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RESEARCH ARTICLE

Influence of metabolic parameters on LDL and HDL size and subclasses in adolescents with type 1 diabetes

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Summary

Alterations in the serum of low-density lipoprotein (LDL) and high-density lipoprotein (HDL) size and subclass contribute to the atherogenesis in coronary artery disease in diabetic patients.

The purpose of this study is to evaluate the effect of metabolic parameters on LDL and HDL size and subclasses in adolescents with type 1 diabetes.

The cross-sectional study included 43 adolescents (23 females, 20 males) with type 1 diabetes of mean age 15.09±1.94 years, with mean disease duration of 5.86 ± 3.08 years. LDL and HDL particles were separated by polyacrylamide gradient gel electrophoresis, while serum lipid parameters were determined by routine laboratory methods.

Patients with inadequate metabolic control (HbA1c ≥ 7.5%) had a higher mean value of triglycerides (TG) (p = 0.041), higher proportions of small, dense LDL particles (p = 0.045), higher proportions of LDL IIA subclasses (p=0.03) and smaller LDL diameter (p = 0.02) and HDL diameter (p = 0.04) than patients with optimal metabolic control (HbA1c < 7.5%). Higher HbA1c and higher TG levels were statistically significantly related to small, dense LDL (ρ=0.341, p=0.025; ρ= 0.394, p= 0.009) and HDL particles (ρ=0.684, p=0.000; ρ=0.421, p=0.005). Predictors of small, dense LDL and HDL particles, which contribute to atherogenesis, were high HbA1c (HR = 1.52, 95% CI: 0.97-2.40; HR 3.87, 95% CI: 2.11-7.10) and elevated TG (HR= 1.10, 95% CI: 1.00-1.20; HR 1.85, 95% CI: 1.07-3.21).

Diabetic adolescents require particular attention in order to minimize factors such as high HbA1c and elevated TGs in the development of future cardiovascular events.

Keywords: type 1 diabetes, adolescents, LDL, HDL

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INTRODUCTION

Clinical manifestations of cardiovascular disease (CVD) are extremely rare in childhood, but the atherosclerotic process starts as early as the first years of life and is significantly accelerated by type 1 diabetes (1,2). Hyperglycemia is considered the primary mediator of atherosclerosis in type 1 diabetes, where for each percent of absolute raise in glycated hemoglobin (HbA1c) the relative risk for future CVD events is increased by 7% (1-4). People with type 1 diabetes and good metabolic control have similar lipid profiles as the healthy population, but it is not clear whether their lipid composition is more atherogenic (5-8). On the other hand, sub-optimal metabolic control could lead to diabetic dyslipidemia, which is characterized by high levels of low-density lipoprotein cholesterol (LDL-C) and triglycerides (TG), and low levels of high-density lipoprotein cholesterol (HDL-C) (9). Alterations in serum lipids and lipoprotein levels, especially the presence of small, dense LDL and HDL particles contribute to atherogenesis in coronary artery disease (CAD) in the general population as well as in diabetic patients, although studies in children and adolescents with type 1 diabetes are limited. (8).

In this cross-sectional study, we investigated the effect of metabolic parameters on LDL and HDL size and subclasses in adolescents with type 1 diabetes. There have been no reports specifically addressed to the evaluation of risk factors of lipoprotein size and subclass distribution that have been involved in premature atherosclerosis in type 1 diabetic adolescents.

PATIENTS AND METHODS

In a cross-sectional study involving 43 adolescents (23 females, 20 males) with type 1 diabetes of mean age 15.09±1.94 years, with mean disease duration of 5.86 ± 3.08 years, we evaluated lipoprotein subclasses and their connection with metabolic risk factors. The patients were treated at the University Children's Hospital of Belgrade, Serbia. The patients were selected according to the following criteria: the duration of type 1 diabetes over 2 years and patients' age of over 11 years. They were classified into two groups according to the HbA1c: adolescents with optimal metabolic control (HbA1c < 7.5%) and adolescents with inadequate metabolic control (HbA1c ≥ 7.5%). None of the patients had any known family history of dyslipidemia or early CVD. Adolescents were checked four times a year or more often if required and assessed at the end of the year. Assessment consisted of measurements of HbA1c, body mass index (BMI), total cholesterol (TC), TGs and calculations of insulin doses during the first visit to the hospital, updated at each following visit. All the patients were on an intensive insulin therapy regimen. Ambulatory blood pressure monitoring (ABPM) was performed and lipoprotein subclasses were determined when adolescents underwent their annual check-up during the 24-hour hospitalization. Adolescents with type 1 diabetes and associated diseases, which may interfere with lipoprotein levels, were excluded. The stage of puberty was evaluated according to Tanner. All the adolescents had either Tanner stage 2-4 (67.4%) or completed pubertal development (32.6%). Using oscillometric type of SpaceLabs 90207 units and appropriate cuffs placed on the non-dominant arm, the ABPM recorded 24-hour values of systolic blood pressure (SBP) and diastolic blood pressure (DBP).

