



Estimation of lipid profile in younger patients suffering from diabetic mellitus type 1

Estimación del perfil lipídico en pacientes más jóvenes con diabetes mellitus tipo 1

320

Ali Jasim Mhaimeed Alsultani¹ College of medicine, University of Babylon, Iraq. alialsultany81@yahoo.com

Ali Jaber Aboob Al-Mamoori² Ass. Prof. College of Medicine, University of Babylon/Iraq minaligaber@gmail.com

Mushtaq Qahtan Ali³ University of Babylon /college of medicine. Iraq, mushtaq.qahtan@uobabylon.edu.iq.

Received: 06/24/2022 Accepted: 08/19/2022 Published: 09/25/2022 DOI: <https://doi.org/10.5281/zenodo.7368769>

Abstract

Background: Dyslipidemia and hyperglycemia are metabolic anomalies ordinarily found in youthful patients with Type 1 diabetes mellitus (T1DM) and both increment the danger of cardiovascular illness, objective: to estimated lipid profile in younger patients and effected of these lipids on DMT1. **Methods:** case control study of (75) younger patients were divided in to three groups according to HbA1C, (25) younger patients with high HbA1C (> 7.5 HbA1C) suffering from DMT1, (25) younger patients with moderate HbA1C (\leq 7.5 HbA1C) and (25) younger patients as control healthy, were with age range from 10 to 19 years who attended Changing Diabetes in Children. The younger patients were visit to clinical provides. All patients and control were from the same ethnic group (Arabic). Blood samples collected from each patients; lipid profile tests made for each blood sample to estimated Cholesterol, Triglyceride, HDL, LDL, and VLDL and compared them between study groups. **Results:** HbA1C, Glycemic control between groups compared and HbA1c was significantly ($P = 0.001$) higher in dyslipidemia group. The mean fasting blood glucose was also significantly higher in dyslipidemia group ($P 0.0001$). The mean differences of lipid profile (mg/dl) (Cholesterol Triglyceride, HDL, LDL, VLDL) according to study groups including (25) younger patients as Control group, younger patients with high HbA1C (> 7.5 HbA1C) suffering from DMT1, and (25) younger patients with moderate HbA1C (\leq 7.5 HbA1C) was investigated. significant differences between means of lipid profile according to study group ($P 0.001$). **Conclusion:** Adolescent patients, which have DMT1, have abnormal lipid profile level and abnormal lipoprotein building. Then after grown up glycemic control is a significant become abnormal.

Keywords: Diabetic mellitus type 1, Younger patients, Cholesterol, HbA1C, Triglyceride.

Resumen

Antecedentes: la dislipidemia y la hiperglucemia son anomalías metabólicas que se encuentran comúnmente en pacientes jóvenes con diabetes mellitus tipo 1 (DM1) y ambas aumentan el riesgo de enfermedad cardiovascular, objetivo: estimar el perfil lipídico en pacientes más jóvenes y la afectación de estos lípidos en DMT1. **Métodos:** estudio de casos y controles de (75) pacientes más jóvenes que se dividieron en tres grupos según la HbA1C, (25) pacientes más jóvenes con HbA1C alta (> 7,5 HbA1C) que padecían DMT1, (25) pacientes más jóvenes con HbA1C moderada (\leq 7,5 HbA1C) y (25) pacientes menores como control sanos, con rango de edad de 10 a 19 años que asistieron Cambiando Diabetes en Niños. Los pacientes más jóvenes fueron visita a proveedores clínicos. Todos los pacientes y el control eran del mismo grupo étnico (árabe). Muestras de sangre recolectadas de cada paciente; Se realizaron pruebas de perfil de lípidos para cada muestra de sangre para estimar el colesterol, los triglicéridos, HDL, LDL y VLDL y compararlos entre los grupos de estudio. **Resultados:** HbA1C, el control glucémico entre los grupos comparados y HbA1c fue significativamente ($P = 0,001$) mayor en el grupo de dislipidemia. La media de glucosa en sangre en ayunas también fue significativamente mayor en el grupo de dislipidemia ($P 0,0001$). Las diferencias medias del perfil lipídico (mg/dl) (triglicéridos de colesterol, HDL, LDL, VLDL) según los grupos de estudio incluidos (25) pacientes más jóvenes como grupo de control, pacientes más jóvenes con HbA1C alta (> 7,5 HbA1C) que padecen DMT1 y (25) Se investigó en pacientes más jóvenes con HbA1C moderada (\leq 7,5 HbA1C) diferencias significativas entre las medias del perfil lipídico según el grupo de estudio ($P 0,001$). **Conclusión:** Los pacientes adolescentes, que tienen DMT1, tienen un perfil lipídico anormal y una formación anormal de lipoproteínas. Luego, después de crecer, el control glucémico es un cambio significativo.

