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Prevalence of Obesity and Dyslipidemia in Maturity Onset Diabetes of the Young Patients

ORIGINAL ARTICLE
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ABSTRACT

Objective: Maturity-onset diabetes of the young (MODY) is an uncommon type of diabetes distinct from type 1 and type 2 diabetes. Diagnosis often depends on clinical suspicion, and misdiagnosis may lead to inappropriate treatment. Obesity and dyslipidemia are increasing in diabetic patients, but the prevalence of obesity and dyslipidemia in this group remains unclear. This study aims to evaluate these conditions in MODY patients.

Methods: A descriptive study involving 108 MODY patients and 108 healthy controls was conducted. Clinical data were collected and analyzed, including body mass index (BMI), fasting plasma glucose, HbA1c, and lipid profile. Statistical comparisons were made between the MODY and control groups to assess the prevalence of obesity and dyslipidemia. Further subgroups, including obesity and dyslipidemia, were analyzed to explore the relationship between these metabolic conditions and clinical parameters within the MODY cohort.

Results: The study revealed a significantly higher prevalence of both obesity and dyslipidemia in MODY patients compared to controls. Specifically, 24.1% of MODY patients were found to be obese, while 72.2% exhibited dyslipidemia. Notably, obese MODY patients had elevated triglyceride levels compared to their non-obese counterparts. Additionally, it was determined that dyslipidemic MODY patients were older, had higher BMI, and exhibited poorer glycemic control. Increased age and obesity were found to be significantly associated with dyslipidemia in MODY patients, according to the regression analysis.

Conclusion: The current study demonstrates that the rate of obesity and dyslipidemia is high among MODY patients, and these conditions may negatively impact the course of the disease.

Keywords: Maturity-onset diabetes of the young, obesity, dyslipidemia

Introduction

Diabetes is increasingly acknowledged as a global public health issue, including type 1 diabetes (T1D), type 2 diabetes (T2D), gestational diabetes (GD), and rare forms of monogenic diabetes.[1] Among these monogenic forms, the most common is maturity-onset diabetes of the young (MODY).[2] Maturity-onset diabetes of the young refers to a diverse group of conditions characterized by a primary defect in beta-cell function, usually identified at a young age and inherited in an autosomal dominant fashion. Clinically, MODY presents distinct features that differentiate it from T1D and T2D. While T1D usually arises from an autoimmune process leading to insulin deficiency, T2D is often characterized by insulin resistance and is strongly associated with obesity. Maturity-onset diabetes of the young typically begins at a young age, shows autosomal dominant inheritance, is linked to similar family histories, and is related to genetic defects in beta-cell function rather than insulin resistance.[1,2] These characteristics enable the distinction of MODY from T1D and T2D and play a critical role in determining treatment strategies.

Obesity and dyslipidemia are significant metabolic disorders that can be both causes and consequences of diabetes.[3] Obesity is broadly acknowledged as a major risk factor for the onset of T2D, whereas dyslipidemia is recognized as a critical contributor to cardiovascular diseases.[3,4] However, it remains unclear whether the prevalence of obesity and dyslipidemia in MODY patients differs from that in T1D or T2D. Current literature suggests that different genetic subtypes of MODY may exhibit distinct metabolic profiles.[5-7] Patients with MODY 2 are often asymptomatic and are most commonly identified through routine screening during pregnancy. Maturity-onset diabetes of the young 2 constitutes approximately 2-6% of patients with GD and can be differentiated by clinical characteristics and fasting blood

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glucose (FBG) measurements.[8] Carriers of MODY 3 are typically lean and not prone to weight gain, and they may also have a lower risk of dyslipidemia.[7] In contrast, individuals with MODY 5 may display dyslipidemia, marked by low high-density lipoprotein cholesterol (HDL-C) levels and elevated triglyceride (TG) concentrations.[6] The rarity of MODY and the diversity of its genetic subtypes underscore the need for more comprehensive and homogeneous studies in this area. While some studies suggest that obesity and dyslipidemia may be more prevalent in MODY patients than previously anticipated, additional data are needed to generalize these findings.[9,10] In particular, the progression of obesity and dyslipidemia in MODY patients, as well as their impact on the long-term risk of complications and treatment responses, remain crucial questions that require further investigation.

