and type 2 diabetes mellitus, it is challenging to interpret actual incidence at the global level. Additionally, because it is an unusual disease, different populations have different incidence of it. (3) MODY is currently responsible for 1-5% of all instances of diabetes mellitus in all over the world (1).

For instance, the minimum incidence of this disorder is predicted at 0.4 cases per 100,000 per year among people aged under 18 in Canada (4**).** Besides its rare nature, specific communities have been found to have high frequencies, including Pima Indians, the Nauru population, and some others in southern India (5). For the European population, the prevalence has been enumerated to be 1 per 10,000 in adults and 1 per 23,000 in children (6). In contrast, the estimated incidence of MODY in children and adolescents under 15 with newly diagnosed diabetes mellitus is 2.4 percent (7**).** According to current data, the prevalence of MODY differs by country and ethnicity, which may be attributed to variations in availability and access to genetic testing facilities (3**).**

MODY families have undergone molecular genetic research, which have revealed that they are not a similar entity but rather a representation of metabolic, genetic, and clinical heterogeneity brought on by mutations in a single gene needed for the normal functioning of the pancreatic beta-cell. In addition, of all forms of diabetes incidences, monogenic diabetes is the most common (8**).**

At least 14 different MODY mutations are now recognized. GCK, HNF1A, HNF4A, HNF1B, INS, PDX1, PAX4, ABCC8, KCNJ11, KLF11, CEL, BLK, and APPL1 are some of them. Age of onset, response to treatment, and the development of extra-pancreatic manifestations are all factors that differ amongst the various genes (1).

30% to 60% of MODY develops by mutations in the hepatocyte nuclear factor 1 alpha (HNF1A) gene. Hepatocyte nuclear factor 1-alpha-HNF1A (MODY 3) mutations in the gene affect pancreatic beta cell mitochondrial metabolism as well as significant processes in glucose transport and metabolism. In addition to pancreatic tissue, HNF1A is also found in the liver, kidney, and intestine. Progressive beta-cell dysfunction is observed. These individuals have a decreased glycosuria renal threshold. Between the ages of 21 and 26 years, HNF1A is frequently diagnosed. The high penetrance of the gene deficiency causes 63% of carriers to acquire DM by age 25, 79% by age 35, and 96% by age 35 (9).

This study aimed to detect clinical characteristics in Egyptian children with MODY 3 as regards to; age of disease onset, insulin dose, family history and complications.

PATIENTS AND METHODS

This case control study was conducted in the Pediatrics Diabetes, Endocrine and Metabolic Pediatrics Unit (DEMPU) in Cairo University pediatric hospitals and pediatrics outpatient clinic, at Aswan University hospitals, and included 20 cases and 10 healthy subjects were considered as the control subjects

Patients were included when suspected to have MODY3 diabetes, aged 10-18 years, had affected parents, had two generations affected with diabetes (onset of diabetes before 35 years), absence of pancreatic islet autoantibodies (Anti GAD-Anti-islets), measurable C-peptide in presence of hyperglycemia, low insulin dose (less than 0.5 IU per kg), normal triglyceride level and normal or elevated LDL-cholesterol and normal body mass index. Patients with secondary causes of diabetes, presented with diabetes ketoacidosis, had significant obesity or acanthosis nigricans were excluded from the study.

The study instruments and protocols were approved from the Ethical Review Board Committee of the Faculty of Medicine, of Cairo and Aswan Universities. Informed consent was obtained from all caregiver included in the study.

All subjects were subjected to full history taking, assessment of socioeconomic status (SES) of the children and their families were assessed using the modified scale of Fahmy et al (10). A total score,

based on the 7 variables studied (mother's education, father's education, working status of the mother, working status of the father, per-capita income, family size, and crowding index) was calculated by summing up the discrete scores for each case into a single index, and the total score was found to range from 4- 48. Complete clinical examination including assessment of systolic and diastolic blood pressure and pubertal development was assessed by the criteria of Tanner (11) according to axillary and pubic hair, breast and testicular size and genital development and laboratory tests, including; C peptide assays, Glycosylated hemoglobin (HbA1c), Fasting and 2 hours postprandial blood sugar levels, Triglyceride, LDL cholesterol, Measurement of anti-bodies to glutamic acid decarboxylase (anti-GAD) and anti-islets antibodies.

