DOI: 10.4149/BLL_2018_140

Bratisl Med J 2018; 119 (12)
770 – 775

CLINICAL STUDY

Is elevated triglyceride high density lipoprotein cholesterol ratio a risk factor that causes acute coronary syndrome to appear earlier?

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ABSTRACT

BACKGROUND: The purpose of this study was to assess the relationship between the triglyceride/high density lipoprotein cholesterol ratio and the risk of acute myocardial infarction in young adults.

PATIENTS AND METHODS: A total of 621 patients, who underwent coronary angiography (CAG) due to Myocardial Infarction (MI) at our hospital were included in this study. Demographic characteristics, risk factor profile, laboratory test results, electrocardiographic and CAG findings were assessed in the selected groups.

RESULTS: Total cholesterol, triglyceride/high density lipoprotein cholesterol (Tg/HDL) ratio, Tg levels, were higher in younger patients with MI, while glucose and high-density lipoprotein levels were lower. Using propensity score matching in the matched population comparing young patients to the older ones, serum triglyceride levels [179 (145–231) vs 148 (101–197)] and triglyceride to high density lipoprotein cholesterol ratio [5.8 (4.1–9.1) vs 3.0 (1.8–4.6)] were significantly higher, whereas high density lipoprotein levels were observed dramatically lower (32.6 ± 8.2 vs 41.7 ± 8.8).

CONCLUSION: This study demonstrated that Tg/HDL ratio may be an important predictor for an acute coronary syndrome in the young adult population. Tg/HDL ratio can be used to prevent MI in young adults *(Tab. 3, Fig. 1, Ref. 32.)* Text in PDF *www.elis.sk.*

KEY WORDS: acute myocardial infarction in young adults, triglyceride, HDL cholesterol.

Introduction

Coronary artery disease (CAD) is relatively rare among young individuals. Based on the age cut-off defined in studies, young patients with acute coronary syndrome account for 0.4–19 % of acute coronary syndrome (ACS) cases (1–5). Despite the lower incidence in young individuals, it is important to prevent and treat CAD in these patients in order to achieve long life expectancy.

In patients with ACS, who had low-density lipoprotein (LDL) levels below 130 mg/dL, triglycerides (Tg) were found to be higher and high-density lipoprotein (HDL) was lower compared to the control group (6). Gaziano et al showed for the first time that Tg/HDL ratio was a strong predictor of myocardial infarction (MI) (7). Furthermore, Tg/HDL ratio is closely associated with cardiovascular risk factors and may predict CAD development and cardiovascular mortality (8, 9). Tg/HDL ratio is associated with insulin resistance and insulin resistance is known to accelerate atherogenesis. Tg/HDL ratio also reflects the balance between atherogenic and protective lipoproteins.

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The effect of Tg/HDL ratio on acute myocardial infarction (AMI) occurrence in young patient groups remains currently unknown. In the present study, we investigated the role of increased Tg/HDL on AMI occurrence in young age by comparing lipid panels of young AMI patients versus AMI patients that are older age.

Patients and methods

The patients were stratified into two groups by their age, those that were older than 50 years were put into the older patient group and those that were younger than 50 years old were put into younger patient group. The present retrospective study included 621 patients diagnosed with AMI [199 non-ST-segment elevation myocardial infarction (non-STEMI), and 422 ST-segment elevation myocardial infarction (STEMI)]. AMI cases were selected from admission to our emergency department, who underwent percutaneous coronary intervention between December 2015 and July 2016. Coronary angiographies were performed with standard Seldinger methods through femoral access. Acute myocardial infarction is defined: when there is acute myocardial injury with clinical evidence of acute myocardial ischemia and with detection of a rise and/or fall of cTn values with at least one value above the 99th percentile URL and at least one of the following: Symptoms of myocardial ischemia, New ischemic ECG changes, Develop-

ment of pathological Q waves, Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality in a pattern consistent with an ischemic etiology.

Exclusion criteria included previous CAD diagnosis, percutaneous or surgical revascularization, myocardial infarction normal coronary artery (MINOCA), previous treatment with any antihyperlipidemic agent, age <18 years, New York Heart Association Class III and Class IV heart failure, ongoing systemic inflammatory disease, renal or hepatic dysfunction, significant valvular disease, myocarditis, cardiomyopathies, malignancy and other pathologies that cause secondary hypertriglyceridemia.