The current study aims to determine the prevalence of obesity and dyslipidemia in MODY patients. Additionally, it seeks to expand the existing literature on the metabolic profiles of MODY patients and provide significant insights for clinical practice. The findings are anticipated to aid in developing new approaches for the accurate diagnosis and management of MODY.

Material and Methods

Study Design and Patient Selection

The study included 108 patients diagnosed with MODY and 108 healthy controls at the Department of Endocrinology and Metabolism in our tertiary center between February 2015 and October 2020. The descriptive study was a retrospective analysis comparing healthy control individuals and MODY patients. The study protocol received approval from the Ethics Committee of our institution on March 8, 2021, under decision number 106/25. The research was conducted in accordance with the Declaration of Helsinki. The participants provided written informed consent for the study. In our center, a risk assessment is performed for all diabetic patients suspected of having MODY using the MODY Probability Calculator, which is freely accessible at http://diabetesgenes.org/content/mody-probability-calculator.[11] Patients identified as high-risk based on the MODY Probability Calculator undergo genetic testing and other diagnostic evaluations for MODY. The diagnosis of MODY was made in patients with early-onset diabetes, a family history of autosomal dominant inheritance, negative pancreatic islet autoantibodies, and mutations in specific genes detected through genetic testing. Patients with T1D and T2D, those who were pregnant or breastfeeding, patients without biochemical parameters, and those using medications that could affect lipid levels were excluded from the study. The control group was selected from healthy individuals matched with MODY patients in terms of age and gender, who had no family history of the disease, visited our hospital for check-ups, and were disease-free upon examination. It was indicated that none of the patients in the study cohort were smokers, had suffered any cardiovascular events, or were taking any lipid-lowering therapies.

Clinical and Biochemical Measurements

Patient records were examined retrospectively to collect information on age, body mass index (BMI), gender, and duration of diabetes. Data on HbA1c, FPG, total cholesterol (TC), HDL-C, low-density lipoprotein cholesterol (LDL-C), and TG levels recorded at the last clinical appointment were retrieved from the medical files. Measurements were performed in our center's laboratory using a biochemical analyzer. HbA1c levels were determined via high-performance liquid chromatography. Lipid profiles were assessed through enzymatic colorimetric assays using spectrophotometry. Dyslipidemia was defined according to guidelines from the American Association of Clinical Endocrinologists and the American College of Cardiology as TC ≥ 200 mg/dL, LDL-C ≥ 100 mg/dL, non-HDL cholesterol ≥ 130 mg/dL, and TG ≥ 150 mg/dL. Dyslipidemia was considered if one or more of these lipid levels were abnormal.[12,13]

Statistical analysis

Statistical Package for Social Sciences (SPSS) software version 28.0, (IBM SPSS Corp.; Armonk, NY, USA) was used to analyze the collected data statistically. Continuous data were initially assessed with the Kolmogorov–Smirnov test to determine normality. Descriptive statistics were presented as mean ± standard deviation for data with a normal distribution. For continuous variables that were not normally distributed, the results were presented as median (range). Participants were divided into 2 groups: MODY patients and a control group. Clinical data, glycemic parameters, and lipid profiles were compared between the 2 groups. Subsequently, MODY patients were subdivided based on the presence of obesity and dyslipidemia. The relationship between these subgroups and lipid profiles and glycemic parameters was analyzed. The Mann–Whitney *U* test was utilized for the analysis of non-normally distributed variables. An independent 2-sample *t*-test was employed to evaluate normally distributed continuous variables. The Chi-square test was used to analyze differences in categorical variables. Univariate and multivariate regression analyses were conducted to identify the factors influencing the development of dyslipidemia in patients with MODY. Initially, a univariate regression analysis was conducted to ascertain the variables impacting dyslipidemia in MODY patients. Subsequently, a multivariate regression analysis was conducted using the variables identified as risk factors for dyslipidemia in the univariate regression analyses. A *P*-value of less than .05 was deemed statistically significant.