METABOLIC PARAMETERS

Glycated hemoglobin (HbA1c) was measured by turbidimetric immunoassay using the Beckman Synchron CX5 Clinical System apparatus. In healthy persons, normal HbA1c test results using this method range from 4.0% to 6.5%.

Blood samples were drawn for analyzing LDL-C, HDL-C, TC, TGs and LDL and HDL subclass distribution. Electrophoretic separation and determination of 7 LDL and 5 HDL subclasses was performed at the research laboratory of the Department for Medical Biochemistry at the Faculty of Pharmacy, University of Belgrade. In brief, for the separation of lipoprotein subclasses we used in-house casted polyacrylamide gradient (3-31%) gels. Electrophoresis was conducted by using the Hoefer SE 600 Ruby electrophoresis unit (Amersham Pharmacia Biotech, Vienna, Austria). For the calibration of the gels, we applied Pharmacia High Molecular Weight protein standards and carboxylate polystyrene microsphere beads. Following electrophoretic separation, protein standards and lipoprotein subclasses were visualized by staining with Coomassie brilliant blue G-250 and Sudan Black, respectively. The analysis of separated fractions and determination of LDL and HDL subclasses distribution was performed by using Image Scanner (Amersham Pharmacia Biotech, Vienna, Austria) and Image Quant software (version 5.2;1999; Molecular Dynamics, GE Healthcare, Waukesha, WI, USA). Dominant LDL and HDL particle diameters were estimated as diameters of major peaks in the LDL and HDL region of the scan. The relative proportion of each subclass was determined as the percentage of the entire LDL or HDL densitometric scan area, which corresponds to the peak of each subclass. The relative proportion of small, dense LDL was assessed by adding the relative proportions of LDL III and IV subclasses. Accordingly, the relative proportion of small HDL subclasses was determined as a sum of percentages of HDL 3a, 3b and 3c subclasses.

STATISTICAL ANALYSES

For statistical data processing, in our study we used the statistical package SPSS for Windows. The obtained data were presented as mean ± SD. The comparison between the two independent groups was evaluated using the independent two-sample Student's t-test or Mann-Whitney U-test when appropriate. The default of $\alpha < 0.05$ specified the level of significance in all the tests. The correlation between the numerical variables was calculated by Spearman's correlation coefficient (ρ). Univariate analysis for predictors of small, dense LDL and HDL particles was performed using simple logistic regression. The study was approved by the Medical Ethics Committee of the University Children's Hospital, affiliated to the Faculty of Medicine, University of Belgrade, Serbia (approval number: 2019-053).

RESULTS

Clinical data and lipid profiles of adolescents with type 1 diabetes with optimal and inadequate metabolic control are presented in **Table 1**. Forty-three subjects, 23 females (53.5%) and 20 males (46.5%) met the inclusion criteria for the study. The mean HbA1c was 7.93 ± 1.38%; 18 (41.9%) patients had inadequate metabolic control with mean value 8.30 ±1.54% which statistically differs from that in patients with optimal metabolic control 7.51±1.04%. The patients with inadequate metabolic control had a higher mean value of TGs (1.03 ± 0.63 vs 0.83 ± 0.39 mmol/L; p = 0.041), higher proportions of small, dense LDL particles (53.62 ± 13.80 vs 49.86 ± 18.83%; p = 0.045), higher proportions of LDL IIA subclasses (12.37 ± 5.54 vs 11 ± 3.65%; p=0.03) and smaller LDL diameter (21.42 ± 1.59 vs 25.88 ± 2.26 nm; p = 0.02) and HDL diameter (9.33 ± 0.94 vs 10.34 ± 0.98 nm, p = 0.04) than patients with optimal metabolic control. With respect to all other proportions of lipoprotein subclasses, there was no statistically significant difference.