Data collection

The clinical and demographic properties of the patients were recorded from hospital files and computer records. Baseline demographic parameters and glucose, urea, creatinine, troponin I were obtained during admission to the hospital. After an overnight fasting, blood samples for cholesterol profile were obtained within 24 h of admission to hospital. The blood samples for lipid profiles were analyzed using standard methods, without any delay. ECG was performed on admission and immediately after the procedure.

Baseline variables of the patients including age, gender, height, weight, and cardiovascular risk factors such as: hypertension, diabetes mellitus, dyslipidemia, current smoking, and renal failure were recorded from hospital files and computer records. Laboratory findings including lipid profile and cardiac markers, biochemistry, hemogram were recorded. The Tg/HDL-C ratio was calculated as Tg (mg/dL) divided by HDL-C (mg/dL).

The patient's information was collected from hospital files and computer records including medical history, diabetes, high blood pressure, body mass index, cigarette consumption. High blood pressure was defined as being on blood pressure medication or over the ≥130/80 by ambulatory blood pressure or over ≥140/90 as described. Body mass index was calculated as (weight in kilograms) / (height in meters)^2. Diagnosis of diabetes mellitus was defined as fasting plasma glucose > 126 mg/dL; or 2-hour plasma glucose > 200 mg/dL during an oral glucose tolerance test; or in a patient with classic symptoms of hyperglycemia or hyperglycemic crisis, a random plasma glucose > 200 mg/dL, a previously established diagnosis or previous treatment was also accepted. Smoking was determined as current or past history of smoking.

We performed the study according to declaration of Helsinki and this study was approved by the Local Medical Ethics Committee. Written informed consent was obtained from all the patients as needed before starting the study.

Statistical analysis

Continuous variables are expressed as the mean ± standard deviation. Categorical variables are expressed as percentages. To compare parametric continuous variables, Student's t-test was used, and the Mann–Whitney U was used to compare nonparametric continuous variables. The chi-square test was employed to compare categorical variables. Since the study was non-randomized and was a retrospective study, we used propensity score matching method in order to minimize selection bias. In order to balance patient characteristics and to generate a propensity-matched analysis for the 2 groups, a propensity score was created for the variables that are frequently used by clinicians to predict acute coronary syndromes (gender, diabetes, hypertension, smoking, MI type and family history of CAD). The propensity score was developed using SPSS, version 22.0 (IBM Corp., Armonk, NY, USA) for Windows and the R statistical package, R version 3.2.3 (R Foundation for Statistical Computing, Vienna, Austria). Two-tailed $p < 0.05$ were considered to indicate a statistical significance.

Results

The present study median Tg/HDL ratio of all patients enrolled in the study was 4.1 (2.6–5.9). Key clinical properties and laboratory results of all patients included in the study are summarized in (Tab. 1).

Baseline characteristics of younger and older MI groups are presented in Table 2. The unmatched analysis revealed that incidence of the classic risk factors, hypertension and diabetes mellitus, was lower among young patients with MI whereas smoking and family history of CAD were more frequent compared to the older patient group. Laboratory investigations indicated that total cholesterol, Tg levels, Tg/HDL ratio (Fig. 1) were higher in younger patients with MI while glucose and HDL levels were lower.

A propensity score matching was created with variables frequently used by clinicians, when predicting an acute coronary syn-

Tab. 1. Demographic properties, comorbidities and basic laboratory results of whole study´s patients.

Demographics	
Age (years)	54 (44–63)
Sex (male)(%)	79.9
Body mass index	27.3 (24.5–30.1)
Comorbidities	
Hypertension (%)	42.7
Diabetes Mellitus (%)	26.6
COPD (%)	1.0
Smoking (%)	62.5
Family history of CAD (%)	34.0
Baseline laboratory results	
Peak troponin I (ng/ml)	8.5 (1.4–35.0)
Urea (mg/dl)	35.0 (28.0–41.0)
Creatinine (mg/dl)	0.8 (0.7–1.0)
Total cholesterol (mg/dl)	184.0 (157.0–217.0)
LDL (mg/dl)	113.0 (91.5–137.0)
HDL (mg/dl)	38.0 (32.0–44.5)
Triglyceride (mg/dl)	153.0 (109.0–197.0)
Glucose (mg/dl)	110.0 (95.7–139.0)
Triglyceride/HDL ratio	4.1 (2.6–5.9)
WBC (10^3/µl)	10.3 (8.6–13.0)
Hemoglobin (g/dl)	13.6 (12.2–14.8)
Platelet (10^3/µl)	233.0 (193.0–287.0)
STEMI (n and %)	422–68.0%
NONSTEMI (n and %)	199–32.0%