Results

The study included 108 patients diagnosed with MODY and 108 healthy adults. Among the MODY patients, 56 (51.9%) were female, with a mean age of 31.40 ± 9.85 years. In the control group, 67 (62.0%) were female, and 31 (38.0%) were male, with a mean age of 32.07 ± 8.91 years. Complete clinical data of the study population are presented in Table 1. The median duration of diabetes was 4 years (range 1-24 years). The mean C-peptide level of MODY patients was 2.42 ± 1.46 ng/mL. The classification of participants according to BMI is shown in Figure 1. The prevalence of obesity in MODY patients was 24.1%, compared to 13.0% in the control group (*P* = .036). The prevalence of dyslipidemia was found to be 72.2% in the MODY group and

MAIN POINTS

- The prevalence of obesity in maturity-onset diabetes of the young (MODY) patients was 24.1%.
- The prevalence of dyslipidemia was found to be 72.2% in MODY patients.
- The most common lipid abnormality was high low-density lipoprotein cholesterol (LDL-C) in MODY patients, which was statistically similar to that in the control group.
- Increased age and obesity were significantly associated with dyslipidemia in MODY patients.

Table 1. The Complete Demographic and Clinical Data of the Patients and Control Subjects

	MODY (n = 108)	Control (n = 108)	*P*
Age (year)	31.40 ± 9.85	32.07 ± 8.91	.601
Gender (F)	56 (51.9%)	67 (62.0%)	.131
BMI (kg/m²)	27.54 ± 6.16	26.20 ± 4.68	.073
Obesity (%)	26 (24.1)	14 (13.0)	**.036**
FPG (mg/dL)	164.18 ± 81.97	89.44 ± 8.23	**< .001**
HbA1c (%)	8.83 ± 2.02	5.35 ± 0.40	**< .001**
Total cholesterol (mg/dL)	185 (123-1075)	166.3 (96-275)	**.017**
Triglyceride (mg/dL)	149 (33-1108)	94.5 (27-564)	**< .001**
LDL cholesterol (mg/dL)	111.64 ± 37.69	103.57 ± 31.21	.073
HDL cholesterol (mg/dL)	44.06 ± 15.89	48.82 ± 13.39	**.018**
Non-HDL cholesterol (mg/dL)	139.5 (51-1055)	121 (40-233)	**.001**
Dyslipidemia (%)	78 (72.2)	63 (58.3)	**.032**

BMI, body mass index; F, female; FPG, fasting plasma glucose; HDL, high-density lipoprotein; LDL, low-density lipoprotein; MODY, maturity-onset diabetes of the young.
The values in bold indicate statistically significant.

58.3% in the control group (*P* = .032). Triglyceride, LDL-C, and non-HDL cholesterol levels were significantly higher in the MODY group compared to the control group. The analysis of participants' lipid profiles is shown in Figure 2. Among MODY patients, the prevalence of hypercholesterolemia was 34.3%, hypertriglyceridemia 49.1%, high LDL-C 61.1%, and high non-HDL cholesterol 59.3%. The most common lipid abnormality was high LDL-C in both groups. The rate of high non-HDL cholesterol and hypertriglyceridemia was significantly higher in MODY patients.

When comparing obese and non-obese individuals among MODY patients, it was found that the duration of diabetes was higher in

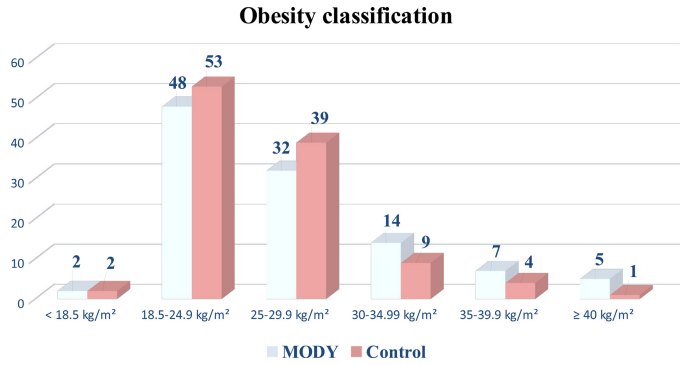

Figure 1. Classification of participants according to body mass index.

obese patients than in non-obese patients. The rate of dyslipidemia reached a high value of 92.3% in obese individuals, whereas it was 65.9% in non-obese individuals (Table 2). Triglyceride levels were also significantly higher in obese patients (*P* = .046), indicating that obesity is closely associated with the development of dyslipidemia in MODY patients.

