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Evaluation of lipid parameters and correlations in patients with clinical atherosclerosis

Klinik aterosklerozlu hastalarda lipid parametrelerinin ve korelasyonlarının değerlendirilmesi

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BACKGROUND
We aimed to evaluate lipid profiles, atherosclerotic index ratios, and correlations as early diagnostic biomarkers for cardiovascular atherosclerotic diseases.
METHODS
The study included 30 individuals with clinical atherosclerosis who underwent coronary artery bypass grafting (CABG) and 30 healthy individuals without a history of cardiovascular disease. Routine lipid profile values of the individuals included in the study were obtained from the hospital database, and non- high-density lipoprotein (HDL) cholesterol, non-HDL/HDL, triglyceride (TG)/glucose, and TG/HDL ratios were calculated from these values. The accepted level of statistical significance was $p<0.05$. Comparisons between groups were conducted using the Mann-Whitney U test.
RESULTS
Clinical atherosclerosis patients demonstrated statistically higher levels of TG, non-HDL cholesterol, and very low-density lipoprotein (VLDL) cholesterol, whereas HDL cholesterol was statistically lower in comparison to the healthy individuals. Furthermore, the patient group demonstrated elevated TG/HDL, TG/glucose, and non-HDL/HDL ratios, while total cholesterol and low-density lipoprotein (LDL) cholesterol levels remained statistically insignificant.
CONCLUSION
It is important for a biomarker or method to be noninvasive rather than invasive. We suggest that atherosclerotic index rates, in addition to routine lipid parameters, have the potential to predict asymptomatic individuals or the severity of atherosclerosis. However, it is necessary to conduct studies with larger samples.

KEYWORDS
Atherosclerosis, diagnostic biomarker, lipid parameters.

ÖZ

AMAÇ
Kardiyovasküler aterosklerotik hastalıklar için erken tanı biyobelirteçleri olarak lipid profillerini, aterosklerotik indeks oranlarını ve korelasyonlarını değerlendirmeyi amaçladık.
YÖNTEM
Çalışmaya koroner arter baypas greft (KABG) cerrahisi kararı verilmiş olan aterosklerozlu 30 birey ve kardiyovasküler hastalık öyküsü olmayan 30 sağlıklı birey dahil edildi. Çalışmamıza dahil edilen bireylerin rutin lipid profili değerleri hastane veri tabanından elde edildi ve bu değerlerden non-yüksek dansiteli lipoprotein (HDL) kolesterol, non-HDL/HDL, trigliserit (TG)/glukoz ve TG/HDL oranları hesaplandı. İstatistiksel anlamlılık düzeyi $p < 0,05$ olarak kabul edilmiştir. Mann-Whitney U testi gruplar arası karşılaştırma için kullanıldı.
BULGULAR
Klinik ateroskleroz hastalarında sağlıklı bireylere kıyasla TG, non-HDL kolesterol, çok düşük dansiteli lipoprotein (VLDL) kolesterol düzeyleri istatistiksel olarak daha yüksek, HDL kolesterol ise istatistiksel olarak daha düşük bulundu. Ayrıca, TG/HDL, TG/glukoz ve non-HDL/HDL oranları hasta grubunda istatistiksel olarak daha yüksek bulunurken, total kolesterol ve düşük dansiteli lipoprotein (LDL) kolesterol anlamlı farklılık göstermemiştir.
SONUÇ
Bir biyobelirteç veya yöntemin invazif olmaktan ziyade noninvazif olması önemlidir. Rutin lipid parametrelerine ek olarak aterosklerotik indeks oranlarının asemptomatik bireyleri veya aterosklerozun şiddetini tahmin etme potansiyeline sahip olduğunu düşünüyoruz. Ancak, daha büyük örneklemlerle çalışmalar yapılması gerekmektedir.

ANAHTAR KELİMELER
Ateroskleroz, lipid parametreleri, tanısal biyobelirteç.