Table 1. Clinical data and lipid profiles of diabetic adolescents with optimal and inadequate metabolic control (X±SD)

Characteristics	**HbA1c ≥ 7.5% N=18**	**HbA1c < 7.5% N=25**	**Total N=43**	**p value**
Age (years)	15.78 ± 1.67	14.3 ± 1.97	15.09 ± 1.94	0.97
Gender (male/female) (%)	61.1/38.9	52/48	46.5/53.5	0.36
Mean diabetes duration (years)	6.88 ± 3.21	4.75 ± 2.57	5.86 ± 3.08	0.71
Pubertal Tanner stage 2-4/5 (%)	69.6/30.4	65/35	67.4/32.6	0.12
BMI (kg/m^2)	22.05 ± 2.63	19.37 ± 2.64	20.71 ± 2.91	0.45
HbA1c (%)	8.30 ± 1.54	7.51 ± 1.04	7.93 ± 1.38	0.03*
Mean insulin dose (units/kg/day)	0.81± 0.34	0.85 ± 0.34	0.83 ± 0.34	0.95
SBP (mmHg)	109.50±11.6	107.1±10.3	108.29 ± 11.15	0.65
DBP (mmHg)	69.60 ± 5.10	67.80 ± 9.30	68.90 ± 7.29	0.75
Triglycerides (mmol/L)	1.03 ± 0.63	0.83 ± 0.39	0.92 ± 0.52	0.041*
Total cholesterol (mmol/L)	4.72 ± 0.92	4.61 ± 0.68	4.68 ± 0.82	0.16
HDL-C (mmol/L)	1.82 ± 0.38	1.73 ± 0.41	1.77 ± 0.39	0.052
LDL-C (mmol/L)	2.57 ± 0.76	2.20 ± 0.58	2.38 ± 0.67	0.11
Small, dense LDL (%)	53.62 ± 13.80	49.86 ± 18.83	51.61 ± 16.60	0.045*
LDL diameter (nm)	21.42 ± 1.59	25.88 ± 2.26	25.86 ± 2.14	0.02*
LDL I (%)	23.53 ± 11.25	21.42 ± 8.59	22.55 ± 10.04	0.25
LDL IIA (%)	12.37 ± 5.54	11 ± 3.65	11.73 ± 4.75	0.03*
LDL IIB (%)	14.23 ± 4.85	13.95 ± 3.55	14.10 ± 4.25	0.34
LDL IIIA (%)	12.52 ± 3.30	13.32 ± 2.86	12.89 ± 3.09	0.36
LDL IIIB (%)	6.37 ± 2.26	6.98 ± 1.92	6.65 ± 2.10	0.36
LDL IVA (%)	12.62 ± 5.60	12.53± 4.40	12.58 ± 5.02	0.19
LDL IVB (%)	18.32 ± 10.71	20.8 ± 8.45	19.47 ± 9.69	0.10
Small HDL (%)	28.03 ± 12.56	29.63 ± 12.34	28.78 ± 12.34	0.97
HDL diameter (nm)	9.33 ± 0.94	10.34 ± 0.98	10.22 ± 0.95	0.04*
HDL 2a (%)	19.39 ± 5.72	20.42 ± 6.14	19.87 ± 5.87	0.64
HDL 2b (%)	52.56 ± 12.77	49.94 ± 13.73	51.34 ± 13.14	0.50
HDL 3a (%)	10.58 ± 4.25	11.93 ± 5.06	11.21 ± 4.64	0.20
HDL 3b (%)	6.29 ± 5.31	6.24 ± 4.22	6.26 ± 4.78	0.78
HDL 3c (%)	11.16 ± 6.46	11.45 ± 6.62	11.29 ± 6.46	0.85

X – mean value; SD – standard deviation; BMI – body mass index (kg/m^2); HbA1c – glycosylated hemoglobin (%); SBP: Systolic Blood Pressure (mmHg); DBP: Diastolic Blood Pressure (mmHg); HDL-C – high-density lipoprotein cholesterol (mmol/L); LDL-C – low-density lipoprotein cholesterol (mmol/L); *p < 0.05 between diabetic adolescents with optimal and inadequate metabolic control

Table 2. Results of univariate logistic regression analysis for small, dense LDL and HDL particles