Whenever MODY patients were categorized into 2 groups according to the presence of dyslipidemia, it was found that patients with dyslipidemia were older, had a higher BMI, longer duration of diabetes, and a worse glycemic profile (Table 3). According to the regression analysis, increased age and obesity were significantly associated with dyslipidemia in MODY patients (Table 4).

Discussion

This study demonstrates that the prevalence of obesity and dyslipidemia is high in MODY patients, and this condition may negatively impact the course of the disease. Additionally, it was found that TC, TG, and non-HDL cholesterol levels were significantly higher in the

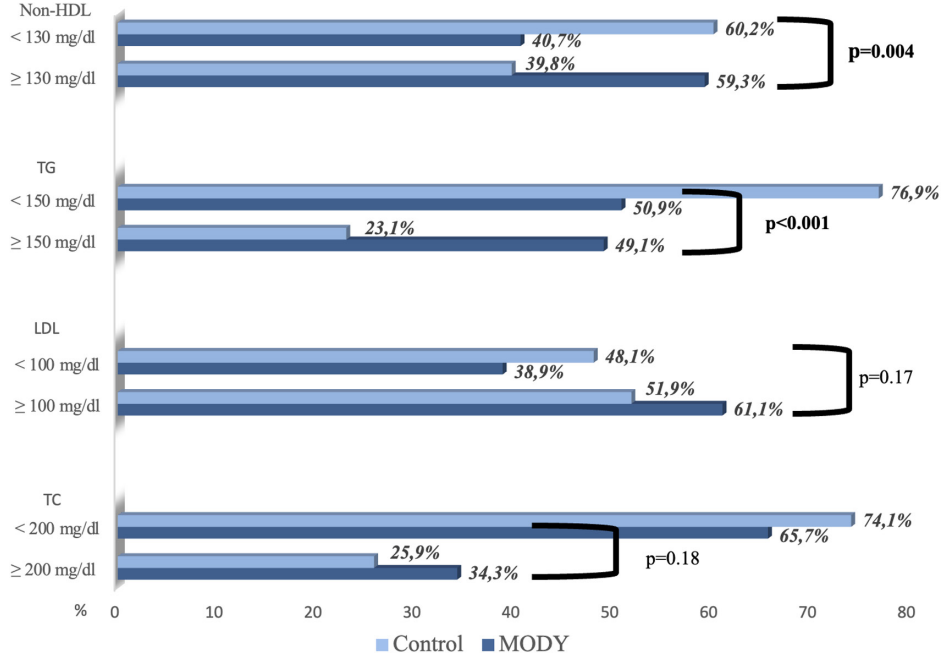

Figure 2. Classification of participants according to lipid profile (HDL, high-density lipoprotein; TG, triglyceride; LDL, low-density lipoprotein; TC, total cholesterol).

Table 2. Comparative Analysis of Demographic and Clinical Characteristics Among Obese and Non-Obese Groups in Patients with MODY

	Non-Obese (n = 82)	Obese (n = 26)	*P*
Age (year)	30.70 ± 9.40	33.65 ± 11.05	.183
Gender (F)	39 (47.6%)	17 (65.4%)	.113
BMI (kg/m^2)	24.42 ± 3.36	35.22 ± 4.51	< **.001**
Diabetes duration (year)	3 (1-24)	7 (1-20)	**.006**
Treatment Diet (%) OAD (%) Insulin (%)	 11 (13.4) 32 (39.0) 39 (47.6)	 3 (11.5) 9 (34.7) 14 (53.8)	.855
FPG (mg/dL)	156.31 ± 80.22	188.13 ± 84.39	.085
HbA1c (%)	8.81 ± 2.10	8.87 ± 1.84	.896
Total cholesterol (mg/dL)	184 (123-1075)	187 (140-259)	.664
Triglyceride (mg/dL)	140 (33-1108)	217.5 (60-504)	**.046**
LDL cholesterol (mg/dL)	111.26 ± 41.44	112.85 ± 22.88	.945
HDL cholesterol (mg/dL)	44.97 ± 17.27	41.06 ± 9.83	.275
Non-HDL cholesterol (mg/dL)	139 (51-1055)	140 (101-223)	.484
Dyslipidemia (%)	54 (65.9)	24 (92.3)	**.009**

BMI, body mass index; F, female; FPG, fasting plasma glucose; HDL, high-density lipoprotein; LDL, low-density lipoprotein; OAD, oral antidiabetic drug.
The values in bold indicate statistically significant.