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Cardiovascular diseases, such as arrhythmia, hypertension, myocardial infarction, atherosclerosis, myocardial ischemia, and heart failure, result in the deaths of around 17.9 million individuals annually (32% of all deaths) (1,2). Atherosclerosis is a chronic condition characterized by a buildup of lipids under the inner layer of the arteries. This leads to the narrowing of the artery, which is the main cause of cardiovascular disorders (3,4). Atherosclerosis initiates with endothelial activation, which is followed by a sequence of occurrences (lipid deposition, fibrous components, and calcification) that stimulate the activation of inflammatory pathways and narrowing of the arterial lumen. The formation of atheroma plaque resulting from these events contributes to cardiovascular complications (5). Atherosclerotic plaques are soft, yellow, clot-like lesions consisting of a fatty ball made up of cholesterol and its esters, together with necrotic debris. These plaques are surrounded by a fibrous cap and grow towards the lumen (6).

Angiogenesis, inflammatory and immunological responses, lipid metabolism, apoptosis, and cell proliferation are all important biological processes that significantly contribute to the formation and progression of atherosclerosis (7). The main processes involved include endothelial injury, proliferation, and miggration of vascular smooth muscle cells (VSMCs), phenotypic changes in VSMCs, production of foam cells, inflammatory reactions, and problems in lipid metabolism. Furthermore, other cellular components and molecules, including vascular endothelial cells, mast cells, platelets, macrophages, and inflammatory cytokines, actively participate in the development and progression of atherosclerosis (3,8,9).

Dyslipidaemia is characterised by high levels of atherogenic lipoproteins/lipids or impaired function of anti-atherogenic lipids or lipoproteins (10). LDL cholesterol is the most important type of cholesterol that contributes to the development of atherosclerosis, although VLDL cholesterol is also atherogenic because it is the main carrier of triglycerides. HDL cholesterol, known as 'good cholesterol', is not atherogenic. The combined levels of LDL and VLDL cholesterol, referred to as non-HDL cholesterol, exhibits greater atherogenicity than each lipoprotein alone. Furthermore, apoB, the primary apolipoprotein present in LDL and VLDL, is a very strong indicator of atherogenicity (11). A research study has shown that a 10% increase in LDL cholesterol raises the risk of getting cardiovascular disease by 20% (12). Additionally, individuals who have been exposed to borderline/slightly raised levels of LDL cholesterol for decades is a 3 to 4 times greater risk of developing atherosclerotic disease compared to those with consistently low levels of LDL cholesterol (13). Dyslipidaemia frequently coexists with hypertension, and together they have a synergistic effect on the occurrence of atherosclerotic events. Hypertension and dyslipidemia are independent factors that increase the risk of atherosclerosis. Atherosclerosis is also a risk factor for hypertension and other cardiovascular diseases (14). Hyperlipidaemia can contribute to the development of diabetes by increasing blood glucose levels, hence promoting the formation of plaque and damage to the endothelium layer (15). Hyperlipidaemia is also known to elevate levels of reactive oxygen species.

In light of the available knowledge, the objective of this study was to investigate lipid profiles and their correlations in patients with clinical atherosclerosis.

Materials and Methods

The study included volunteers who completed informed consent forms and agreed to participate. Patients admitted to the University of Istanbul Hospital, Department of Cardiovascular Surgery, who were decided to undergo bypass based on clinical and laboratory evaluations were included in our study. Some of the individuals comprising this patient group had been using statins and other medications (such as beta-blocker, anticoagulant) prior to CABG surgery. Individuals without any cardiovascular disease were included in the control group. The HDL cholesterol, LDL cholesterol, non-HDL cholesterol, VLDL cholesterol, total cholesterol, and TG data were obtained from the hospital database of the patient (n=30) and control (n=30) groups. TG/HDL, non-HDL/HDL, and TG/glucose ratios were calculated from these data. The study was conducted with the approval of the University of Health Sciences Türkiye Scientific Research Ethics Committee, under approval number 2021/36-2. The research was conducted in accordance with the principles outlined in the Declaration of Helsinki.

Statistical analysis of the findings and data obtained was performed with tatistical Package for the Social Sciences (SPSS) 23.0 programme (IBM Corp, Chicago, IL, USA). The Mann-Whitney U test was used for non-parametric distribution of binary independent variables, Spearman's correlation analysis was used for correlation between all lipid profile parameters. $P < 0.05$ was accepted as significance level.

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Results

Eight of the participants were female (26.67%) and 22 were male (73.33%) in the patient group, while 10 of the healthy individuals were female (33.33%) and 20 were male (66.67%) ($p = 0.573$). The mean age of the patient group was 62.3 ± 9.42 years, while the mean age of the control group was 59.40 ± 6.01 years ($p = 0.118$).