Statistical analysis

The collected data was revised, coded, and tabulated using Statistical package for Social Science. Descriptive statistics: Mean, Standard deviation (± SD) for parametric numerical data, Frequency and percentage of non-numerical data. Analytical statistics: Student T Test was used to assess the statistical significance of the difference between the two-study group means. Chi-Square test was used to examine the relationship between two qualitative variables. Fisher's exact test: was used to examine the relationship between two qualitative variables when the expected count is less than 5 in more than 20% of cells. A p value is considered significant if <0.05 at confidence interval 95%.

RESULTS

The mean age of the studied cases and controls were 13.63±2.74 and 12.50±1.35 years respectively. The percentage of boys and girls were 45% and 55% for cases and 50%: 50% for controls. Three quarters of cases (75%) were from Upper Egypt vs. 100% of controls. Concerning maternal and paternal age, the mean age for cases were 41.20 ± 7.50 & 41.10 ± 16.24 respectively, while for controls were 39.30 ± 5.87 and 39.90 ± 15.27 respectively. More than one half of mothers had no or low education (55 % for cases and 70 % for controls). On the other hand, 40% of fathers of both case and control groups had low/ no education. The majority of mothers (80%) in the cases group were housewives while all fathers (100%) were working. Regarding control group, 60% of mothers were housewives while 90% of fathers were working. Overall, the mean crowding index was 2.26 ± 0.77 for cases and 2.17 ± 0.48 for controls. The majority of the studied families (75% of cases and 100% of controls) had insufficient per-capita income. Regarding socioeconomic standard (SES) levels, one half of families of cases scored low SES, 40% were of medium SES, while 10% of high SES. On the other hand, 60% of families of controls scored low SES, 20% were of medium SES, while 20% of high SES. Table 1

Regarding pubertal staging, 20% of cases (4 cases) were prepubertal, 80 % (16 cases) were pubertal. The mean height was154.20 ± 12.10 and, mean height SDS was - 0.03 ± 1.09, while mean weight was SDS 0.38 ± 1.0 and, mean BMI SDS 0.49 ± 1.19. Regarding sexual maturity rating, 20% (2 controls) were prepubertal, 80% (8 controls) were pubertal. The mean height was147.80 ± 7.08, the mean height SDS was -0.34 ± 0.72, while mean weight SDS was 0.03 ± 0.6 and mean BMI SDS was 0.36 ± 0.76. table 2

The mean fasting blood sugar (96.05± 17.53 mg/dl) was statistically higher in cases group compared to controls (81.20 ± 9.27 mg/dl), p=0.042. The mean postprandial blood sugar was statistically higher in cases group (151.65 ± 16.43 mg/dl) compared to controls (137.00 ± 4.69 md/dl), p<0.001. The mean HbA1c was statistically higher in cases group (8.23 ± 1.56) compared to controls (4.49 ± 0.45), p=0.001. There was no statistical difference between cases and control regarding the mean cholesterol level (mg/dl), mean TG level or mean c- peptide levels. Table 3

All cases (100%) had a positive family history of DM, and none had neonatal hypo or hyper glycaemia and none had previous DKA. All cases were negative for Anti-Gad and Anti-Islet. The mean age on

onset of DM was 11.08 ± 2.77 years, the mean duration of DM was 2.2 ± 0.16 years. 10% had neuropathy, none of cases had acanthosis nigricans or lipodystrophy. 5% of cases were treated by oral hypoglycemic drugs, 15 % were treated by insulin and oral hypoglycemic drugs, 15% were managed by healthy life style and 65% of cases treated by insulin with a mean dose 0.34 ± 0.19. Table 4

