

# The relationship between non-HDL cholesterol and coronary collateral circulation

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## Research

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# Abstract

**Objective** Non high density lipoprotein cholesterol (non HDL-C) covers all atherogenic lipoproteins and correlates with C reactive protein (CRP) which is reliable marker of inflammation. CRP is related to poor angiographic coronary collateral circulation (CCC). We aimed to show whether non HDL-C is associated with CCC.

**Methods** Patients who underwent coronary angiography for stable coronary artery disease and at least one epicardial coronary artery occluded in the proximal or middle region were included in the study. According to the Rentrop scoring system Rentrop 0 and 1 were considered to be poor CCC, and Rentrop 2 and 3 were considered to be good CCC. Non-HDL-C was calculated by subtracting HDL-C from total cholesterol (TC).

**Results** 84 patients were included in the study. While 44 patients (52%) had good CCC, 40 patients (48%) had poor CCC. TC was found to be higher in the poor CCC group than in the good CCC group ( $224.3 \pm 35.6$  vs  $179.2 \pm 25.5$   $p = 0.000$ ). HDL-C levels were found to be lower in the poor CCC group than in the good CCC group ( $37.3 \pm 9.8$  vs  $44.1 \pm 8.6$   $p = 0.001$ ). Non-HDL-C cholesterol levels were higher in the group with poor CCC ( $185.7 \pm 39.2$  vs  $132.8 \pm 28.1$   $p = 0.000$ ). CRP levels were found to be higher in the poor CCC group ( $3.73 \pm 2.5$  vs  $1.67 \pm 1.4$   $p = 0,000$ ).

**Conclusion** Non-HDL-C is independent predictors of poor CCC.

# Introduction

Coronary collateral circulation (CCC) is a congenital pathway and provides blood flow to the ischemic region in case of occlusion of the coronary artery. Percutaneous coronary intervention or surgical treatment are not suitable for approximately one-third of stable coronary artery disease (CAD) patients. The most important way to protect the ischemic area in these patients is to increase coronary collateral flow. The coronary collateral development mechanism is complex and not fully elucidated. However, endothel, tangential shear stress on the endothelial surface, transc collateral pressure gradient, hypoxia, inflammation and many cytokines have been shown to play a role in this process in both clinical and experimental studies [1, 2, 3, 4, 5, 6]. Inflammation has been shown to play an effective role in the formation of new vessels and the transformation of immature collaterals into mature collaterals in the process of collateral development [5, 7, 8].

Non-high-density lipoprotein cholesterol (non-HDL-C) is calculated by subtracting high-density lipoprotein cholesterol (HDL-C) from total cholesterol (TC) and covers all of the atherogenic lipoproteins [9]. It is a well-known fact that dyslipidaemia causes vascular endothelial dysfunction [10, 11]. Increasing the level of low-density lipoprotein cholesterol (LDL-C) decreases the degree of coronary collateral development by reducing the number of endothelial progenitor cells [12]. Increasing the HDL-C level increases endothelium-mediated vasodilation by correcting endothelial functions [13]. The C-reactive protein (CRP) level is correlated negatively with angiographic coronary collateral development [14]. Non-HDL-C is

correlated with C-reactive protein (CRP), which is a reliable marker of inflammation, but LDL-C has not been shown to correlate with CRP [15]. Therefore, it is important to investigate the relationship between non-HDL-C and coronary collateral development. Since we did not find any studies on non-HDL-C and coronary collateral development in the literature, we aimed to investigate the relationship between coronary collateral development and non-HDL-C in stable CAD patients.

