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Neutrophil–lymphocyte ratio is associated with low high-density lipoprotein cholesterol in healthy young men

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Abstract

Objective: It has been reported that the neutrophil–lymphocyte ratio is significantly elevated in patients with low high-density lipoprotein cholesterol (<35 mg/dL). But in this study, some patients had hypertension that may have affected the neutrophil–lymphocyte ratio. This study consisted of 1274 asymptomatic healthy young men. In contrast with the previous study, we investigated the neutrophil–lymphocyte ratio in healthy young men with low high-density lipoprotein cholesterol compared with controls.

Methods: We studied 1274 asymptomatic young males (military personnel screening) who underwent routine health check-up. Of them, 102 subjects had low high-density lipoprotein cholesterol.

Results: The neutrophil–lymphocyte ratio was significantly higher among the men with low high-density lipoprotein cholesterol than that of the control group ($P < 0.001$).

Conclusion: We conclude that the neutrophil–lymphocyte ratio is significantly elevated in asymptomatic healthy young men with low high-density lipoprotein cholesterol compared with control participants.

Keywords

Neutrophil–lymphocyte ratio, high-density lipoprotein cholesterol, inflammation

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Introduction

A low level of high-density lipoprotein cholesterol (HDL-C) is an important independent risk factor of cardiovascular disease.[1,2] The antiatherosclerotic effects of HDL occur in several ways. HDL reverses cholesterol transport, reduces inflammation, promotes endothelial cell nitric oxide production, and inhibits low-density lipoprotein cholesterol (LDL-C) oxidation, endothelial cell apoptosis, platelet activation, and expression of adhesion molecules.[3–6]

The neutrophil–lymphocyte ratio (NLR) is a universally available laboratory marker used to evaluate systemic inflammation.[7,8] The NLR is related to the severity of coronary heart disease (CHD) and clinical outcome in patients undergoing angiography as well as angiographic progression of coronary atherosclerosis. The NLR is also an independent predictor of adverse outcomes among patients with ST-segment elevation myocardial infarction undergoing primary percutaneous coronary intervention.[9,10] Varol et al.[11] have reported that the NLR is significantly elevated in patients with low HDL-C (<35 mg/dL). In this study, some patients had hypertension and that may have affected the NLR.[11,12] For example, Fici et al.[12] showed that nebivolol and metoprolol decreased NLR in patients with newly diagnosed essential hypertension. Therefore, we investigated the NLR in healthy young men with low HDL-C compared with controls.

Methods

Subjects

A cross-sectional, retrospective, observational analysis was conducted among asymptomatic healthy young men (military personnel screening). Between July 2012 and July 2013,

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Table 1. Clinical and laboratory characteristics of the subjects with low HDL-C (<35 mg/dL) and the control group.

	Low HDL-C group	Control group	P
n	102	1172	
Age, years	22.1 + 2.0	21.6 + 3.1	.17
BMI, kg/m^2	22.6 + 1.8	21.7 + 1.7	.81
Smoking, %	15	19	.33
Glucose, mg/dL	90 + 4	90 + 5	.18
Creatinine, mg/dL	1.01 + 0.1	1.00 + 0.22	.06
Total cholesterol, mg/dl	146 + 35	147 + 24	.38
Triglycerides, mg/dL	124 + 69	122 + 36	.19
LDL-C, mg/dL	102 + 17	101 + 15	.16
WBC, ×1000 cells/mm^3	6.17 + 1.92	6.28 + 1.58	.32
Hemoglobin, g/dL	14.5 + 1.2	14.5 + 1.1	.15
RDW, %	14.3 + 1.2	14.3 + 1.6	.11
Neutrophils, ×1000 cells/mm^3	4.7 + 1.3	3.6 + 1.3	<.001
Lymphocytes, ×1000 cells/mm^3	1.2 + 0.6	2.4 + 0.5	<.001
NLR	3.7 + 0.7	1.5 + 0.4	<.001

BMI: body mass index; LDL-C: low-density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol; WBC: white blood cells; RDW: red cell distribution view; NLR: neutrophil–lymphocyte ratio.