Palabras clave: Diabético mellitus tipo 1, Pacientes jóvenes, Colesterol, HbA1C, Triglicéridos.

Introduction

Diabetes mellitus is a non-irresistible sickness that has a high commonness around the world¹. Changed degree of numerous hematological boundaries have been seen in patients with diabetes². The degrees of lipids are likewise influenced in diabetes by numerous elements since sugar digestion influence lipid digestion. Diabetes is a significant risk factor for cardiovascular contagion (CVD)³. Patients have diabetes type 1, atherosclerosis happens prior throughout everyday life, prompting expanded bleakness and mortality contrasted and those in everyone⁴. In addition, investigations of the regular history of atherosclerosis advancement highlight a cause of the injuries in youth and pre-adulthood⁵. Lipid fixations are unequivocally identified with the risk of cardiac problems when DMT1 developed⁶, patients have type 1 diabetes are documented to have a progressive danger for atherosclerotic illness^{7,8}. It not known if the lipid profile "lipid fixations and lipoprotein piece" occur in adolescence they have type 1 diabetes is encourages atherogenic action when compared with nondiabetic childhood⁹. Patients with diabetes, lipoprotein relationship is additional atherogenic¹⁰ and is knowingly distress by glycemic control^{11,12}. Aim of study: To estimated lipid profile in younger patients and effected of these lipids on DMT1

Methods

Case control study done from January to June 2021, (75) patients arranged to 3 groups according HbA1C, (>7.5, ≤ 7.5 have DMT1 and normal healthy persons), with age 10-19 years old, from all take fasting blood sugar, lipid profile (Cholesterol, Triglyceride, HDL, LDL, VLDL). Statistical analysis done by SPSS version 23. Continuous variables presented as (Means ± SD). Student t-test used to compare means between two groups. ANOVA test used to compare means between three groups or more. Correlation coefficient (r) used to assess the relationship between two continuous variables. A p-value of ≤ 0.05 considered as significant.

Results

The groups of this study were divided according to HbA1C, Glycemic control between two groups compared and HbA1c was significantly (P = 0.001) higher in dyslipidemic group. The mean fasting blood glucose was also significantly higher in dyslipidemic group (P 0.0001). The different types of dyslipidemia were statistically significant while compared between good and poor glycemic control as shown in Table 1.

Table 1. Mean difference between study groups according to HbA1C.

marker	Study groups			P value
	High HbA1C > 7.5	Moderate HbA1C ≤ 7.5	Control healthy	
	Mean ± SD	Mean ± SD	Mean ± SD	
HbA1C	9.140 ± 2.0376	6.524 ± 0.2332	5.865 ± 0.4837	0.001*

In this study, the mean differences of lipid profile (mg/dl) (Cholesterol Triglyceride, HDL, LDL, VLDL) according to study groups including (25 younger patients as Control group, younger patients with high HbA1C (> 7.5 HbA1C) suffering from DMT1, and (25) younger patients with moderate HbA1C (≤ 7.5 HbA1C)) was investigated in Table 2. The results showed that there was significant differences between means of lipid profile according to study group (P 0.001). The mean of cholesterol in high HbA1C groups was increased (265.848±) when compared with Moderate HbA1C (±) and control healthy patients (172.155±19.4425). Triglyceride was increased in mean of high HbA1C (±) compared with moderate (±) and control (±). However, there were increased in mean of high HbA1C (±), (±) respectively according to HDL and LDL compared with other groups (±), (±), while decrease in VLDL (35.144±2.4850) compared with moderate HbA1C (137.264±16.7629), but increase compared with control healthy (16.295±2.3273).

The correlation between high HbA1C patients and lipid profile shown in table 3.

The correlation between moderate HbA1C patients and lipid profile were shown in table 4.

The correlation between control patients and lipid profile were shown in table 5.