In the patient group, 8 individuals (26.67%) suffered an acute myocardial infarction (MI). There were no significant statistical differences in terms of age and gender between those patients with and without MI ($p > 0.05$). The levels of HDL cholesterol and the non-HDL/HDL ratio were significantly lower in individuals who had a MI compared to those who did not ($p = 0.018$ and $p = 0.005$, respectively). DM was present in 16 of individuals in the patient group. The statistical analyses regarding the presence or absence of DM indicated no significant differences in terms of age and gender ($p > 0.05$). The patients with DM had significantly lower levels of HDL cholesterol, significantly higher non-HDL/HDL level, and significantly higher TG/HDL ratio compared to those without DM ($p = 0.016$, $p = 0.003$, $p = 0.035$, respectively). However, no significant difference was found between the presence/absence of DM and the TG/glucose ratio ($p = 0.379$)

The laboratory parameters were analyzed between the patient and control groups utilizing the Mann-Whitney U test. The patient group demonstrated highergreater levels of total cholesterol, TG, LDL cholesterol, non-HDL cholesterol, VLDL cholesterol, TG/HDL, TG/glucose, and non-HDL/HDL, whereas HDL cholesterol level was lower in comparison to the control group. However, elevated total cholesterol and LDL cholesterol levels were no statistically insignificant between the patient and control groups (Figure 1, Table 1).

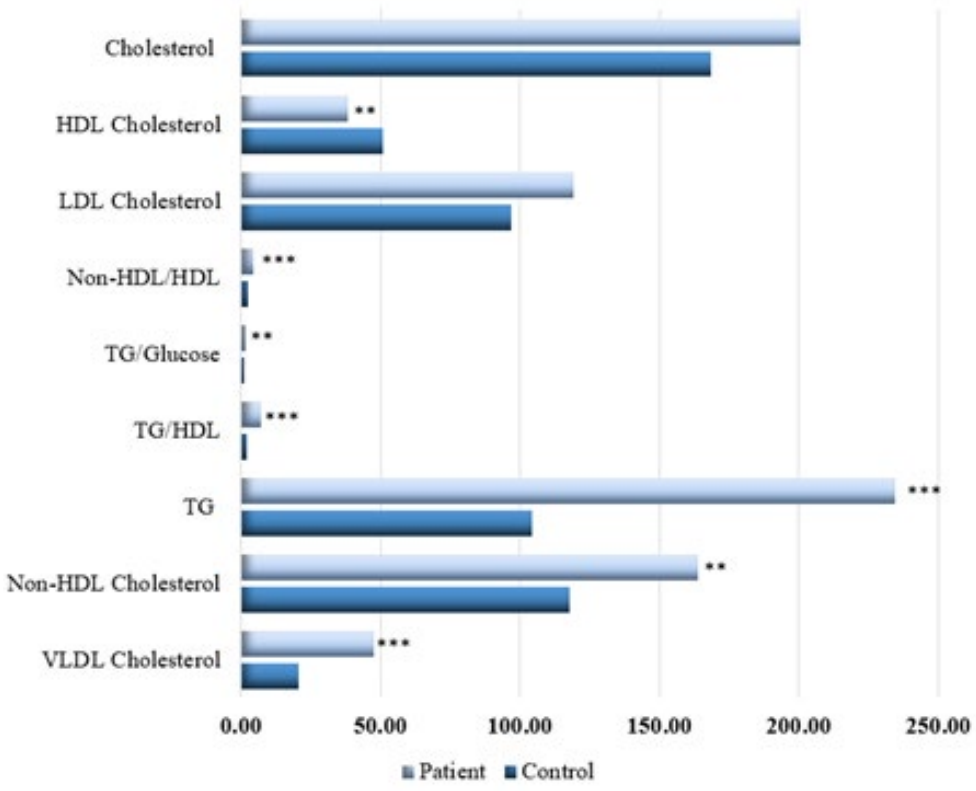


Figure 1. The lipid profiles between the patient and control groups
HDL, High-density lipoprotein; LDL, Low-density lipoprotein; TG, Triglyceride; VLDL, Very low-density lipoprotein
** $p < 0.01$; *** $p < 0.001$