DISCUSSION

Maturity-onset diabetes of the young (MODY) is a rare form of monogenic diabetes. To date, multiple distinct forms of MODY (MODY1-14) have been identified with varied clinical and genetic heterogeneity. MODY frequently represents a diagnostic challenge for clinicians as it is a rare condition that shares clinical features with both type 1 and type 2 diabetes mellitus. The genetic basis of MODY in the middle eastern population is unknown (12). In this study we aim to detect the clinical characteristic of suspected MODY3 cases as regards to; age of diabetes onset, family history, insulin dose and complications in Egyptians children and select suspected cases of MODY3 for DNA sequencing of HNF1A for better management, better outcome and family counseling.

The cases group included 9 males and 11 females, this was in line with the findings of *Katashima et al.* (13) in Japan, who enrolled 45 (17 males and 28 females) unrelated diabetic persons who were clinically diagnosed as cases of MODY. They investigated the presence of HNF4 and GCK genetic variants in juvenile study population with onset of DM before the age of seventeen years old. In contrast to the work of *Trhanint et al.* (14), who evaluated young adults with diabetes at the genetic and clinical levels in Moroccan households. Twenty patients were included in the analysis, with men making up 60% of the group.

In the current study, the mean age was 13.63±2.74 years, this was in the same way with *Al-Kandari et al.,* (15), who conducted research on mutations among patients diagnosed with MODY in a nation where DM is an epidemic disease**.** Similarly, in the study by *Katashima et al.* (13), the mean age at the time of diagnosis was 10.5±3.3 years. In contrast to *Trhanint et al.* (14), who noted that the individuals' ages ranged anywhere from 13 to 19 years on average (range: 5–31 years old).

In the present study, the mean height SDS was - 0.03 ± 1.09. While the mean weight SDS was 0.38 ± 1.07, the mean BMI was 21.52 ± 2.06. Tanner staging was prepubertal in 4 cases (20%) and pubertal in 16 cases (80%). Our results were in agreement with *Haliloglu et al.,* (16)**,** who reported that the mean BMI was 17·2 ± 1·9 kg/m2 (14·5–21·2 kg/m^2), and all participants had normal body weight, except for one patient who was overweight. While in the study by *Trhanint et al.* (14), the average BMI was 21.24 kg/M^2 (range,13–30 kg/M^2).

In the current study, the mean fasting blood sugar (96.05± 17.53 mg/dl) was statistically higher in cases group. The mean postprandial blood sugar was 151.65 ± 16.43 mg/dl. The mean HbA1c was 8.23 ± 1.56. There was no statistical difference between cases and control regarding the mean cholesterol level (mg/dl), mean TG level or mean c- peptide levels. In accordance with, *Al-Kandari et al.,* (15), the mean HbA1c for the index patients was 8.26% (SD ± 1.73).

According to the findings of the research conducted by *Haliloglu et al.,* (16)**,** the average levels of glucose in the blood during fasting (FBG) and after two hours of testing were 123 14 mg/dl (107–157 mg/dl) and 181 30 mg/dl (136–247 mg/dl), respectively. At the time of presentation, the patient's HbA1c level was 5.9–7.6%, with a standard deviation of 0.05% (47.5 mmol/mol).

In the current study, all cases (100%) had a positive family history of DM. Our results were in agreement with *Katashima et al.* (13) who reported that the incidence of a family history of diabetes in children with MODY was 100%, and 73.3% (33/45) spanned three generations. Similarly, *Corrales et al.,* (17) reported that all patients with MODY 2 & MODY 3 had family history of DM.