Table 2. lipid profile (mg/dl) (Cholesterol Triglyceride, HDL, LDL, and VLDL) in younger patients with high HbA1C and compered with moderate HbA1C and control groups.

marker	Study groups			P value
	High HbA1C > 7.5	Moderate HbA1C ≤ 7.5	Control healthy	
	Mean ± SD	Mean ± SD	Mean ± SD	
Cholesterol (mg/dl)	265.848±12.4408	175.302±11.7579	172.155±19.4425	0.001*
Triglyceride (mg/dl)	430.068±72.2505	222.776±6.9082	129.860±11.4351	0.001*
HDL (mg/dl)	55.432±3.2290	43.372±1.1190	30.020±6.8592	0.001*
LDL (mg/dl)	126.928±10.5925	65.860±3.1595	51.455±8.8246	0.001*
VLDL (mg/dl)	35.144±2.4850	137.264±16.7629	16.295±2.3273	0.001*

Table 3. The correlation between high HbA1C patients and lipid profile

		Correlations					
		Cholesterol	TG	HDL	LDL	VLDL	HbA1C
Cholesterol	Correlation	1	.179	-.239-	.098	.517**	.025
	significant		.392	.250	.642	.008	.907
TG	Correlation	.179	1	-.025-	.214	.134	.172
	significant	.392		.907	.305	.522	.411
HDL	Correlation	-.239-	-.025-	1	.029	-.178-	.352
	significant	.250	.907		.890	.395	.085
LDL	Correlation	.098	.214	.029	1	-.014-	-.020-
	significant	.642	.305	.890		.948	.926
VLDL	Correlation	.517**	.134	-.178-	-.014-	1	.010
	significant	.008	.522	.395	.948		.963
HbA1C	Correlation	.025	.172	.352	-.020-	.010	1
	significant	.907	.411	.085	.926	.963	

** . Correlation is significant ≤ 0.05.

Table 4. The correlation between moderate HbA1C patients and lipid profile

		Correlations					
		Cholesterol	TG	HDL	LDL	VLDL	HbA1C
Cholesterol	Correlation	1	.088	.073	.029	.006	.047
	significant		.675	.727	.891	.977	.822
TG	Correlation	.088	1	.210	.005	.065	.257
	significant	.675		.313	.981	.759	.216
HDL	Correlation	.073	.210	1	-.044-	.123	-.117-
	significant	.727	.313		.836	.557	.577
LDL	Correlation	.029	.005	-.044-	1	.107	.427*
	significant	.891	.981	.836		.610	.033
VLDL	Correlation	.006	.065	.123	.107	1	.205
	significant	.977	.759	.557	.610		.325
HbA1C	Correlation	.047	.257	-.117-	.427*	.205	1
	significant	.822	.216	.577	.033	.325	

*. Correlation is significant ≤ 0.05.

Table 5. The correlation between control patients and lipid profile

		Correlations					
		Cholesterol	TG	HDL	LDL	VLDL	HbA1C
Cholesterol	Correlation	1	.247	.128	-.135-	.269	.135
	significant		.234	.541	.519	.194	.520
TG	Correlation	.247	1	.110	-.230-	.062	-.016-
	significant	.234		.602	.270	.770	.939
HDL	Correlation	.128	.110	1	.131	.019	-.212-
	significant	.541	.602		.532	.928	.310
LDL	Correlation	-.135-	-.230-	.131	1	.016	-.002-
	significant	.519	.270	.532		.941	.991
VLDL	Correlation	.269	.062	.019	.016	1	.176
	significant	.194	.770	.928	.941		.400
HbA1C	Correlation	.135	-.016-	-.212-	-.002-	.176	1
	significant	.520	.939	.310	.991	.400	

*. Correlation is significant ≤ 0.05.

Discussion

In this study, the cholesterol, TG, HDL, LDL was increase in younger patients with DMT1 similar to results of¹³ and decrease in VLDL as results of¹⁴. The pervasiveness of dyslipidemia in youngsters with T1DM fluctuates somewhere in the range of 29% and 66% in investigations from various countries¹⁵. The wide scope of commonness in different examinations might be because of numerous hereditary variables in various ethnic gatherings. The fundamental problem was hypertriglyceridemia in our examination. This finding is in harmony with the different studies¹⁶, however the most regular dyslipidemia was high LDL in different studies¹⁷. Younger patients with type 1 diabetes and perfect HbA1c levels have less atherogenic "HDL cholesterol, fatty material, fatty/HDL amount"¹⁸. Similarly, childhood with type 1 diabetes and atypical glycemic control have better lipid levels and ubiquity of lipid anomalies¹⁹. Childhood with type 1 diabetes have general raised lipoprotein and great LDL than nondiabetic childhood, and unusual glycemic control^{20,21}. Our awareness that adolescent with type 1 diabetes and perfect glycemic control have a fewer atherogenic lipid profile^{22,23}. Nevertheless, the deficiency of uncommon lipid levels does not exclude the accidental of compositional changes that might be atherogenic, chiefly among those with poor glycemic control^{24,25}. Critically, the evidently ordinary serum cholesterol fixations habitually saw in T1DM shroud an atherogenic lipid profile, with expanded transitional thickness lipoprotein and little thick low-thickness lipoprotein (sdLDL), and useless high-thickness lipoprotein (HDL)²⁶. HDL cholesterol levels are frequently ordinary or even high except if glycaemic control is poor or nephropathy has created Hypertriglyceridaemia may likewise happen, and the danger related with it is more grounded than in everybody²⁷. LDL molecule size, its glycation and oxidation are completely connected with endothelial brokenness and CVD²⁸. sdLDL enters the blood vessel divider more effectively than enormous light LDL, is more helpless to oxidative pressure, has a delayed plasma half-life and has a decreased restricting fondness for LDL receptors²⁹. These attributes would all be required to add to expanded atherogenicity. There is impressive proof involving lipid peroxidation and oxidative alteration of LDL in atherosclerotic sore turn of events³⁰, yet non-enzymatic glycation of LDL might be comparably significant. sdLDL is more promptly glycosylated than bigger more light LDL both in vivo and in vitro, potentially on the grounds that a higher extent of the apolipoprotein B (apoB) particle is presented to glucose³¹.

