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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

The relationship between ischaemia-modified albumin and good coronary collateral circulation

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Abstract

Background: It is important to determine the grade of the coronary collateral circulation (CCC) in patients with stable coronary artery disease.

Aim: In this study, we aimed to investigate the relationship between the ischaemia-modified albumin (IMA) level and good CCC.

Methods: A total of 95 patients with coronary angiography and at least one epicardial coronary artery obstruction were included in the study. The Rentrop classification was used with CCC grading, where 0 and 1 were defined as poor collateral, and 2 and 3 were defined as good collateral. The IMA level of the patients was measured using an enzyme-linked immunosorbent assay (ELISA). The receiver–operating characteristic curve was used to show the sensitivity and specificity of IMA levels and the optimal cut-off value for predicting good CCC.

Results: The multiple logistic regression analysis revealed that the IMA level in the good CCC group was higher ($p < 0.045$). Conversely, the high-sensitivity C-reactive protein level was lower in the good CCC group ($p < 0.023$). We found an IMA cut-off value (4.7 ng/mL) that indicated good CCC level, and this shows good CCC with 70.2% sensitivity and 60.3% specificity.

Conclusions: The IMA level could serve as a simple and useful predictor of well-developed CCC.

Key words: coronary collateral circulation, ischaemia-modified albumin, inflammation

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INTRODUCTION

Coronary collateral circulation (CCC) provides alternative blood circulation for the ischaemic myocardium, and its effect on mortality is known [1]. Specifically, mortality, and morbidity have been explained by the effect it has on the infarct area [2]. Numerous collateral vessels that connect the main coronary arteries exist in the normal human heart [3], and for patients presenting with normal or mild coronary artery disease (CAD), the collateral channels are very small. Consequently, they frequently go undetected on coronary angiograms because they only carry low blood flow. To make the collateral vessels visible, it is necessary for the coronary artery to be obstructed 99% or 100% [4]. It is noteworthy that the CCC process involves clinical parameters such as the duration of cardiac ischaemia, blood vessel formation, and inflammation [5, 6]. Referred to as angiogenesis, blood vessel formation involves a series of important steps, including the activation, migration, and proliferation of various endothelial cells [7, 8]. Researches have demonstrated that coronary angiogenesis is driven by myocardial ischaemia and inflammatory processes [9, 10].

Ischaemia-modified albumin (IMA) is produced due to the alteration of human serum albumin by ischaemia and it has been identified as a novel biomarker for the detection of myocardial ischaemia. In a recent study, it was evaluated as a marker of ischaemia [11]. Endogenous stress conditions, including ischaemia, lead to ischaemia-related hypoxia and acidosis, the formation of reactive oxygen radicals, and cell membrane dysfunction. This condition decreases the binding capacity of albumin's N-terminal region for cobalt, copper, and nickel with the resultant conversion of serum albumin to IMA [12]. In a study that performed myocardial perfusion scintigraphy, IMA levels were higher in the ischaemic group than in the normal group [13].

In view of these considerations, the purpose of the present study was to investigate the predictability of good CCC.

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METHODS

Study design

Our sample included patients with stable CAD, who underwent coronary angiography and CCC in Ankara Numune Educational and Research Hospital (ANERH) between August 2015 and August 2016. Two hundred patients were planned to be included in the total sample, but 105 patients had to be disqualified according to the exclusion criteria. All of the patients had Canadian Cardiovascular Society class 2–3 angina despite optimal medical treatment according to the current clinical guidelines. At least one major epicardial coronary vessel in the study population was 100% chronically obstructed. Chronic total occlusion was defined according to the 2013 European Society of Cardiology guidelines on the management of stable CAD [14]. The following criteria for exclusion from the study were considered: acute or chronic infections and inflammatory diseases, operated CAD, chronic renal failure (serum creatinine > 2.0 mg/dL), liver cirrhosis, cerebrovascular disease, previous stroke at any time, severe heart valve disease, and acute coronary syndrome within the last month. Cardiovascular risk factors were identified based on the medical records of patients. Transthoracic echocardiography was performed on all study participants, and left ventricular ejection fraction was calculated by employing Simpson's technique. After the Ethics Committee of ANERH approved the study, each patient provided written, informed consent and the study commenced.