MODY group compared to the control group. These findings indicate that lipid metabolism is impaired in MODY patients and that there may be an increased risk of cardiovascular diseases. Subgroup analysis based on obesity status revealed that the dyslipidemia rate reached a high value of 92.3% in obese MODY patients. These results suggest a strong association between obesity and the development of dyslipidemia in MODY patients.

Unlike patients with T2D, obesity is less commonly observed in MODY patients. However, the increasing prevalence of obesity in recent years is also affecting the phenotype of MODY patients. According

Table 3. Comparative Analysis of Demographic and Clinical Characteristics Among Dyslipidemic and Non-Dyslipidemic Groups in Patients with MODY

	Non-Dyslipidemia (n = 30)	Dyslipidemia (n = 78)	*P*
Age (year)	26.13 ± 6.12	34.02 ± 9.36	< **.001**
Gender (F)	17 (56.7%)	39 (50.0%)	.535
BMI (kg/m^2)	24.01 ± 3.19	28.91 ± 6.86	< **.001**
Obesity (%)	2 (6.7)	24 (30.8)	**.009**
Diabetes duration (year)	2 (1-18)	5.5 (1-20)	**.003**
Treatment Diet (%) OAD (%) Insulin (%)	 5 (16.7) 15 (50.0) 10 (33.3)	 9 (11.5) 27 (34.6) 42 (53.9)	.161
FPG (mg/dL)	139.68 ± 81.44	175.85 ± 78.89	**.037**
HbA1c (%)	7.63 ± 1.50	9.18 ± 1.96	< **.001**

BMI, body mass index; F, female; FPG, fasting plasma glucose; OAD, oral antidiabetic drug.
The values in bold indicate statistically significant.

Table 4. Results of the Logistic Regression Analysis Performed on MODY Patients for the Presence of Dyslipidemia

	Univariate Analysis		Multivariate Analysis	
	OR (95% CI)	*P*	OR (95% CI)	*P*
Age (year)	1.15 (1.09-1.21)	< **.001**	1.16 (1.02-1.32)	**.021**
Obesity (%)	7.22 (2.12-24.63)	**.002**	10.43 (1.04-104.54)	**.046**
Diabetes duration (year)	1.17 (1.03-1.34)	**.018**	1.02 (0.87-1.20)	.798
FPG (mg/dL)	1.01 (1.00-1.02)	**.010**	0.99 (0.99-1.01)	.557
HbA1c (%)	1.52 (1.22-1.91)	< **.001**	1.34 (0.85-2.11)	.208

CI, confidence interval; FPG, fasting plasma glucose; OR, odds ratio.
The values in bold indicate statistically significant.

to data from the World Health Organization, adult obesity has more than doubled worldwide since 1990, and adolescent obesity has increased 4-fold. In 2022, it was reported that 43.0% of adults aged 18 and older were overweight, and 16.0% were obese.[14] The impact of this rising obesity prevalence has been reflected in recent MODY studies.[6,10,15] Although obesity is more frequently observed in MODY 4, MODY 6, MODY 11, and MODY 14 patients, the rate of obesity is increasing in other MODY types as well.[16,17] Some MODY patients, especially those belonging to ethnic groups with a high prevalence of obesity (e.g., Hispanics), may become overweight or obese as a result of their dietary habits and sedentary lifestyles.[18] Bhat and colleagues evaluated 66 MODY patients and reported that 27% of them were overweight or obese.[10] In a study conducted in Qatar by Elashi and colleagues, it was shown that 83.2% of MODY patients were overweight or obese.[18] In a study by Kleinberger and colleagues involving 488 overweight and obese youth with T2D, 4.5% of the patients were found to have MODY.[9] The increasing prevalence of obesity in children and young adults in recent years is making it clinically more challenging to distinguish MODY from T2D.