Conclusions

A Adolescent patients, which have DMT1, have abnormal lipid profile level and abnormal lipoprotein building. Then after grown up glycemic control is a significant become abnormal.

References

1. Chen, P., Takeuchi, F., Lee, J. Y., Li, H., Wu, J. Y., Liang, J., & CHARGE Hematology Working Group. Multiple nonglycemic genomic loci are newly associated with blood level of glycosylated hemoglobin in East Asians. *Diabetes*, 2014, 63(7), 2551-2562.
2. Hardikar, P. S., Joshi, S. M., Bhat, D. S., Raut, D. A., Katre, P. A., Lubree, H. G., & Yajnik, C. S. Spuriously high prevalence of prediabetes diagnosed by HbA1c in young Indians partly explained by hematological factors and iron deficiency anemia. *Diabetes Care*, 2012, 35(4), 797-802.
3. Kabootari M, Hasheminia M, Azizi F, Mirbolouk M, Hadaegh F. Change in glucose intolerance status and risk of incident cardiovascular disease: Tehran Lipid and Glucose Study. *Cardiovasc Diabetol*. 2020 Mar 30;19(1):41.
4. Monnier L, Colette C. Postprandial and basal hyperglycaemia in type 2 diabetes: Contributions to overall glucose exposure and diabetic complications. *Diabetes Metab*. 2015 Dec;41(6 Suppl 1):659-6515.
5. Fox, C. S., Coady, S., Sorlie, P. D., Levy, D., Meigs, J. B., D'Agostino, R. B., & Savage, P. J. Trends in cardiovascular complications of diabetes. *Jama*, 2004, 292(20), 2495-2499.
6. Parthasarathy, P., & Vivekanandan, S. Urate crystal deposition, prevention and various diagnosis techniques of GOUT arthritis disease: a comprehensive review. *Health information science and systems*, 2018, 6 (1), 1-13.
7. Carroll, Aaron E. *The Bad Food Bible: Why You Can (and Maybe Should) Eat Everything You Thought You Couldn't*. First Mariner Books edition. Boston: Mariner Books/Houghton Mifflin Harcourt, 2019.
8. Gilpin VL; American Diabetes Association; American Heart Association; American College of Cardiology Foundation. Review of an article: aspirin for primary prevention of cardiovascular events in people with diabetes. A position statement of the American Diabetes Association (ADA), a scientific statement of the American Heart Association (AHA), and an expert consensus document of the American College of Cardiology Foundation (ACCF). *Diabetes Care*, 33:6; June 2010; 1395-1402. *J Vasc Nurs*. 2010 Dec;28(4):154-5.
9. Quispe R, Martin SS, Jones SR. Triglycerides to high-density lipoprotein-cholesterol ratio, glycemic control and cardiovascular risk in obese patients with type 2 diabetes. *Curr Opin Endocrinol Diabetes Obes*. 2016 Apr;23(2):150-6.
10. Grøntved A, Koivula RW, Johansson I, Wennberg P, Østergaard L, Hallmans G, Renström F, Franks PW. Bicycling to Work and Primordial Prevention of Cardiovascular Risk: A Cohort Study Among Swedish Men and Women. *J Am Heart Assoc*. 2016 Oct 31;5(11):e004413.