However, *Zhao et al., (18)* who studied clinical Characteristics of Patients With HNF1-alpha MODY, reported that among the patients identified, 89.8% (95% CI: 54.1–98.5) had a family history of diabetes, and *Zubkova et al.*, (19)**,** included 312 patients (162 boys and 150 girls) aged 3 months to 25 years with suspected MODY, Twenty mutations were detected in the HNF1A gene (MODY3) in 19 (6.1%) probands. A hereditary history of DM was present in 15 (78.9%) families.

In the current study, none of cases had neonatal hypo or hyper glycaemia. None of patients had previous

DKA. Similarly, *Johansson et al.,* (20)**,** reported that patients with MODY 3 presents without history of ketosis. While in the study by *Zubkova et al.,* (19)**,** At the disease onset, Three (15.8%) patients had classical signs of diabetes and high hyperglycemia had ketosis in onset. Ketosis is generally believed to be untypical of monogenic forms of DM. However, several studies (in particular, the first description of MODY3 in Russia have demonstrated that the presence of ketosis in onset does not exclude MODY.

In the current study, all cases were negative for Anti-Gad and Anti-Islet. In the same way, *McDonald et al.,* (21)**,** observed that GAD and/or IA-2 antibodies were present in 80/98 (82%) patients with Type 1 diabetes and 5/508 (< 1%) patients with maturity-onset diabetes of the young. In the current study, the mean age on onset of DM was 11.08 ± 2.77 years, the mean duration of DM2.2 ± 0.16 years.

In the same way, *Al-Kandari et al.,* (15), who studied identification of Maturity-Onset-Diabetes of the Young (MODY) mutations in a Kuwait, reported that the mean age at diagnosis of diabetes was 10.02 (SD ± 5.85) years. Similarly, *Zubkova et al.,* (19)**,** reported that the median age of DM diagnosis in patients with MODY 3 was 10.6 years (0.1; 15 years). However, *Zhao et al., (18),* reported that age of DM diagnosis in their study was 20.3 years (95% CI: 18.3–22.2).

In the current study 10% had neuropathy, none of cases had acanthosis nigricans, or lipodystrophy. This was comparable with *Bhat et al.,* (22)**,** who reported that a considerable number of MODY patients in their study were found to have microvascular complications. Diabetic peripheral neuropathy was the most frequent complication observed in 14% of patients which was unrelated to duration of DM. Diabetic retinopathy and nephropathy was observed in 1.7% and 1.5% patients respectively.

However, Zhao et al. (18) found that of the patients, 47.6% (95% CI: 30.6-65.2; 21.5% (95% CI: 14.5-30.8) had diabetic retinopathy, 16.6% (95% CI: 10.3-25.5) had diabetic kidney disease, 11.8% (95% CI: 6.2-21.2) had diabetic neuropathy, and 11.1% (95% CI: 7.3-16.6) had macrovascular complications.

In the current study, 5% of cases were treated by oral hypoglycemic, 15 % were treated by insulin and oral hypoglycemic drugs, 15% were managed by healthy lifestyle and 65% of cases treated by insulin with a mean dose 0.34 ± 0.19. Our results were comparable with *Johansson et al.,* (20)**,** reported that 10/25 of patients were treated by Insulin, 7/25 were treated by Sulfonylurea and 5/25 were treated by diet only. While in the study by *Zubkova et al.,* (19)**,** they reported that at the disease onset, 8 (42.1%) patients started Insulin at a dose of 0.6 U/kg/day (0.06; 3); of these, the highest doses (1.1 and 3 U/kg/day) were used in two young children who manifested with a typical clinical picture of diabetes and high hyperglycemia; during treatment, insulin was discontinued in one patient because of hypoglycemia.