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Table 1. The lipid profiles between the patient and control groups

Parameter	Patient	Control	p value
Cholesterol (mg/dL)	189 [161-241] (80-338)	174.5 [150.5-188] (96-238)	0.074
HDL cholesterol (mg/dL)	34.5 [32-46] (23-64)	48.5 [43.5-56.5] (29-89)	**0.001**
LDL cholesterol (mg/dL)	106 [91.5-141.5] (36-238)	98 [79-119.5] (26-156)	0.130
TG (mg/dL)	178.5 [137.5-272.5] (102-876)	81.5 [72.5-139] (44-223)	**<0.001**
Non-HDL cholesterol (mg/dL)	145.5 [121.5-210.5] (57-291)	118 [96.5-141.5] (37-189)	**0.004**
Non-HDL/HDL	3.91 [2.745-6.06] (1.33-10.38)	2.38 [1.735-3.095] (0.63-5.79)	**<0.001**
VLDL cholesterol (mg/dL)	36 [27.5-54.5] (20-175)	16.3 [14.5-27.8] (8.8-44.6)	**<0.001**
TG/HDL	5.1 [3.10-8.135] (1.63-28.26)	1.66 [1.23-3.14] (0.91-7.31)	**<0.001**
TG/glucose	1.415 [1.065-2.22] (0.56-3.05)	0.86 [0.72-1.365] (0.42-2.75)	**0.003**

HDL, High-density lipoprotein; LDL, Low-density lipoprotein; TG, Triglyceride; VLDL, Very low-density lipoprotein

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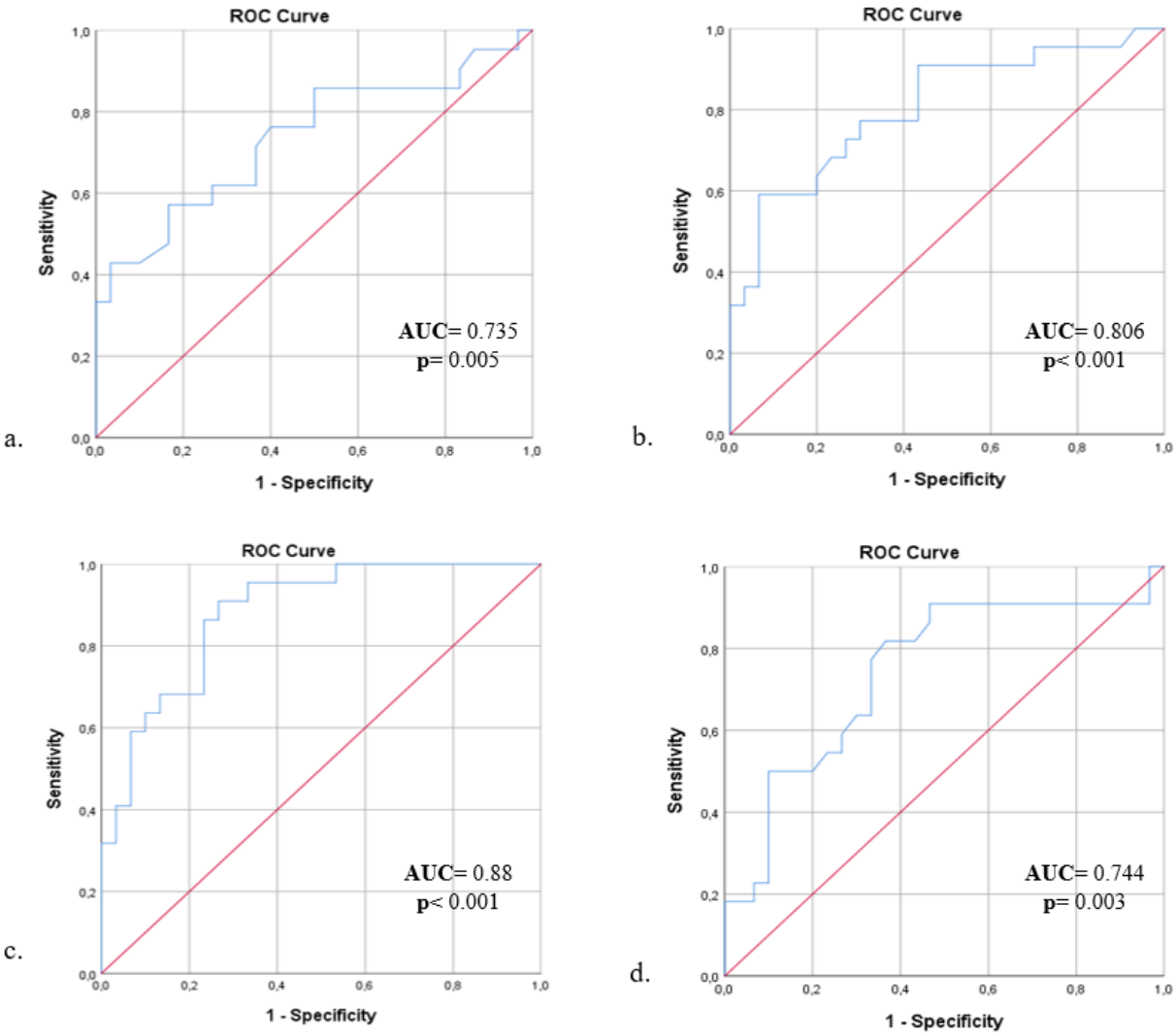