## Material And Methods

### *Patients*

The study was undertaken after approval was granted by the ethics committee of Balıkesir University. Among the patients between 18 and 75 years old who underwent coronary angiography for stable CAD between 2017 and 2019 and at least one epicardial coronary artery occluded in the proximal or mid-region were included in the study. Demographic, clinical and laboratory parameters were obtained from patients' electronic records. Hypertension (HT) was determined if blood pressure measured at rest was higher than 140 mmHg systolic and higher than 90 mmHg diastolic or patients were taking anti-hypertensive medication [16]. Diagnoses of stable CAD were made according to the American College of Cardiology/American Heart Association (ACC/AHA) criteria [17]. Percutaneous intervention history of coronary artery, history of coronary artery bypass graft operation, hypolipidemic drug intake, patients with diabetes mellitus, triglyceride (TG) levels > 400mg/dl, hospitalisation due to acute coronary syndrome, severe valvular heart disease, New York Heart Association (NYHA) class 3 or 4, renal or hepatic insufficiency, neoplastic diseases, systemic inflammatory or infectious diseases, and anti-inflammatory drug use were accepted as exclusion criteria.

### *Angiographic evaluation of coronary collateral flow*

Coronary angiographies were performed through the femoral and radial arteries. A Philips Allura Xper device was used for angiography. Coronary angiographies were evaluated by two interventional cardiologists. The grading of the lesions in the coronary arteries was done according to ACC/AHA criteria [18]. When there was an inconsistency in the collateral grading done by the two cardiologists, the angiography was evaluated by a third cardiologist and the grading was decided by consensus. The Rentrop scoring system was used to evaluate the coronary collaterals between epicardial coronary arteries. Grading according to collateral filling was performed as follows: 0 = no visible filling; 1 = visualisation of small branches with contrast; 2 = major branches of the main vessel are visible; 3 = considered the main epicardial vessel to be fully visible with the collateral. Scores of Rentrop 0 and 1 were considered to be poor collateral flow, and Rentrop 2 and 3 were considered to be good collateral flow [19]. The highest collateral flow rating for collateral grading was accepted for analysis in patients with multiple occluded vessels.

### *Biochemical parameters*

Venous blood samples were taken after at least 8 hours of fasting before coronary angiography. Complete blood count, renal functions, liver function tests and fasting lipid profile were examined. TG, total cholesterol and HDL-C measurements were calculated by automatic bioanalysis (Beckman Coulter, Indianapolis, IN). LDL-C was calculated using the Friedewald formula. Non-HDL-C was calculated by subtracting HDL-C from total cholesterol [20]. Latex-enhanced immunonephelometric assay was used for high-sensitivity CRP level measurement (Siemens Healthcare Diagnostics, BN2 Nephelometer, Camberley, Surrey, UK).

### *Statistical analysis*

Statistical software programme SPSS for Windows 15.0 (SPSS Inc., Chicago, Illinois, USA) was used for statistical analysis. Normal distributed continuous data were expressed as the mean  $\pm$  standard deviation (SD); non-normal distributed continuous variables were presented as the median (minimum-maximum). Categorical data were expressed as numbers with percentages. Differences between the sufficient and the insufficient collateral groups were analysed using the Student's t-test for normally distributed continuous variables and the Mann-Whitney U test for non-normally distributed continuous variables. Categorical data were compared using the Chi-squared test. The result was accepted as statistically significant when the p-value was under 0.05.

## **Results**

A review was conducted of the files of 1,920 patients undergoing coronary angiography under elective conditions, and a total of 84 patients were included in the study after the exclusion criteria were applied. While 44 patients (52%) had good coronary collateral, 40 patients (48%) had poor coronary collateral. There was no difference between groups in terms of age, gender, body mass index or HT. TC was found to be higher in the poor CCC group than in the good CCC group ( $224.3 \pm 35.6$  vs  $179.2 \pm 25.5$   $p = 0.000$ ). There was no difference between the groups in terms of LDL-C levels ( $142.3 \pm 21.7$  vs  $136.1 \pm 24.1$   $p = 0.222$ ). HDL-C levels were found to be significantly lower in the poor CCC group than in the good CCC group ( $37.3 \pm 9.8$  vs  $44.1 \pm 8.6$   $p = 0.001$ ). When TG levels were evaluated, there was no difference between the groups ( $185 \pm 69.7$  vs  $186 \pm 66.2$   $p = 0.941$ ). Non-HDL-C levels were significantly higher in the group with poor CCC ( $185.7 \pm 39.2$  vs  $132.8 \pm 28.1$   $p = 0.000$ ). CRP levels were found to be significantly higher in the poor CCC group ( $3.73 \pm 2.5$  vs  $1.67 \pm 1.4$   $p = 0,000$ ). There was no significant difference between the groups in the numbers of diseased vessels ( $2.07 \pm 0.79$  vs  $2.25 \pm 0.74$   $p = 0.28$ ). The main characteristics of the groups are shown in Table 1.