1274 asymptomatic healthy young males underwent routine health check-up. All were submitted to a standard protocol that included a complete clinical examination, laboratory evaluation, and 12-lead electrocardiogram (ECG). Of them, 102 subjects had low HDL-C (<35 mg/dL). Exclusion criteria were hypertension, history of stroke, valvular heart disease, heart failure, peripheral arterial disease, diabetes mellitus, renal or hepatic dysfunction, hematological disorders, history of malignancy, acute or chronic infection, and drug use affecting HDL-C and NLR, and whose laboratory tests were measured when enrolled in this study. The control group consisted of 1172 subjects. An average systolic blood pressure (SBP) and diastolic blood pressure (DBP) was calculated from two measurements taken with the subjects in a sitting position after a 5-min rest period. Hypertension was considered to be present, if the SBP was >140 mmHg and/or DBP was >90 mmHg.

This was a cross-sectional, retrospective, observational, and anonymized analysis. So according to our rules, there was no need for the approval by the institutional ethics committee.

Laboratory measurements

Height and body weight were measured barefoot wearing light clothing. The body weight was measured with the subjects wearing light clothes provided by our center and the weight of the clothing was subtracted from the measured body weight. The body mass index (BMI) was calculated as the weight in kilograms divided by the square of the height in meters. After an overnight fast, blood samples were obtained to measure routinely evaluated laboratory values: plasma glucose, triglycerides, HDL-C, LDL-C, hemoglobin (Hb), blood cell counts, and liver and kidney function tests. The simple qualitative urinalyses were performed with test strips. The biochemical measurements were all performed at Diyarbakır Military Hospital (DMH), Diyarbakır, Turkey, using routine laboratory methods. Diabetes mellitus was defined as a fasting blood glucose level >126 mg/dL. Patients who were smoking before hospitalization were classified as smokers.

Statistical analysis

Data were analyzed with the SPSS software version 15.0 for Windows. Continuous variables from the study groups were reported as mean + standard deviation and categorical variables as percentages. To compare continuous variables, the Student's t-test was used, as appropriate. Categorical variables were compared using the chi-square test. A two-sided $P < 0.05$ was considered significant. Analysis of covariance (ANCOVA) was used to analyze the confounding effects of variables on the NLR. The variables for ANCOVA were smoking and BMI because smoking and BMI can affect the HDL level.[13,14]

Results

Comparisons of the clinical and laboratory characteristics of those with low HDL-C and the control group were shown in Table 1. There were no significant differences between the two groups with respect to age; gender; BMI; smoking; glucose, creatinine, total cholesterol, triglyceride, LDL-C, and Hb levels; white blood cell count; and red cell distribution width.

Neutrophil count was significantly higher among the patients with low HDL-C than that of the control group ($P < 0.001$). Lymphocyte count was significantly lower among the patients with low HDL-C than that of the control

group (P < 0.001). The NLR was significantly higher among the patients with low HDL-C than that of the control group (3.7 + 0.7 vs 1.5 + 0.4, P < 0.001), even after data adjustment for confounders (3.5 + 0.8 vs 1.5 + 0.4, P < 0.001).

Discussion

This study consisted only of asymptomatic healthy young men. The study by Varol et al.[11] is similar to ours since their study population also consisted of healthy participants. However, their study included patients with hypertension that may affect the NLR.[12]

The exact cause of increased NLR in those with low HDL-C is not known. The NLR is a combination of two independent markers of inflammation and general health status.[15] The combination of these two markers, the NLR, has proved to be a useful simple marker of inflammation.[16]

We conclude that the NLR is significantly elevated in asymptomatic healthy young men with low HDL-C compared with control participants.

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Declaration of conflicting interests

There is no conflict of interest.

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