	Small, dense LDL particles		Small HDL particles	
Variable	p value	Odds ratio (95% CI) mean (range)	p value	Odds ratio (95% CI) mean (range)
HbA1c (≥7.5%)	0.04*	1.52 (0.97-2.40)	0.034*	3.87 (2.11-7.10)
BMI (kg/m^2)	0.78	0.84 (0.25-2.87)	0.73	1.40 (0.20-9.50)
TG (>1.5mmol/L)	0.04*	1.10 (1.00-1.20)	0.02*	1.85 (1.07-3.21)
TC (>5.2mmol/L)	0.42	1.01 (0.98-1.05)	0.76	1.03 (0.84-1.27)
LDL-C (>2.6mmol/L)	0.05	13.18 (2.62-66.19)	0.50	0.58 (0.12-2.88)
HDL-C (<1.1mmol/L)	0.80	1.43 (0.09-2.11)	0.78	1.07 (0.67-1.70)
SBP (mmHg)	0.77	0.99 (0.93-1.05)	0.73	1.39 (0.20-9.48)
DBP (mmHg)	0.95	1.00 (0.92-1.09)	0.49	1.04 (0.93-1.15)

HbA1c – glycosylated hemoglobin (%),BMI – body mass index (kg/m^2), TG – triglycerides (mmol/L), TC – total cholesterol (mmol/L), LDL-C – low-density lipoprotein cholesterol (mmol/L), HDL-C – high-density lipoprotein cholesterol (mmol/L), SBP: Systolic Blood Pressure (mmHg), DBP: Diastolic Blood Pressure (mmHg)); *p < 0.05

We found a statistically significant correlation between HbA1c, TGs and small, dense LDL particles (ρ=0.341, p=0.025; ρ= 0.394, p= 0.009), indicating the association between increased HbA1c, TGs, and small, dense LDL particles and vice versa. Also, the small HDL particles were in a statistically significant correlation with HbA1c and TGs (ρ=0.684, p=0.000; ρ=0.421, p=0.005). No correlation was found between the duration of diabetes, insulin doses, TC, LDL-C, HDL-C, SBP, DBP and small, dense LDL and HDL particles.

In our patients, higher TG levels were in a statistically significant negative correlation with LDL and HDL diameters (ρ=-0.650, p=0.000; ρ=-0.394 p=0.009;).

We constructed univariate logistic regression models to examine factors associated with atherosclerosis in CAD in diabetic adolescents **(Table 2)**. Factors associated with small, dense LDL and HDL particles were high HbA1c (hazard ratio (HR)= 1.52, 95% confidence interval (CI): 0.97-2.40; HR 3.87, 95% CI: 2.11-7.10) and elevated TGs (HR= 1.10, 95% CI: 1.00-1.20; HR 1.85, 95% CI: 1.07-3.21) **(Table 2)**.

DISCUSSION

Pediatric patients with type 1 diabetes have a higher risk of CVD development at an earlier age. (9). Hyperglycemia predisposes atherogenesis in type 1 diabetes and silent coronary atherosclerosis in young adults with diabetes is strongly associated with poor glycemic control (9-11). Poor glycemic control is also associated with a potentially more atherogenic lipoprotein profile (12). Well-controlled type 1 diabetes is not associated with gross blood lipid disturbance, but more advanced lipoprotein subclass examinations reveal atherogenic profiles (13). In patients on intensive insulin therapy or continuous subcutaneous insulin infusion, the TG level is usually optimal and we showed that diabetic adolescents with inadequate metabolic control didn't have hypertriglyceridemia (>1.5 mmol/L) but they had significantly higher TGs, higher proportions of small, dense LDL particles, smaller LDL and HDL particle sizes and more LDL IIA subclasses than adolescents with optimal metabolic control.

Previous studies documented a presence of smaller LDL and HDL particles in people with type 1 diabetes (14,15), but this is the first study to report abnormal lipoprotein composition (LDL and HDL size and subclasses) in adolescents with type 1 diabetes. Cholesterol levels of our type 1 diabetic adolescents did not significantly correlate with LDL and HDL size and with the amount of small, dense LDL and HDL particles possibly because of a small sample of subjects. Also, several studies showed that the TC levels of the diabetic children did not vary throughout puberty in contrast to TG levels (1,7,16). Our results indicated that high HbA1c and higher TGs were the main factors associated with the higher amount of small, dense LDL and HDL particles and higher TG was associated with smaller LDL and HDL particle sizes. Our current data extend previous limited observations on lipid profile in people with type 1 diabetes with the finding of significantly reduced HDL size, but not LDL size. Investigations confirmed a strong association of smaller HDL size with elevated oxidative stress and low-grade inflammation in asymptomatic individuals, suggesting that the abnormalities in HDL particles occur even before the onset of atherosclerosis (15,16). A correlation between higher TG and smaller LDL size has not been reported previously and the precise mechanism underlying this association is unknown. These studies also showed that highly increased TGs rich in lipoprotein subclasses are observed in relation to reduced lipoprotein lipase activity, as well as the increased presence of small, dense LDL and HDL particles which contribute to early atherosclerosis (15-17).