In logistic regression analysis, non-HDL-C, HDL-C and CRP were found to be independent predictors for poor coronary collateral development (Table 2).

Non-HDL-C and TC were superior to other parameters for CCC in ROC curve analysis (Fig. 1). In ROC analysis performed to demonstrate the effect of non-HDL-C on CCC, sensitivity was found 90% and specificity was found 70%. The area under the curves of non-HDL-C and CCC were 0.865 (95% CI: 0,773 to 0,930,  $p < 0.001$ ; Fig. 2).

## Discussion

This is the first study to investigate CCC with non-HDL-C in patients with stable coronary artery disease (CAD). The main finding of our study is that non-HDL-C negatively affects the coronary collateral development in patients with stable CAD.

Coronary collateral vessels are also present in normal hearts, and they provide an alternative way of providing blood flow to the ischemic area when coronary obstruction develops. In the collateral development process, the diameter of the collateral vessels can become up to four times larger [21]. Although CCC can supply enough blood to the heart during rest, it is often insufficient in cases of increased oxygen demand [22]. Coronary collaterals maintain myocardial functions in acute myocardial infarction, limit the infarct area and have a positive effect on remodelling [23,24,25]. The degree of CCC development shows marked heterogeneity in CAD. Numerous theories have been put forward regarding the mechanism underlying this heterogeneity, but the underlying mechanism has not been fully elucidated. It has been demonstrated in many clinical and in vivo studies that inflammation is closely related to the development of CCC and the presence of inflammation negatively affects coronary collateral development. Kerner et al. showed that elevated CRP levels in patients with chronic CAD are associated with decreased coronary collateral flow [26]. Similarly, it has been found in other studies that elevated CRP levels negatively affected the development of coronary collateral flow [14,27,28]. In our study, CRP levels were higher in patients with poor collateral circulation. This finding is compatible with the literature. In addition, there are publications indicating that the inflammatory markers tumor necrosis factor alpha and soluble adhesion molecules negatively affect coronary collateral circulation [29,30].

It has been reported in some studies that non-HDL-C is associated with inflammation. In a study conducted by Wang and Chang in a diabetic patient group, it was shown that non-HDL-C correlates with CRP levels and non-HDL-C is an independent predictor for inflammation in this patient group [15]. Hsu et al. found results which were similar to those in Wang's work [31]. It has been suggested in a recent study that non-HDL-C may be superior to CRP in determining future cardiovascular events in children and adolescents with type 1 diabetes [32]. In fact, one study has shown that non-HDL-C has a high correlation with proinflammatory macrophages in adipose tissue [33]. Moreover, in an experimental study on apolipoprotein E-deficient mice, non-HDL-C reduction was reported to have a positive effect on the development of atherosclerosis by inhibiting leucocyte transmigration [34]. Evidence from these studies made it clear that there is a close relationship between non-HDL-C and inflammation. There are a limited number of studies investigating the relationship between HDL-C and CCC. In one of these studies, Kadi et al. showed that there was a positive relationship between high HDL-C levels and CCC in patients with at least one occluded coronary artery, and they suggested that the possible underlying cause may be related to the anti-inflammatory properties of HDL-C [35]. In addition, Hsu et al. claimed that there was no relationship between HDL-C and coronary collateral circulation in patients with coronary stenosis above 70% [36]. There are a limited number of studies investigating the relationship between LDL-C and coronary collateral flow. In a study of stable CAD patients with chronic total occlusion with type 2 diabetes mellitus, Shen et al. found LDL-C and non HDL-C as predictors for poor collateral development