Coronary angiography and collateral grading

Coronary angiography was carried out by using the Judkins technique, and the angiographic evaluations were performed by two cardiologists. A third cardiologist was consulted when these two practitioners disagreed on an issue. The grading of the collateral flow was conducted according to the Rentrop classification [15]. To be specific, this meant that Grade 0 indicated no filling of any collateral vessels; Grade 1, the filling of side branches of the artery to be perfused by collateral vessels without visualisation of the epicardial segment; Grade 2, the partial filling of the epicardial artery by collateral vessels; and Grade 3, the complete filling of the epicardial artery by a collateral vessel. Finally, Rentrop 0 and 1 collateral flow were evaluated as bad CCC, whereas Rentrop 2 and 3 collateral flow were evaluated as good CCC.

Measurements

All blood samples were obtained from peripheral venous blood after the diagnostic coronary angiography. If percutaneous coronary intervention was planned, it was performed after sample collection. The samples were centrifuged at 3000 g for 10 min, and then the serum was stored at –80°C until the IMA level was measured. Serum lipid parameters, creatinine levels, haemotoogical parameters, and high sensitivity C-reactive protein (hsCRP) levels were obtained from local laboratory records. For relevant measurements, human ischaemia-modified albumin (ELABSCIENCE, Wuhan, Hubei Province China) kits were used. The IMA levels were given as ng/mL.

Statistical analysis

For all statistical analyses, the analysis of variables was carried out using SPSS 22.0 (IBM Corporation, Armonk, New York, United States) and MedCalc 14 (Acacialaan 22, B-8400 Ostend, Belgium). The normal distribution of univariate data was assessed by the Shapiro-Wilk test, and variance homogeneity by Levene's test. The Mann-Whitney U test was used in conjunction with the Monte Carlo results, while Independent Samples t-test was used with bootstrap results when comparing quantitative data between two independent groups quantitative data. The categorical variables were compared using the Pearson χ^2 Monte Carlo Simulation technique and Fisher's exact test. To determine the causal relationship with explanatory variables in the diatom and multinomial categories, multivariate logistic regression analyses were performed. Relative sensitivity and specificity between the classification and the actual classification, which were made by the cut-off values calculated according to the group variables, were examined and expressed by receiver operating curve (ROC) analysis. Quantitative variables were shown as mean ± standard deviation (SD) and median with a range (minimum–maximum), and categorical variables were number (%). In be considered statistically significant, the confidence interval was determined to be 95% and p-values were less than 0.05.

RESULTS

Of the 95 patients who were included in the study, 60 were in the good coronary collateral group and 35 were in the poor coronary collateral group. Demographic, clinical, and angiographic data for the two groups are compared in Table 1. HsCRP, high density lipoprotein, and IMA levels were found to be statistically significant in the good coronary collateral group, and no significant difference was found for the other parameters. In univariate and multivariate logistic regression analysis, predictors of good CCC were investigated (Table 2). The IMA level in the good CCC group was higher ($p < 0.045$). Conversely, the hsCRP level was lower in the good CCC group ($p < 0.023$). The correlation between IMA level and Rentrop score is shown in Figure 1. Figure 2 shows the relationship between IMA level and hsCRP. Finally, Figure 3 shows that IMA can serve as an effective diagnostic test for patients with good CCC (area under curve: 0.715; 95% confidence interval 0.595–0.835). After employing ROC analysis, we found that IMA level > 4.7 ng/mL cut-off values showed good CCC with 70.2% sensitivity and 60.3% specificity.