CAD – coronary artery disease, COPD – chronic obstructive pulmonary disease, HDL – high density lipoprotein, NONSTEMI – non ST-segment elevation myocardial infarction, LDL – low density lipoprotein, STEMI – ST-segment elevation myocardial infarction, WBC – white blood cell. Data are expressed as the median (25th–75th percentages) and percentage (%).

Tab. 2. Basic characteristics of young and older myocardial infarction patients before propensity matching.

Pre match			
Baseline clinical properties	Young MI	Older MI	p
Age (years)	42.0 (38.0–46.0)	61.0 (55.0–68.0)	<0.001
Sex (male)	85.9	76.2	0.004
Body mass index	27.3 (25.3–30.3)	27.4 (24.4–30.1)	0.624
Hypertension (%)	22.6	54.8	<0.01
Diabetes Mellitus (%)	16.2	32.8	<0.01
COPD (%)	0.4	1.3	0.215
Smoking (%)	80.8	51.4	<0.01
Family history of CAD (%)	47.0	26.1	<0.01
Clinical presentation			
Anterior STEMI	57.4	47.0	
İnferior STEMI	26.5	24.4	0.152
NONSTEMI	16.1	18.6	
Baseline laboratory results			
Peak troponin I (ng/ml)	11.5 (2.5–49.2)	8.3 (1.1–31.0)	0.207
Urea (mg/dl)	30.9 (24.0–36.0)	36.4 (29.8–43.0)	<0.001
Creatinine (mg/dl)	0.8 (0.7–0.9)	0.8 (0.7–1.0)	0.002
Total cholesterol (mg/dl)	189.5 (159.0–225.2)	182.0 (156.0–213.0)	0.047
LDL (mg/dl)	117.0 (92.0–140.0)	112.0 (91.0–135.0)	0.175
HDL (mg/dl)	32.0 (26.0–38.0)	40.0 (35.0–47.0)	<0.001
Triglyceride (mg/dl)	181.0 (145.0–247.0)	129.0 (88.0–173.0)	<0.001
Glucose (mg/dl)	104.0 (92.0–128.0)	114.0 (100.0–151.0)	<0.001
Triglyceride/HDL ratio	5.7 (4.2–9.1)	3.1 (2.0–4.6)	<0.001
WBC (10^3/μl)	11.0 (9.0–13.3)	10.0 (8.3–12.4)	0.002
Hemoglobin (g/dl)	14.0 (12.9–15.0)	13.2 (12.0–14.5)	<0.001
Platelet (10^3/μl)	252 (214.0–301.0)	220.0 (183.0–274.0)	<0.001

CAD – coronary artery disease, COPD – chronic obstructive pulmonary disease, HDL – high density lipoprotein, LDL – low density lipoprotein, NONSTEMI – non ST-segment elevation myocardial infarction, STEMI – ST-segment elevation myocardial infarction, WBC – white blood cell. Data are expressed as the median (25th–75th percentages) and percentage (%). $p < 0.05$ was considered statistically significant.

drome risk for individual patients (gender, diabetes, hypertension, smoking, MI type and family history of CAD) in order to balance patient characteristics and to generate a propensity-matched analysis for the 2 groups (Tab. 3). In the matched population, while young MI patients were compared to older MI patients, serum Tg levels [179 (145–231) vs 148 (101–197)] and Tg/HDL ratio [5.8 (4.1–9.1) vs 3.0 (1.8–4.6)] were significantly higher whereas HDL levels were observed as dramatically lower (32.6 ± 8.2 vs 41.7 ± 8.8).