[37]. We did not include diabetic patients in our study group since in DM patients HDL-C is low and LDL-C and TG-rich lipoproteins are high [38,39]. There are no studies investigating the relationship between non-HDL-C and CCC. Our study is the first on this subject. In our study, we showed the relationship of non-HDL-C with CCC in both univariate and multivariate analyses. We also found that there is a close relationship between non-HDL-C and CRP levels, and CRP and non-HDL-C are independent predictors for CCC. Based on an analysis of our study findings, we can state that HDL-C is also an independent predictor for CCC because the p value was found within the limit of significance in logistic regression analysis. This may be related to the number of cases. This close relationship between non-HDL-C and CCC can be explained by increased inflammatory activity in patients with high non-HDL-C.

As a result, non-HDL-C, TC, elevated CRP and low HDL-C are independent predictors for poor coronary collateral development. TG level and LDL-C do not predict coronary collateral development.

### *Limitations of the study*

First, coronary collateral flow was qualitatively evaluated with Rentrop scoring. The collateral flow index, which is a quantitative measurement and gives more accurate results, was not used. Second, patients with TG levels of 400 mg/dl were excluded from the study because LDL-C was calculated using the Friedewald formula. Finally, only the CRP of inflammation markers was evaluated, and no specific test for endothelial functions was performed.

## **Abbreviations**

**ACC/AHA** American College of Cardiology/American Heart Association

**CAD** Coronary artery disease

**CCC** Coronary collateral circulation

**CRP** C-reactive protein

**HDL-C** High density lipoprotein cholesterol

**HT** Hypertension

**LDL-C** Low density lipoprotein cholesterol

**Non-HDL-C** Non high density lipoprotein cholesterol

**NYHA** New York Heart Association

**ROC** Receiver operating characteristic

**TC** Total cholesterol

TG Triglyceride

## Declarations

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#### Contributions

All authors contributed to the study conception and design. YSE collected the data; AO collected the data ; KH analysed the data; KHL analysed the data; SO analysed the data; TO prepared the material; YT prepared the manuscript, AE prepared the manuscript. All authors read and approved the final version of manuscript.

#### Corresponding author

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#### Ethics Declarations

##### Ethics approval

This study was approved Balıkesir University Ethics Comitte (Approval ID and date are 2020/23 and 05/02/2020).

#### Competing interest

The authors declare that they have no competing of interest.

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## Tables

**Table 1 Demographic, laboratory and angiographic characteristics of the patients**

Variable	Poor CCC (n=40)	Good CCC (n=44)	<i>p</i>
Age, Years (mean±SS)	62,4±12,4	59,9±8,5	0,295
Gender, female, n (%)	11(25)	7(18)	0,438
TC, mg/dl	224,3±35,6	179,2±25,5	0,000
HDL-C, mg/dl	37,3±9,8	44,1±8,6	0,001
LDL-C, mg/dl	142,3±21,7	136,1±24,1	0,222
TG, mg/dl	185±69,7	186±66,2	0,941
Non-HDL-C, mg/dl	185,7±39,2	132,8±28,1	0,000
CRP, mmol/L	3,73±2,5	1,67±1,4	0,000
Creatinin, mg/dl	0,94±0,28	0,99±1,87	0,287
Diseased number of vessels, n	2,07±0,79	2,25±0,74	0,281

CRP: C-reactive protein, HDL-C: High density lipoprotein cholesterol, LDL-C; Low density lipoprotein cholesterol, Non-HDL-C: Non high density lipoprotein cholesterol, TC: total cholesterol, TG: triglyceride

**Table 2 Regression analysis**

	B	SE	Wald	<i>p</i>	Exp (B)	95% CI	
						Lower	Upper
Age	-0,37	0,042	0,765	0,382	0,964	0,887	1,047
HDL-C	0,086	0,044	3,878	0,049	1,090	1,000	1,187
Non-HDL-C	-0,061	0,017	13,000	0,000	0,941	0,910	0,972
CRP	-0,759	0,261	8,453	0,004	0,468	0,280	0,781