Metformin at a dose of 500—1,000 mg/day was prescribed to 4 (21.1%) patients; a diet was recommended to 7 (36.8%) patients. By the time of molecular genetic testing, 9 patients received Insulin at a dose of 0.48 U/kg/day (0.2; 1.2); 5 patients received Metformin at a dose of 500—2,000 mg/day; 5 patients had no treatment. After molecular genetic confirmation of the diagnosis, 7 patients receiving Insulin and 5 patients receiving Metformin were successfully switched to pathogenetic therapy with sulfonylurea (SU) drugs: 4 patients received Glibenclamide at a dose of 5.25—7.5 mg/day, and 8 patients received Gliclazide at a dose of 30—60 mg/day. Insulin was continued in 2 patients with an early diagnosis of diabetes due to high insulin requirements (1.1—1.2 U/kg/day) and a low level of endogenous insulin (19)**.**

In addition, *Corrales et al.,* (17) reported that 50% of patients were treated by oral antidiabetics, 30% were treated with Insulin and oral antidiabetics, 20% of patients were treated with insulin only, while none of patients were treated with diet only.

And according to Zhao et al.'s study (18), 17.0% (95% CI: 13.2-21.6) of patients received lifestyle counselling, 40.3% (95% CI: 32.4-48.6) received medicines for oral hypoglycemic medications, 35.5% (95% CI: 31.3-40.0) received prescriptions for insulin, and 9.5% (95% CI: 5.4-16.2) received prescriptions for oral hypoglycemic medications along with insulin.

CONCLUSION

MODY3 is more frequent than previously thought when less stringent criteria are used to select patients with diabetes for gene screening. An early diagnosis is essential for starting the best course of therapy,

provide adequate genetics counselling, and make it easier to identify monogenic diabetes in family members who are asymptomatic. Our findings suggest that a thorough clinical evaluation, including a family history, phenotypic features, a response to diabetes-specific autoantibodies, and an assessment of endogenous insulin secretion, may be a more effective first step in identifying diabetic children who require genetic testing for MODY.

Conflict of interest:

The authors declare no conflict of interest.

Funding:

The authors declare that this research work did not receive any fund.

Acknowledgement:

Authors would like to thank all patients and their family members for their valuable contributions to the study.

REFERENCES

1. **Hoffman LS, Fox TJ, Anastasopoulou C, Jialal I(2021):** Maturity Onset Diabetes in the Young. In: StatPearls [Internet]. StatPearls Publishing.
2. **Urakami T (2019):** Maturity-onset diabetes of the young (MODY). current perspectives on diagnosis and treatment. Diabetes, Metab Syndr Obes targets Ther ;12:1047.
3. **Behl R, Malhotra N, Joshi V, Poojary S, Middha S, Gupta S, et al. (2022):** Meta-analysis of HNF1A-MODY3 variants among human population. J Diabetes Metab Disord;1–10.
4. **Amed S, Oram R. (2016):** Maturity-onset diabetes of the young (MODY): making the right diagnosis to optimize treatment. Can J diabetes ;40(5):449–54.
5. **Firdous P, Nissar K, Ali S, Ganai BA, Shabir U, Hassan T, et al. (2018):** Genetic testing of maturity-onset diabetes of the young current status and future perspectives. Front Endocrinol (Lausanne) ;9:253.
6. **Nkonge KM, Nkonge DK, Nkonge TN (2020):** The epidemiology, molecular pathogenesis, diagnosis, and treatment of maturity-onset diabetes of the young (MODY). Clin Diabetes Endocrinol ;6(1):1–10.
7. **Kleinberger JW, Pollin TI (2015):** Undiagnosed MODY: time for action. Curr Diab Rep ;15(12):1–11.
8. **Sanyoura M, Philipson LH, Naylor R (2018):** Monogenic diabetes in children and adolescents: recognition and treatment options. Curr Diab Rep. 2018;18(8):1–13.
9. **Wędrychowicz A, Tobór E, Wilk M, Ziółkowska-Ledwith E, Rams A, Wzorek K, et al.(2017):** Phenotype heterogeneity in glucokinase–maturity-onset diabetes of the young (GCK-MODY) patients. J Clin Res Pediatr Endocrinol ;9(3):246.
10. **Fahmy SI, Nofal LM, Shehata SF, El Kady HM, Ibrahim HK(2015):** Updating indicators for scaling the socioeconomic level of families for health research. J Egypt Public Health Assoc ;90(1):1–7.
11. **Tanner JM(1979):** Education and physical growth: implications of the study of children's growth for educational theory and practice. Adolescence ;14(54):437.
12. **Al-Kandari H, Al-Abdulrazzaq D, Davidsson L, Nizam R, Jacob S, Melhem M, et al.(2020):**