Given that the guidelines of the International Society for Pediatric and Adolescent Diabetes suggest considering a MODY diagnosis in clinical cases lacking the phenotypic traits of T2D, the rising prevalence of obesity may result in fewer individuals with MODY being evaluated through genetic testing.[19] However, distinguishing between obese MODY patients and those with T2D is challenging phenotypically and can only be differentiated through genetic studies. Our findings are consistent with recent studies, confirming the high number of overweight and obese patients among those with MODY. This highlights the importance of genetic testing for MODY in young diabetic patients at high risk for MODY.

Studies evaluating lipid profiles in MODY patients show variability in dyslipidemia rates depending on MODY type, country of the study, and the number of obese patients included.[20-23] A study conducted in the UK found that HNF1A-MODY patients displayed lipid profiles that were similar to those of non-diabetic controls.[22] A study in Russia reported a dyslipidemia prevalence of 27% among non-obese MODY patients with a short duration of diabetes.[20] In a study conducted in Türkiye, TG levels were higher and HDL-C levels lower in MODY patients compared to the control group.[21] Research in Poland reported that LDL-C levels in Glucokinase (GCK)-MODY patients were similar to those in the control group, but very low-density lipoprotein (VLDL) levels were lower than those in the control group.[23] Although low HDL-C and high TG levels are not typically expected

in MODY patients, recent changes in dietary habits, sedentary lifestyles, and low exercise adherence may have led to increased obesity prevalence among MODY patients. This, in turn, could result in lower HDL-C levels and higher TG levels than anticipated. Obesity is a significant risk factor for dyslipidemia, associated with increased TG levels and decreased HDL-C levels.[24] This condition may increase the risk of early-onset atherosclerosis and other cardiovascular complications in MODY patients. This study is the first to investigate the prevalence of obesity and dyslipidemia across all MODY types rather than focusing on a specific type. Our findings indicate that patients diagnosed with MODY should be regularly monitored for dyslipidemia, and preventing or treating dyslipidemia should become a higher priority for this patient group.

Dyslipidemia results from the interplay of both genetic predispositions and environmental influences. Recent studies reported that the risk of developing dyslipidemia is higher in diabetic patients aged ≥ 30 years, in those who are obese, and in those with elevated blood glucose levels.[25,26] High TG levels increase lipolysis in adipose tissue, raising free fatty acid levels. Free fatty acids disrupt insulin signaling and impair glucose uptake by cells.[27] Moreover, elevated TG may contribute to insulin resistance.[28] Low levels of HDL-C are linked to diminished anti-inflammatory activity, heightened insulin resistance, and compromised β-cell function.[28,29] High LDL-C and TC levels can lead to endothelial dysfunction, further disrupting insulin signaling pathways.[30,31] Furthermore, glycemic fluctuations may be more frequent in dyslipidemic patients.[32] All these factors combined can negatively impact glycemic control. Similar to previous studies, we found that glycemic control was worse in dyslipidemic patients.[26,28,32] Additionally, obesity and age were identified as contributing factors to the development of dyslipidemia in MODY patients in the present study. Improving lipid metabolism may aid in better diabetes management; when the lipid profile of a diabetic patient returns to normal, less antidiabetic therapy may be required.

There are several limitations to this study that need to be addressed. First, the study design was based on a single-center observational cohort. Second, the sample size was relatively small and was limited to specific MODY subtypes prevalent in Türkiye. Another limitation is the inability to examine the association between dyslipidemia and obesity with microvascular complications due to a lack of data. The sedentary lifestyle, physical activity status, and dietary habits that could contribute to obesity could not be assessed. Finally, the study did not evaluate lipid parameters such as VLDL, intermediate-density lipoprotein, and lipoprotein(a).

In conclusion, this study demonstrates that the prevalence of obesity and dyslipidemia is high among MODY patients, and this condition may negatively impact the course of the disease. Obesity and dyslipidemia should be closely monitored and treated in the management of MODY patients. Although current challenges like cost and limited access hinder genetic testing for MODY in large populations, it should still be considered in the differential diagnosis, particularly in young diabetic patients with a family history, regardless of obesity. Clinicians should evaluate the necessity of genetic testing for patients at high risk for MODY. Additionally, further prospective multicenter studies involving more participants are needed to better understand cardiovascular risk factors and manage dyslipidemia in this patient group.

Availability of Data and Materials: The data that support the findings of this study are available on request from the corresponding author.