Table 1. Clinical, laboratory, and angiographic characteristics of the study population

Variables	Poor CCC (n = 35)	Good CCC (n = 60)	p
Male	26 (74.3%)	44 (73.3%)	0.608
Age [years]	65.28 ± 13.56	63.00 ± 11.14	0.476
Diabetes mellitus	10 (28.5%)	12 (20.0%)	0.310
Current smoker	6 (17.1%)	21 (35.0%)	0.098
Arterial hypertension	17 (48.5%)	37 (61.6%)	0.373
White blood cell count [$\times 10^9$/L]	7.7 (4.6–19.7)	8.1 (1.0 – 19.7)	0.836
Haemoglobin [g/L]	134.6 ± 19.4	134.8 ± 17.5	0.933
Neutrophil count [$\times 10^9$/L]	5.0 (3.0–17.2)	5.2 (2.1–17.2)	0.550
Lymphocyte count [$\times 10^9$/L]	1.86 ± 0.6	2.05 ± 0.71	0.218
Monocyte count [$\times 10^9$/L]	0.6 (0.2–1.2)	0.6 (0.2–1.4)	0.400
Platelet count [$\times 10^9$/L]	254 ± 64	237 ± 54	0.235
Triglyceride [mmol/L]	1.39 (0.58–4.75)	1.69 (0.66–5.06)	0.148
Total cholesterol [mmol/L]	4.44 (2.85–7.74)	4.48 (2.31–14.76)	0.996
LDL cholesterol [mmol/L]	2.81 ± 0.96	2.75 ± 1.06	0.747
HDL cholesterol [mmol/L]	1.10 (0.57–1.65)	0.96 (0.60–1.81)	0.021
Creatinine [μmol/L]	55.48 (45.75–122.00)	77.78 (45.75–129.63)	0.672
Left ventricular ejection fraction [%]	48.69 ± 12.27	47.14 ± 12.36	0.391
Highly sensitive C-reactive protein [nmol/L]	114.2 (8.57–1123.8)	39.05 (3.81–1238.12)	< 0.001
Ischaemia modified albumin [ng/mL]	2.45 (0.06–65.68)	11.41 (0.3–67.85)	< 0.001
Systolic blood pressure [mmHg]	137 ± 13	136 ± 14	0.911
Diastolic blood pressure [mmHg]	80 ± 8	80 ± 7	0.602
Prior medications:			
Angiotensin converting enzyme inhibitors or sartans	30 (85.7%)	56 (93.3%)	0.542
Statins	31 (88.5%)	58 (96.6%)	0.180
Beta-blocker	32 (91.4%)	52 (86.6%)	0.159
Position of occluded coronary artery:			0.816
Left anterior descending artery	10 (28.5%)	15 (25.0%)	
Left circumflex	14 (40.0%)	28 (46.6%)	
Right coronary artery	11 (31.4%)	17 (28.3%)	
Rentrop score:			
0	13 (37.1%)		
1	22 (62.8%)		
2		32 (53.3%)	
3		28 (46.6%)	

Data are expressed as median (interquartile range), mean ± standard deviation, or number (percentage) as appropriate; CCC — coronary collateral circulation; HDL — high-density lipoprotein; LDL — low-density lipoprotein

Table 2. Univariate and multivariate logistic regression analysis showing independent predictors of good coronary collateral circulation

Variables	Univariate logistic regression analyses		Multivariate logistic regression analyses	
	Odds ratio (95% CI)	p	Odds ratio (95% CI)	p
Current smoker	2.459 (0.872–6.935)	0.089	2.615 (0.731–9.352)	0.139
Triglyceride	1.003 (0.997–1.008)	0.357	0.999 (0.993–1.006)	0.868
High-density lipoprotein cholesterol	0.964 (0.927–1.003)	0.072	0.983 (0.938–1.030)	0.472
Highly sensitive C-reactive protein	0.976 (0.954–0.997)	0.021	0.977 (0.958–0.997)	0.023
Ischaemia-modified albumin	1.062 (1.013–1.112)	0.012	1.056 (1.001–1.113)	0.045