Figure 2. The ROC curve analysis of lipid parameters/ratios
a. Non-HDL, b. Non-HDL/HDL, c. TG/HDL, d. TG/glucose. HDL: High-density lipoprotein, TG: Triglyceride
HDL, High-density lipoprotein; TG, Triglyceride

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Table 2. Gender-based comparison between the patient and control groups

Female			
Parameter	**Patient**	**Control**	**p value**
Total cholesterol (mg/dL)	173 [159.75-204.5] (159-236)	181 [137.25-187.75] (96-213)	0.664
HDL cholesterol (mg/dL)	41.5 [33.75-46.25] (33-50)	56.5 [48-71.75] (44-89)	**0.008**
LDL cholesterol (mg/dL)	96 [88-107.75] (85-134)	88.5 [60.75-111.75] (26-134)	0.384
TG (mg/dL)	189.5 [146.5-288.25] (130-313)	80 [70.75-129.75] (56-223)	**<0.005**
Non-HDL (mg/dL)	136.5 [115-161.75] (115-197)	115 [76.75-135.25] (37-150)	0.092
Non-HDL/HDL	3.395 [2.598-4.57] (2.56-5.05)	2 [1.223-2.463] (0.63-3.23)	**0.007**
VLDL cholesterol (mg/dL)	38 [29-57.75] (26-63)	16 [14.15-25.95] (11.2-44.6)	**<0.005**
TG/HDL	4.59 [3.273-8.083] (2.95-8.24)	1.41 [0.995-2.643] (0.91-4.55)	**0.003**
TG/glucose	1.415 [0.988-1.6] (0.56-1.96)	0.86 [0.808-1.295] (0.71-1.41)	0.092
Male			
Parameter	**Patient**	**Control**	**p value**
Total cholesterol (mg/dL)	198.5 [159.25-258.75] (80-338)	166 [157.25-188.5] (115-238)	0.077
HDL colesterol (mg/dL)	34 [29.5-45.75] (23-64)	46 [40.25-50.5] (29-76)	**0.031**
LDL cholesterol (mg/dL)	117.5 [90.5-147.75] (36-238)	100 [86-123] (55-156)	0.202
TG (mg/dL)	178.5 [134.25-264.75] (102-876)	82.5 [70.25-149] (44-212)	**0.001**
Non-HDL	159.5 [125.75-229.5] (57-291)	119.5 [101-141.75] (72-189)	**0.030**
Non-HDL/HDL	4 [3.03-7.158] (1.33-10.38)	2.67 [2.148-3.345] (1.45-5.79)	**0.004**
VLDL	36 [26-53] (20-175)	16.5 [14.05-29.8] (8.8-42.4)	**0.001**
TG/HDL	5.53 [3.05-8.463] (1.63-28.26)	1.805 [1.44-3.405] (0.96-7.31)	**0.001**
TG/glucose	1.45 [1.053-2.705] (0.56-3.05)	0.89 [0.703-1.383] (0.42-2.75)	**0.014**

HDL, High-density lipoprotein; LDL, Low-density lipoprotein; TG, Triglyceride; VLDL, Very low-density lipoprotein

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When a gender-based comparison was made between the patient and control groups, statistically significant differences were observed in HDL cholesterol, TGs, VLDL, TG/HDL level, and non-HDL/HDL level in the female sex. In male gender, HDL cholesterol, TG, non-HDL, VLDL, TG/HDL, TG/glucose, and non-HDL/HDL levels were also statistically significant (Table 2).