Ethics Committee Approval: This study was approved by the Ethics Committee of Dışkapı Yıldırım Beyazıt Training and Research Hospital, University of Health Sciences. (Approval number: 106-25; date: March 8, 2021).

Informed Consent: Written informed consent was obtained from the patients who agreed to take part in the study.

Peer-review: Externally peer reviewed.

Author Contributions: Concept – M.C.; Design – M.C., I.O.U.; Supervision – I.O.U., M.O.; Materials – M.C.; Data Collection and/or Processing – M.C., I.O.U., M.E.S., D.S., M.O.; Analysis and/or Interpretation – M.C.; Literature Search – M.C.; Writing – M.C.; Critical Review – E.C.

Declaration of Interests: The authors have no conflict of interest to declare.

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References

1. American Diabetes Association Professional Practice Committee. 2. Diagnosis and classification of diabetes: standards of care in Diabetes-2024. *Diabetes Care*. 2024;47(suppl 1):S20-S42. [CrossRef]
2. Salguero MV, Arosemena M, Pollin T, et al. Monogenic forms of diabetes. 2023 Dec 20. In: Lawrence JM, Casagrande SS, Herman WH, Wexler DJ, Cefalu WT, eds. *Diabetes in America [Internet]*. Bethesda (MD): National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK); 2023. PMID: 38117925.
3. Feingold KR. Obesity and dyslipidemia. In: Feingold KR, Anawalt B, Blackman MR, et al., eds. *Endotext*. South Dartmouth (MA): MDText.com, Inc; 2000. Available at: http://www.ncbi.nlm.nih.gov/books/NBK305895/. Accessed August 31, 2024.
4. Powell-Wiley TM, Poirier P, Burke LE, et al. Obesity and cardiovascular disease: a scientific statement from the American Heart Association. *Circulation*. 2021;143(21):e984-e1010. [CrossRef]
5. Amed S, Oram R. Maturity-Onset Diabetes of the Young (MODY): making the right diagnosis to optimize treatment. *Can J Diabetes*. 2016;40(5):449-454. [CrossRef]
6. Urakami T. Maturity-onset diabetes of the young (MODY): current perspectives on diagnosis and treatment. *Diabetes Metab Syndr Obes*. 2019;12:1047-1056. [CrossRef]
7. Hoffman LS, Fox TJ, Anastasopoulou C, Jialal I. Maturity onset diabetes in the young. In: *StatPearls*. Treasure Island, FL: StatPearls Publishing; 2024. Available at: http://www.ncbi.nlm.nih.gov/books/NBK532900/. Accessed August 31, 2024.
8. Rudland VL, Hinchcliffe M, Pinner J, et al. Identifying glucokinase monogenic diabetes in a multiethnic gestational diabetes mellitus cohort: new pregnancy screening criteria and utility of HbA1c. *Diabetes Care*. 2016;39(1):50-52. [CrossRef]
9. Kleinberger JW, Copeland KC, Gandica RG, et al. Monogenic diabetes in overweight and obese youth diagnosed with type 2 diabetes: the TODAY clinical trial. *Genet Med*. 2018;20(6):583-590. [CrossRef]
10. Bhat JA, Bhat MH, Masoodi SR, et al. Prevalence and clinical profile of maturity onset diabetes of the young among people with diabetes attending a tertiary Care Centre. *Indian J Endocrinol Metab*. 2022;26(6):543-550. [CrossRef]
11. Exeter diabetes app. Available at: https://www.diabetesgenes.org/exeter-diabetes-app/. Accessed August 31, 2024.
12. Grundy SM, Stone NJ, Bailey AL, et al. AHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA guideline on the management of blood cholesterol: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice guidelines. *J Am Coll Cardiol*. 2019;73(24):e285-e350. [CrossRef]
13. Jellinger PS, Handelsman Y, Rosenblit PD, et al. American Association of Clinical Endocrinologists and American college of endocrinology