CRP: C-reactive protein, HDL-C: High density lipoprotein cholesterol, Non-HDL-C: Non high density lipoprotein cholesterol

## Figures

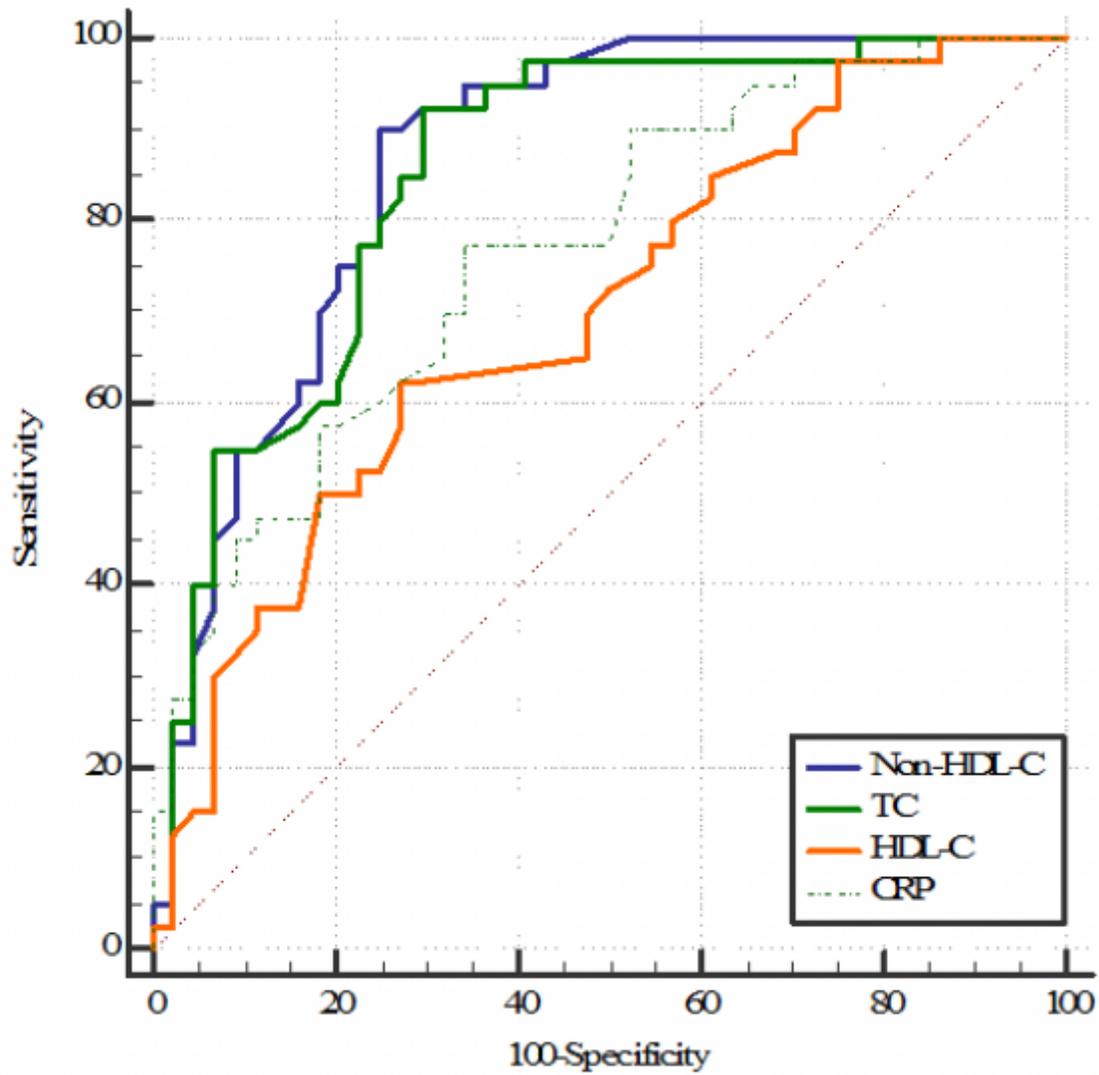