ROC curve were used to assess the diagnostic values of the potential patients. The results of ROC curve analysis of non-HDL, non-HDL/HDL, TG/HDL, and TG/glucose parameters, which were significant between the two groups, are given in Figure 2. According to the ROC curve analysis of the non-HDL value, the Area Under the Curve (AUC) was calculated as 0.735. The cut-off value was calculated as 127.5 with a sensitivity of 0.762 and a specificity of 0.6. According to the ROC curve analysis of the Non-HDL/HDL ratio, the AUC value was calculated as 0.806. The cut-off value was calculated as 2.77 with a sensitivity of 0.773 and specificity of 0.667. According to the ROC curve analysis of the TG/HDL ratio, the AUC was calculated as 0.88. The cut-off value was calculated as 2.9205 with 0.864 sensitivity and 0.767 specificity. According to the ROC curve analysis of TG/glucose ratio, AUC was calculated as 0.744. The cut-off value was calculated as 1.01 with 0.818 sensitivity and 0.633 specificity.

The relationship between lipid parameters was analyzed using Spearman's Correlation analysis. The findings, including correlation coefficients and significant values, are presented in Table 3.

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Table 3. The correlation between lipid parameters

Parameter	Parameter	r	p value
Total cholesterol	LDL cholesterol	0.947	<0.001
	TG	0.520	0.013
	Non-HDL cholesterol	0.986	<0.001
	VLDL cholesterol	0.519	0.016
	TG/HDL	0.449	0.036
	TG/Glucose	0.760	<0.001
	Non-HDL/HDL	0.758	<0.001
HDL cholesterol	TG/HDL	-0.509	0.020
	Non-HDL/HDL	-0.607	0.003
LDL cholesterol	TG/Glucose	0.613	0.002
	Non-HDL cholesterol	0.942	<0.001
	Non-HDL/HDL	0.741	<0.001
TG	Non-HDL cholesterol	0.565	0.008
	VLDL cholesterol	1.000	<0.001
	TG/Glucose	0.649	0.001
	TG/HDL	0.967	<0.001
	Non-HDL/HDL	0.626	0.002
TG/Glucose	Non-HDL cholesterol	0.741	<0.001
	VLDL cholesterol	0.648	0.001
	TG/HDL	0.551	0.008
	Non-HDL/HDL	0.512	0.015
Non-HDL cholesterol	VLDL cholesterol	0.568	0.007
	TG/HDL	0.525	0.014
	Non-HDL/HDL	0.846	<0.001
Non-HDL/HDL	VLDL cholesterol	0.626	0.002
	TG/HDL	0.706	<0.001
VLDL cholesterol	TG/HDL	0.967	<0.001

HDL, High-density lipoprotein; LDL, Low-density lipoprotein; TG, Triglyceride; VLDL, Very low-density lipoprotein

Discussion

Despite significant advances in atherosclerosis research, current treatments are inadequate in effectively managing this disease. It is reported that currently available drugs are unable to prevent even 70% of clinical events from occurring. Early detection of coronary atherosclerosis is critical to prevent plaque rupture and sudden death. Regardless of the effectiveness of cholesterol level management, at least 20% of patients have recurrent MI within 3 years of the first clinical manifestation and therefore other therapies are urgently needed to reduce atherosclerosis-related morbidity and mortality (16).

Enhanced turnover of endothelial cells, disturbance of gap junctions, heightened inflammation and oxidation might impair the intactness of the endothelium, leading to an increase

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in endothelial permeability and facilitating the entry of lipoproteins and inflammatory monocytes. During atheroma plaque development, VSMCs transition from a contractile phenotype to a synthetic phenotype and accumulate in the intima via proliferation or migration, significantly contributing to atherosclerosis progression (8,17). When arterial branches are damaged, the endothelial layer, increased nuclear factor kappa B expression and decreased endothelial nitric oxide synthase expression lead to infiltration of LDLs, leukocyte adhesion, platelet aggregation, and inflammatory response. LDL accumulation enhances the recruitment of monocytes to the injured area of the arterial walls and initiates a sustained inflammatory response, which contributes to the development of atherosclerotic plaques (5,18–21).