Identification of Maturity-Onset-Diabetes of the Young (MODY) mutations in a country where diabetes is endemic. Sci Rep ;11(1):1–11.

13. **Katashima R, Matsumoto M, Watanabe Y, Moritani M, Yokota I(2021):** Identification of Novel GCK and HNF4α Gene Variants in Japanese Pediatric Patients with Onset of Diabetes before 17 Years of Age. J Diabetes Res.
14. **Trhanint S, Bouguenouch L, Abourazzak S, El Ouahabi H, Latrech H, Benyakhlef S, et al. (2022)**: Molecular and clinical assessment of maturity-onset diabetes of the young revealed low mutational rate in Moroccan families. Int J Pediatr Adolesc Med ;9(2):98–103.
15. **Al-Kandari H, Al-Abdulrazzaq D, Davidsson L, Nizam R, Jacob S, Melhem M, et al. (2021)**: Identification of Maturity-Onset-Diabetes of the Young (MODY) mutations in a country where diabetes is endemic. Sci Rep ;11(1):1–11.
16. **Haliloglu B, Hysenaj G, Atay Z, Guran T, Abalı S, Turan S, et al (2016):** GCK gene mutations are a common cause of childhood-onset MODY (maturity-onset diabetes of the young) in Turkey. Clin Endocrinol (Oxf). 2016;85(3):393–9.
17. **Corrales PJP, Garrido MPL, Rodríguez SA, Rubio LL, Jiménez LML, Oliveira CL, et al (2010):** Clinical differences between patients with MODY-3, MODY-2 and type 2 diabetes mellitus with I27L polymorphism in the HNF1α gene. Endocrinol y Nutr. 2010;57(1):4–8.
18. **Zhao Q, Ding L, Yang Y, Sun J, Wang M, Li X, et al (2022):** Clinical Characteristics of Patients With HNF1-alpha MODY: A Literature Review and Retrospective Chart Review. Front Endocrinol (Lausanne) ;13.
19. **Zubkova NA, Gioeva OA, Tikhonovich Y V, Petrov VM, Vasilyev E V, Timofeev A V, et al(2018):** Clinical and molecular genetic characteristics of MODY1—3 cases in the Russian Federation as shown by NGS. Probl Endocrinol ;63(6):369–78.
20. **Johansson BB, Irgens HU, Molnes J, Sztromwasser P, Aukrust I, Juliusson PB, et al.(2017):** Targeted next-generation sequencing reveals MODY in up to 6.5% of antibody-negative diabetes cases listed in the Norwegian Childhood Diabetes Registry. Diabetologia. 2017;60:625–35.
21. **McDonald TJ, Colclough K, Brown R, Shields B, Shepherd M, Bingley P, et al.(2011):** Islet autoantibodies can discriminate maturity-onset diabetes of the young (MODY) from Type 1 diabetes. Diabet Med ;28(9):1028–33.
22. **Bhat JA, Masoodi SR, Bhat MH(2022):** Prevalence and clinical profile of maturity onset diabetes of the young among people with diabetes attending a tertiary care institute in North India. Indian J Endocrinol Metab ;26(Suppl 1):S25.

Table 1. Demographic and socioeconomic data of the studied children.