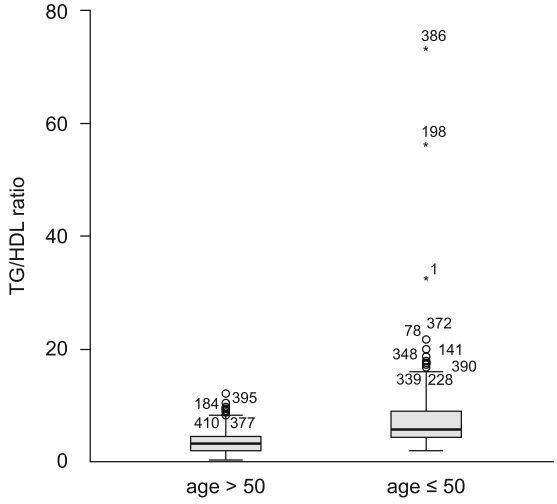

Fig. 1. Triglyceride high density lipoprotein ratio (Tg/HDL) of young and older myocardial infarction patients. The x-axis of this graph shows the Tg/HDL ratios and the y-axis shows the age groups (age < 50 as young, age ≥ 50 as older patients).

The analysis performed after propensity score matching revealed that there was a significant difference between young and older MI groups in terms of Tg, HDL levels and Tg/HDL ratio.

Discussion

The results of the present study indicated that increased Tg/HDL ratio was associated with the risk of myocardial infarction in young adults based on the propensity score analysis. The risk of MI in young individuals with high Tg/HDL ratio was increased regardless of serum total cholesterol levels and low-density lipoprotein.

Tg rich lipoprotein cholesterol in post hoc analysis of TNT study showed that patients with coronary artery disease, who had higher TG rich lipoprotein levels were at a higher risk of major cardiac adverse events even if they had the same LDL levels (10 circ). Latest studies showed that in addition to lowering TG levels, trying to increase the HDL levels in treatment might not be as useful as previously thought, it might even worsen the treatment (11–13). Subtyping the HDL levels and planning treatment according to HDL subtype may prove better results in near future. (14). These results showed that TG or HDL might not be the only

Tab. 3. Basic characteristics of young and older myocardial infarction patients after propensity matching.

Post match			
	Young MI (n:154)	Older MI (n: 154)	p
Baseline clinical properties			
Age (years)	40.8 ± 6.2	60.4 ± 7.4	<0.001
Sex (male)	85.1	85.1	1.000
Body mass index	27.4 ± 4.6	25.7 ± 4.2	0.017
Hypertension (%)	29.2	28.6	0.900
Diabetes Mellitus (%)	19.5	18.8	0.885
COPD (%)	0.8	1.7	0.511
Smoking (%)	77.9	77.3	0.891
Family history of CAD (%)	35.7	36.4	0.906
Clinical presentation			
Anterior STEMI	37	36.4	0.989
İnferior STEMI	40.3	40.3	
NONSTEMI	22.7	23.4	
Baseline laboratory results			
Peak troponin I (ng/ml)	6.9 (1.2–18.6)	14.5 (1.92–50.0)	0.114
Urea (mg/dl)	29.3 ± 8.1	35.0 ± 11	0.002
Creatinine (mg/dl)	0.76 ± 0.15	0.79 ± 0.20	0.486
Total cholesterol (mg/dl)	194.0 ± 49.0	187.0 ± 42.0	0.211
LDL (mg/dl)	123 (94–153)	119 (98–141)	0.267
HDL (mg/dl)	32.6 ± 8.2	41.7 ± 8.8	<0.001
Triglyceride (mg/dl)	179 (145–231)	148 (101–197)	<0.001
Glucose (mg/dl)	122.0 ± 58	137.0 ± 69	0.001
Triglyceride/HDL ratio	5.8 (4.1–9.1)	3.0 (1.8–4.6)	<0.001
WBC (10^3/µl)	10.9 ± 3.1	10.9 ± 3.7	0.852
Hemoglobin (g/dl)	13.8 ± 1.7	13.4 ± 1.7	0.023
Platelet (10^3/µl)	262.0 ± 88.0	235.0 ± 78	0.003

CAD – coronary artery disease, COPD – chronic obstructive pulmonary disease, HDL – high density lipoprotein, LDL – low density lipoprotein, NONSTEMI – non ST-segment elevation myocardial infarction, STEMI – ST-segment elevation myocardial infarction, WBC – white blood cell. Data are expressed as the median (25th–75th percentages), the mean ± standard deviation and percentage (%). $p < 0.05$ was considered statistically significant.

treatment parameters and maybe statins combined with pcsk9 inhibitor or ezetimib will allow us to reach the LDL goal or reduce the relative risk especially in young adults.