Atherogenic dyslipidemia is defined by elevated levels of LDL cholesterol, along with an elevation in triglyceride levels, an abundance of tiny dense LDL particles, reduced levels of HDL cholesterol, and postprandial hyperlipidemia (22). Dyslipidaemia was found to account for nearly 50% of the risk for developing MI in a comprehensive investigation involving 52 countries (23). Consistent with the results of previous research, our study found that triglyceride, non-HDL cholesterol, VLDL levels, TG/HDL ratio, TG/glucose, and non-HDL/HDL ratio were significantly higher in our patient group in comparison to the control group.

It is well known that triglyceride-rich lipoproteins and their cholesterol-enriched remnants have been demonstrated to exert a direct impact on the progression of atherosclerosis. Genetic studies have shown association between triglyceride levels and the risk of developing atherosclerotic cardiovascular diseases. Additionally, these investigations have discovered important proteins that control the triglycerides transport (24,25). Among the general population, individuals with fasting TGs of 580 mg/dL have a 5.1 times higher risk of MI, a 3.2 times higher risk of ischemic heart disease, a 3.2 times higher risk of ischemic stroke, and a 2.2 times higher risk of all-cause mortality compared to individuals with fasting TGs of 70 mg/dL (24). A study has demonstrated a correlation between childhood non-HDL cholesterol and LDL cholesterol levels and the incidence of cardiovascular disease occurrences in middle age. Furthermore, it has been found that non-HDL cholesterol is a more accurate marker of adult cardiovascular disease events compared to LDL cholesterol, particularly among individuals with normal LDL cholesterol levels but high non-HDL cholesterol levels. (26). In our study, similar to the literature, TG, non-HDL cholesterol, VLDL levels, TG/HDL ratio, TG/glucose, and non-HDL/HDL ratio were significantly high in our patient group.

Numerous investigations have indicated that elevated levels of triglycerides in the circulation and low levels of HDL cholesterol are linked to a higher likelihood of experiencing both stable coronary syndrome and acute coronary artery disease, even if LDL cholesterol levels are within the desired range (27). A recent study conducted in the Chinese population has demonstrated that the TG/HDL ratio serves as a reliable indicator for the existence of coronary artery calcification (28). Detecting an elevated TG/HDL ratio in prediabetic individuals at an early stage can aid in identifying atherosclerotic issues sooner. This can assist doctors in developing primary preventive strategies to combat atherosclerosis diseases in patients with prediabetes and DM (29). The investigation revealed a strong correlation between cholesterol levels and TG, LDL cholesterol, non-HDL cholesterol, VLDL cholesterol, non-HDL/HDL ratio, TG/glucose ratio, and TG/HDL ratio. However, the investigation also revealed a negative correlation between cholesterol levels and HDL cholesterol.

Although low levels of HDL cholesterol are recognized as a risk factor for atherosclerotic cardiovascular diseases, the use of medication to boost HDL cholesterol has shown negative outcomes, which raises doubts regarding the significance of HDL in these disorders (30,31). The HDL cholesterol levels of the individuals in the control group were markedly higher than those of the participants in the patient group in our study. Recent studies have emphasized the significance of the functionality of HDL cholesterol, rather than its levels (32,33). As a result, HDL cholesterol serves as a valuable diagnostic biomarker but cannot be considered as a therapeutic alternative (34). These findings might explain the occurrence of atherosclerotic illnesses even in individuals with normal amounts of HDL cholesterol.