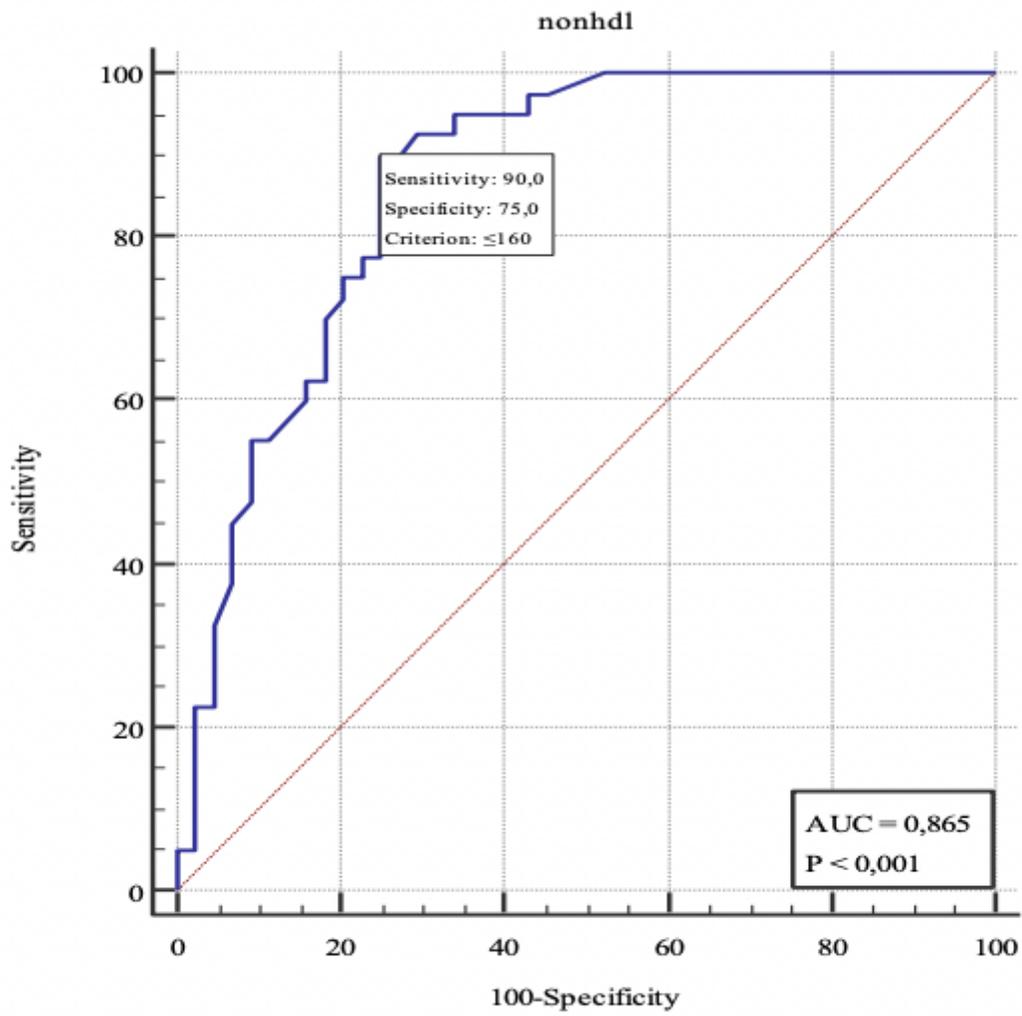


Figure 1

Receiver operating characteristic (ROC) curves for non-HDL-C, TC, HDL-C and CRP



**Figure 2**

Receiver operating characteristic (ROC) curves for non-HDL-C