Characteristics	Cases (No. 20)		Control (No. 10)		Test of significance #
	No.	%	No.	%	
Mean children age (years)	13.63±2.74		12.50±1.35		P=0.654
Gender					
Male	9	45	5	50	P=0.796
Female	11	55	5	50	
Residence					
Upper Egypt	15	75	10	100	P=0.122
Lower Egypt	5	25	0	0	
Mean maternal age (years)	41.20 ± 7.50		39.30 ± 5.87		P=0.823
Mean paternal age (years)	41.10 ± 16.24		39.90 ± 15.27		P=0.811
Mother's education					
Low/No education	11	55	7	70	P=0.232
Educated	9	45	3	30	
Mother's working status					
Not working	16	80	6	60	P=0.243
Working	4	20	4	40	
Father's education					
Low/No education	8	40	4	40	P=1.000
Educated	12	60	6	60	
Father's working status (n=27) ♦					
Not working	0	0	0	0	-
Working	18	100	9	90	
Mean crowding index	2.26 ± 0.77		2.17 ± 0.48		P=0.834
Per-capita income					
Enough	5	25	0	0	P=0.122
Not enough with loan	15	75	10	100	
Socioeconomic standard					
Low SES	10	50	6	60	P=0.176
Medium SES	8	40	2	20	
High SES	2	10	2	20	

♦*3 fathers died (2 cases and 1 control), #; Independent sample t test was used for continuous data and chi-square test or Fischer exact test were used for categorical data,*

Table 2. Anthropometric data and pubertal staging of the studied children

Characteristics	Cases (No. 20)	%	Control (No.10)	%	Test of significance #
Tanner staging					
Prepubertal	4	20	2	20	P=1.000
Pubertal	16	80	8	80	
Mean height (cm)	154.20 ± 12.10		147.80 ± 7.08		P=0.437
Mean height (SDS)	- 0.03 ± 1.09		-0.34 ± 0.72		P=0.332
Mean weight (Kg)	51.60 ± 11.87		47.00 ± 7.59		P=0.511
Mean weight (SDS)	0.38 ± 1.07		0.03 ± 0.60		P=0.188
Mean BMI (Kg/m^2)	21.52 ± 2.06		21.12 ± 1.96		P=0.837
Mean BMI (SDS)	0.49 ± 1.19		0.36 ± 0.76		P=0.445

#; Independent sample t test was used for continuous data and chi-square test, or Fischer exact test were used for categorical data, ******* *Significant (p<0.05)*

Table 3. Laboratory data of the studied children

Characteristics	Cases		Control		Test of significance♦
	No. (20)	%	No. (10)	%	
Mean fasting blood sugar(mg/dl)	96.05± 17.53		81.20 ± 9.27		P= 0.042*
Mean post prandial blood sugar (mg/dl)	151.65 ± 16.43		137.00 ± 4.69		P<0.001*
Mean cholesterol level (mg/dl)	94.05 ± 9.84		86.20 ± 8.34		P= 0.651
Mean TG level(mg/dl)	111.65 ± 14.76		95.90 ± 10.83		P= 0.277
Mean HbA1c %	8.23 ± 1.56		4.49 ± 0.45		P= 0.001
Mean c- peptide (ng/ml)	0.85 ± 0.64		1.66 ± 0.18		P= 0.061

♦ Independent sample t test, * Significant (p<0.05)

Table 4. Clinical data of the studied cases

Characteristics	Number (N. 20)	%
Positive family history of DM	20	100
Negative history of previous DKA	20	100
Negative history of neonatal hypo/ hyperglycemia	20	100
Mean onset of DM (years)	11.08 ± 2.77	
Mean duration of DM (years)	2.2 ± 0.16	
Clinical examination		
Acanthosis nigricans	0	0
Neuropathy	2	10
Lipodystrophy	0	0
Negative Anti-Gad	20	100
Negative Anti-Islet	20	100
Management of D.M		
Insulin	13	65
Oral hypoglycemic	1	5
Insulin and oral hypoglycemic drugs	3	15
Healthy life style	3	15
Mean insulin dose	0.34 ± 0.19	