STUDY LIMITATIONS

The main limitation of our study is the inclusion of a relatively small group of participants. However, taking into

consideration that we analyzed adolescents over the age of 11, with the duration of type 1 diabetes of over two years, results can be considered valuable. To obtain more reliable data, it would be advisable to perform a similar analysis on a large sample of adolescents with type 1 diabetes.

CONCLUSION

In conclusion, the risk factors identified for the presence of small, dense LDL and HDL particles in our adolescents with type 1 diabetes were poorer glycemic control and elevated TG levels. Since these particles could be one of the factors that accelerate the development of atherosclerosis in type 1 diabetes, screening for their presence and identification of the factors affecting their excessive production, could have beneficial effects on reducing the risk of future cardiovascular events.

Conflict of interest

None to declare.

Author contributions

The conception or design of the work – Maja Ješić, Smiljka Kovačević; the acquisition- Smiljka Kovačević, Vera Zdravković, Stefan Đorđević, analysis, or interpretation of data- Maja Ješić, Smiljka Kovačević, Aleksandra Zeljković, Dejana Stanisavljević; preparing the draft of the manuscript or interpretation of revised version of manuscript- Maja Ješić, Miloš Ješić, Milica Vuković.

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UTICAJ METABOLIČKIH PARAMETARA NA VELIČINU I SUBKLASU LDL I HDL KOD ADOLESCENATA SA DIJABETESOM TIP 1

Smiljka Kovačević[1], Vera Zdravković [1,2], Stefan Đorđević[1], Miloš M Ješić[1,2], Aleksandra Zeljković[3], Dejana Stanisavljević[4], Milica Vuković[5], Maja D Ješić[1,2]

Sažetak

Promene veličine i potklasa lipoproteina male gustine (LDL) i lipoproteina velike gustine (HDL) u serumu doprinose nastanku aterogeneze tokom razvoja koronarne arterijske bolesti kod pacijenata sa dijabetesom.

Cilj rada je proceniti uticaj metaboličkih parametara na veličinu i podklasu LDL i HDL čestica kod adolescenata sa dijabetesom tipa 1.

U studiju preseka bilo je uključeno 43 adolescenta (23 žene, 20 muškaraca) sa dijabetesom tipa 1 prosečne starosti 15,09 ± 1,94 godina, sa prosečnim trajanjem bolesti od 5,86 ± 3,08 godina. Čestice LDL i HDL razdvojene su elektroforezom u gradijentnom poliakrilamidnom gradijentu, dok su vrednosti parametara serumskih lipida određene rutinskim laboratorijskim metodama.

Pacijenti sa neadekvatnom metaboličkom kontrolom (HbA1c ≥ 7,5%) imali su veću srednju vrednost triacilglicerola (TG) (p = 0,041), veći udeo potklase malih, gustih LDL čestica (p = 0,045), veći udeo potklase LDL IIA (p = 0,03) i manji prečnik LDL -a (p = 0,02) i prečnika HDL -a (p = 0,04) od pacijenata sa optimalnom metaboličkom kontrolom (HbA1c <7,5%). Više koncetracije HbA1c i TG bile su statistički značajno povezanie sa postojanjem malih, gustih LDL čestica (r = 0,341, p = 0,025? r = 0,394, p = 0,009) i HDL česticama (r = 0,684, p = 0,000? r = 0,421, p = 0,005). Prediktori malih, gustih čestica LDL i HDL, koji doprinose aterogenezi, bili su visoki HbA1c (HR = 1,52, 95% CI: 0,97-2,40; HR 3,87, 95% CI: 2,11-7,10) i povišena koncentracija TG (HR = 1,10, 95% CI: 1,00-1,20; HR 1,85, 95% CI: 1,07 - 3,21).

Da bi se kod adolescenata obolelih od dijabetesa sprečio razvoj budućih kardiovaskularnih poremećaja posebnu pažnju treba obratiti na faktore kao što su visoke koncentracije HbA1c i TG.

Ključne reči: tip 1 dijabetes, adolescenti, HDL, LDL.

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