11. Ortega Loubon, C., Fernández Molina, M., Singh, G., & Correa, R. Obesity and its cardiovascular effects. *Diabetes/metabolism research and reviews*, 2019, 35(4), e3135.
12. Bastien M, Poirier P, Lemieux I, Després JP. Overview of epidemiology and contribution of obesity to cardiovascular disease. *Prog Cardiovasc Dis*. 2014 Jan-Feb;56(4):369-81.
13. Ghio, A., Bertolotto, A., Resi, V., Volpe, L., & Di Cianni, G. Triglyceride metabolism in pregnancy. *Advances in clinical chemistry*, 2011, 55, 134.
14. Mitrovi, M., Popovi, Đ. S., Nagli, D. T., Paro, J. N., Ili, T., & Zaviši, B. K. Markers of inflammation and microvascular complications in type 1 diabetes. *Central European Journal of Medicine*, 2014, 9(6), 748-753.
15. Noori, N., Nakhaee-Moghadam, M., Teimouri, A., & Bagheri, H. Tissue Doppler Imaging Findings and Lipid Profile Changes in Diabetes Mellitus Type I Children. *International Journal of Pediatrics*, 2019, 7(12), 10423-10439.
16. Kung, W. J., Shih, C. T., Lee, C. H., & Lin, C. C. The divalent element changes in early stages of chronic kidney disease. *Biological trace element research*, 2018, 185(1), 30-35.
17. Safarova MS, Kullo IJ. My Approach to the Patient With Familial Hypercholesterolemia. *Mayo Clin Proc*. 2016;91(6):770-786.
18. Liu, T. A Comparison of Biological and Physical Risk Factors for Cardiovascular Disease in Overweight/Obese Individuals with and Without Prediabetes. *Clinical Nursing Research*. 2017, 26, 674–693.
19. Ode, K. L., Frohnert, B. I., & Nathan, B. M. Identification and treatment of metabolic complications in pediatric obesity. *Reviews in endocrine and metabolic disorders*, 2009, 10(3), 167-188.
20. Allen RW, Schwartzman E, Baker WL, Coleman CI, Phung OJ. Cinnamon use in type 2 diabetes: an updated systematic review and meta-analysis. *Ann Fam Med*. 2013 Sep-Oct;11(5):452-9.
21. Suadicani P, Hein HO, Gyntelberg F. Antihypertensive treatment, high triglycerides, and low high-density lipoprotein cholesterol and risk of ischemic heart disease mortality: a 16-year follow-up in the Copenhagen male study. *Metab Syndr Relat Disord*. 2010 Jun;8(3):215-22.
22. Brown JM, Everett BM. Cardioprotective diabetes drugs: what cardiologists need to know. *Cardiovasc Endocrinol Metab*. 2019;8(4):96-105. Published 2019 Nov 13.
23. Raymond, T., Raymond, R., & Lincoff, A. M. Management of the patient with diabetes and coronary artery disease: a contemporary review. *Future cardiology*, 2013, 9(3), 387-403.
24. Longato E, Di Camillo B, Sparacino G, Gubian L, Avogaro A, Fadini GP. Cardiovascular outcomes of type 2 diabetic patients treated with SGLT-2 inhibitors versus GLP-1 receptor agonists in real-life. *BMJ Open Diabetes Res Care*. 2020 Jun;8(1):e001451.
25. Peterson RG, Jackson CV, Zimmerman K, de Winter W, Huebert N, Hansen MK. Characterization of the ZDSD Rat: A Translational Model for the Study of Metabolic Syndrome and Type 2 Diabetes. *J Diabetes Res*. 2015;2015:487816.
26. Zali nas R, Slapikas R, Gustiene O, Siurkus J, Vaitkus E. Mazo tankio lipoproteinų afereze [Low density lipoprotein apheresis]. *Medicina (Kaunas)*. 2003;39(12):1158-64.
27. Hirano T. Pathophysiology of Diabetic Dyslipidemia. *J Atheroscler Thromb*. 2018 Sep 1;25(9):771-782.
28. Bock ME, Wall L, Dobrec C, Chandran M, Goebel J. Management of dyslipidemia in pediatric renal transplant recipients. *Pediatr Nephrol*. 2021 Jan;36(1):51-63.
29. Malekmohammad K, Sewell RDE, Rafieian-Kopaei M. Antioxidants and Atherosclerosis: Mechanistic Aspects. *Biomolecules*. 2019;9(8):301. Published 2019 Jul 25.
30. Tsikas D. Assessment of lipid peroxidation by measuring malondialdehyde (MDA) and relatives in biological samples: Analytical and biological challenges. *Anal Biochem*. 2017 May 1;524:13-30.
31. Sparks JD, Chamberlain JM, O'Dell C, Khatun I, Hussain MM, Sparks CE. Acute suppression of apo B secretion by insulin occurs independently of MTP. *Biochem Biophys Res Commun*. 2011 Mar 11;406(2):252-6.