With such an active role in atherogenesis, the Tg/HDL ratio is known to predict cardiovascular events both in healthy individuals and patients with CAD. In healthy individuals, the relationship between this ratio and insulin resistance, cardiometabolic risk and cardiovascular disease was shown in the previous studies (15, 16–19).

When Tg and HDL are considered separately, increased Tg and decreased HDL was shown to predict cardiovascular disease, independent from LDL levels (20).

Determining the risk factors and adequate management is one of the corner stones of preventing coronary artery disease (CAD). Age, smoking, hypertension, diabetes mellitus, hypercholesterolemia are the most acclaimed risk factors; however, they may fall short when determining the definite cardiovascular risk in young adults. Atherogenesis, which is the most common cause of CAD pathogenesis, has an established association with dyslipidemia. Management of dyslipidemia targets the apo-B containing LDL (21). In patients with high risk, a residual cardiovascular risk persists despite a successful LDL lowering with statins (22, 23). Tg/HDL ratio appears as a valuable parameter for identifying this residual risk. Tg/HDL ratio has been shown to be a strong predictor of cardiovascular disease, independent from age, smoking, hypertension, diabetes mellitus and cholesterol (15).

Typical plaque rupture is rare in young patients with ACS (24, 25); therefore, other risk factors apart from the conventional risk factors should be taken into account in these patients. Smoking, family history, obesity and hypercholesterolemia were more common in our patient group of young MI cases. In the lipid analysis, particularly Tg and total cholesterol levels were distinctly higher, while HDL levels were significantly lower, and no difference was observed in terms of LDL levels. These findings are consistent with the studies reporting that insulin and hypertriglyceridemia are also important risk factors in patients < 50 years of age while smoking, obesity, hypercholesterolemia and male gender tend to be more common (26). In young patients with ACS, hypertriglyceridemia has been associated with premature CAD (27–29) and Tg/HDL ratio was also held responsible for increased arterial stiffness (30). Although the mechanisms leading to the effect on the pathogenesis of atherosclerosis are currently unclear, some relevant mechanisms of Tg/HDL ratio are; Tg/HDL ratio is associated with insulin resistance, reflects the balance between atherogenic and protective lipoproteins (31, 32) and also Tg/HDL ratio triggers inflammatory mechanisms.

In our study, despite the fact that LDL was not different in the young MI group, the higher Tg/HDL ratio and lower HDL levels were critical and essential findings. Our findings are supported by studies, which demonstrated that higher triglyceride levels and lower HDL levels are common in patients with ACS. However, previous studies demonstrated that patients with lower HDL and

higher TG levels are still under higher ACS risk even though they receive a lipid-lowering treatment, when compared to the control group. Tg or HDL alone are significant predictors of ACS in patients with cardiovascular disease (20), the combination of these parameters as Tg/HDL may be more valuable than utilizing these two separately in order to determine the cardiovascular risk in these patients. Taken together, the present study results suggest that in young individuals Tg/HDL are not only a cardiovascular risk marker, but also a potential target for therapeutic intervention.

Conclusion

In our study, our findings have given us the idea that the Tg/HDL ratio can be used to predict the occurrence of acute coronary syndrome in young adults. Especially in adults under the age of 50, Tg/HDL ratio may be determined and necessary precautions might be taken to prevent ACS.

Limitations

The number of patients in our single-centered study is relatively low. Since it was not a prospective study, no patients were followed up and healthy people were not taken as a control group. Increased thrombogenicity associated with an increased susceptibility to acute coronary syndrome in young people has not been examined for genetic and laboratory tests. During first week of acute myocardial infarction, lipid levels may be measured differently, however, since both of the groups' lipid levels were obtained at the same time frame after the infarction, the difference effects both groups.

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Received August 14, 2018.
Accepted September 18, 2018.