Another important risk factor for atherosclerosis is elevated LDL cholesterol levels. LDL cholesterol plays a causal or etiologic role in the development of atherosclerosis (35). One large study showed that 49.7% of those without cardiovascular risk factors had subclinical atherosclerosis and that LDL

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cholesterol, even at levels deemed normal in the absence of significant cardiovascular risk factors, was independently correlated with the presence and degree of early systemic atherosclerosis (36). Genetically based targets have been confirmed by studies showing that lowering LDL cholesterol levels through pharmacological procedures or treatments is accompanied by regression of atheromatous plaques and a reduction in atherosclerosis-related cardiovascular events (35,37). It promotes the reduction of LDL cholesterol using more efficient methods of prevention, even in persons who are often regarded to be at low risk. Nevertheless, a fascinating study revealed that LDL cholesterol and total cholesterol do not pose a risk for atherosclerosis and related diseases. Surprisingly, patients with acute MI had lower than normal levels of LDL cholesterol, and individuals with familial hypercholesterolemia demonstrated similar LDL cholesterol levels as untreated individuals (38). In our study, although LDL cholesterol levels were higher in the patient group, it was not statistically significant. This is likely attributable to the fact that the patients in our patient cohort were taking medication to decrease their cholesterol levels.

Generally, LDL cholesterol is the primary target in the management of dyslipidaemia. However, non-HDL cholesterol has recently attracted attention as an alternative and reliable target. Non-HDL cholesterol, which includes all plasma lipoproteins such as LDL, triglyceride-rich lipoproteins (VLDL), their remnants and lipoprotein(a), has also been reported to be atherogenic and to contribute to the pathophysiology of atherosclerosis and is considered an independent risk factor/predictor for cardiovascular diseases. It has also been stated that non-HDL cholesterol may become a primary or secondary target in the management of dyslipidaemia (39,40). Likewise, high circulating lipoprotein(a) levels, which are mainly determined by heredity factors, directly induce atherosclerotic diseases via VLDL receptors on macrophage. Lipoprotein(a) is a VLDL receptor ligand and is taken up directly via VLDL receptors on macrophage for macrophage foam cell formation (41). A study has demonstrated a correlation between levels of LDL and non-HDL cholesterol during childhood and the occurrence of cardiovascular disease events in middle age. Furthermore, it has been found that non-HDL cholesterol is a more accurate predictor of adult cardiovascular disease events compared to LDL cholesterol, especially among individuals with normal LDL cholesterol levels but elevated non-HDL cholesterol levels (26). A study found that the non-HDL/HDL cholesterol ratio is a robust indicator of the risk of carotid atherosclerosis in postmenopausal women. Furthermore, this notable correlation highlights the significance of maintaining a proper balance between proatherogenic and antiatherogenic lipid particles in order to regulate vascular remodeling (42). The non-HDL/HDL ratio has been shown to reflect combined lipid abnormalities and serves as a more reliable predictor of conditions associated with dyslipidemia, such as coronary heart disease, metabolic syndrome, chronic kidney disease, and insulin resistance when compared to a single lipoprotein ratio. A recent experimental study in individuals with type 2 DM revealed that the non-HDL/HDL cholesterol ratio was a more accurate predictor of the risk of coronary heart disease compared to non-HDL cholesterol alone (43). Another study reported that the non-HDL/HDL cholesterol ratio was strongly associated with the stability of carotid plaque, and could serve as an important marker for the early detection of high-risk carotid plaques (44). Our study demonstrated a significant disparity between the patient and control groups. In addition, the presence of DM and MI and the non-HDL/HDL ratio was higher in the patient group.

The TG/glucose index has been recognized as a strong candidate biomarker for insülin resistance, and multiple research have shown strong statistical proof that this index is linked to the occurrence and outlook of cardiovascular disease. However the use of this index as an indicator of cardiovascular disease has not been dequately assessed (45). A recent study has demonstrated a significance association between the TG/glucose index and the risk of future cardiovascular mortality, stroke, MI, and type 2 diabetes. Furthermore, the study highlights the crucial role of insulin resistance in the development of cardiovascular and metabolic disorders (46). For this reason, we aimed to evaluate this index in our patient group with atherosclerosis since there were patients with and without DM. According to the results of our study, the TG/glucose index was higher in the patient group. However, no association was observed between this index and the presence/absence of DM or MI. However, when we compared the TG/glucose ratio with laboratory in our study, we found that it was positively correlated with total cholesterol, LDL,

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TG, non-HDL, VLDL, TG/HDL levels, and non-HDL/HDL ratio.

Conclusion

It is important for a biomarker or method to be noninvasive rather than invasive. We suggest that atherosclerotic index rates, in addition to routine lipid parameters, have the potential to predict asymptomatic individuals or the severity of atherosclerosis. However, it is necessary to conduct studies with larger samples.

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