guidelines for management of dyslipidemia and prevention of cardiovascular disease. *Endocr Pract*. 2017;23(suppl 2):1-87. [CrossRef]
14. Obesity and overweight. Available at: https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight. Accessed August 31, 2024.
15. Al-Kandari H, Al-Abdulrazzaq D, Davidsson L, et al. Identification of Maturity-Onset-Diabetes of the Young (MODY) mutations in a country where diabetes is endemic. *Sci Rep*. 2021;11(1):16060. [CrossRef]
16. Naylor R, Knight Johnson A, del Gaudio D. Maturity-onset diabetes of the young overview. In: Adam MP, Feldman J, Mirzaa GM, et al. eds. *GeneReviews®*. Seattle: University of Washington, 1993. Available at: http://www.ncbi.nlm.nih.gov/books/NBK500456/. Accessed August 31, 2024.
17. Delvecchio M, Pastore C, Giordano P. Treatment options for MODY patients: a systematic review of literature. *Diabetes Ther*. 2020;11(8):1667-1685. [CrossRef]
18. Elashi AA, Toor SM, Diboun I, et al. The genetic spectrum of Maturity-Onset Diabetes of the Young (MODY) in Qatar, a population-based study. *Int J Mol Sci*. 2022;24(1):130. [CrossRef]
19. Hattersley AT, Greeley SAW, Polak M, et al. ISPAD Clinical Practice Consensus Guidelines 2018: the diagnosis and management of monogenic diabetes in children and adolescents. *Pediatr Diabetes*. 2018;19(suppl 27):47-63. [CrossRef]
20. Ovsyannikova A, Rymar O, Shakhtshneider E, Voevoda M. The prevalence of dyslipidemia in patients with different types of MODY diabetes. *J Diabetes Metab*. [CrossRef]
21. Kanca Demirci D, Yılmaz Aydoğan H, Gül N, Tütüncü Y, vd. HNF1A Geni rs1169288 (A>C) Mutasyonunun MODY3 üzerine Etkisinin Araştırılması. *Haliç Univ Fen Bilimleri Derg*. 2018;1(1):21-32.
22. McDonald TJ, McEneny J, Pearson ER, et al. Lipoprotein composition in HNF1A-MODY: differentiating between HNF1A-MODY and type 2 diabetes. *Clin Chim Acta*. 2012;413(9-10):927-932. [CrossRef]
23. Fendler W, Rizzo M, Borowiec M, et al. Less but better: cardioprotective lipid profile of patients with GCK-MODY despite lower HDL cholesterol level. *Acta Diabetol*. 2014;51(4):625-632. [CrossRef]
24. Liu LY, Aimaiti X, Zheng YY, et al. Epidemic trends of dyslipidemia in young adults: a real-world study including more than 20,000 samples. *Lipids Health Dis*. 2023;22(1):108. [CrossRef]
25. Haile K, Timerga A. Dyslipidemia and its associated risk factors among adult Type-2 diabetic patients at Jimma University Medical Center, Jimma, Southwest Ethiopia. *Diabetes Metab Syndr Obes*. 2020;13:4589-4597. [CrossRef]
26. Peng J, Zhao F, Yang X, et al. Association between dyslipidemia and risk of type 2 diabetes mellitus in middle-aged and older Chinese adults: a secondary analysis of a nationwide cohort. *BMJ Open*. 2021;11(5):e042821. [CrossRef]
27. Sears B, Perry M. The role of fatty acids in insulin resistance. *Lipids Health Dis*. 2015;14:121. [CrossRef]
28. Parhofer KG. Interaction between glucose and lipid metabolism: more than diabetic dyslipidemia. *Diabetes Metab J*. 2015;39(5):353-362. [CrossRef]
29. Yahya R, Jainandunsing S, Rashid M, et al. HDL associates with insulin resistance and beta-cell dysfunction in South Asian families at risk of type 2 diabetes. *J Diabetes Complications*. 2021;35(10):107993. [CrossRef]
30. Hadi HAR, Suwaidi JA. Endothelial dysfunction in diabetes mellitus. *Vasc Health Risk Manag*. 2007;3(6):853-876.
31. Higashi Y. Endothelial function in dyslipidemia: roles of LDL-cholesterol, HDL-cholesterol and triglycerides. *Cells*. 2023;12(9):1293. [CrossRef]
32. Salsa-Castelo M, Neves C, Neves JS, Carvalho D. Association of glycemic variability and time in range with lipid profile in type 1 diabetes. *Endocrine*. 2024;83(1):69-76. [CrossRef]