CI — confidence interval

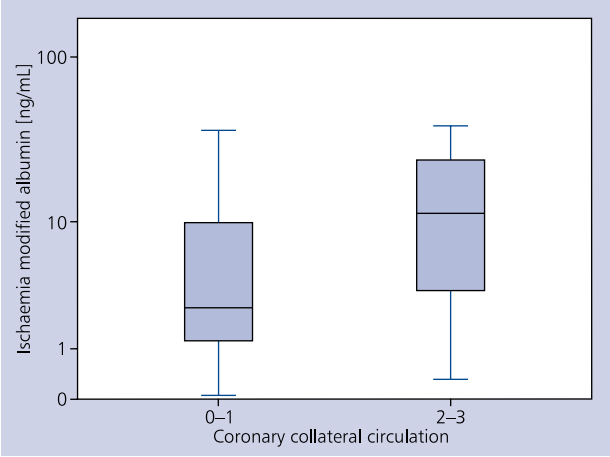

Figure 1. The association between the ischaemia-modified albumin level and the Rentrop score

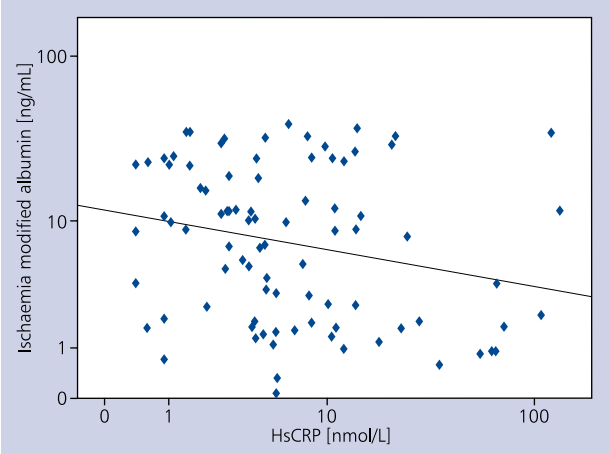

Figure 2. The association between the ischaemia-modified albumin and high-sensitivity C-reactive protein (hsCRP)

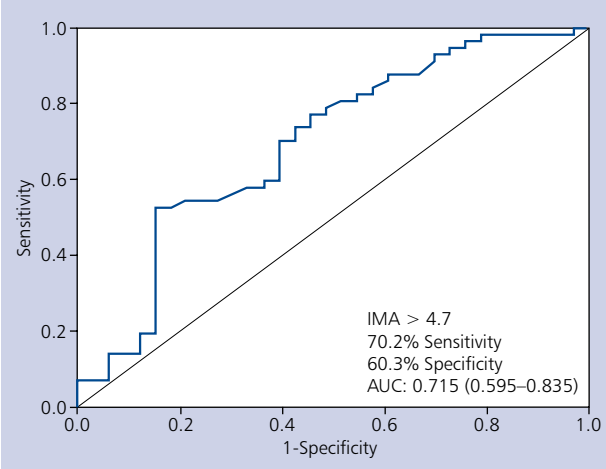

Figure 3. The receiver–operating characteristic curve of ischaemia-modified albumin (IMA) level for the prediction of coronary collateral circulation; AUC — area under curve

DISCUSSION

The main finding of our study is that high hsCRP levels are associated with poor CCC, while high IMA levels predict good CCC. Notably, no study in the extant literature has examined the relationship between IMA level and CCC.

Coronary collateral circulation is important because it has beneficial effects on good collateralisation, ventricular function, infarct size, and aneurysm formation [16]. The degree of coronary collateral development varies among patients with ischaemic cardiac disease, and the factors that affect functional coronary collateral formation are not well defined [17]. CCC formation is affected by many factors, including the severity and duration of coronary artery stenosis, endothelial dysfunction, shear stress, endogenous mediators, hypertension, diabetes mellitus, dyslipidaemia, cigarette smoking, and the duration of medications. Endogenous mediators involved in angiogenesis include vascular endothelial growth factor, tumour necrosis factor alpha or beta, fibroblast growth factor, nitric oxide, inflammatory markers, and neurohumoral factors. It is also known that proinflammatory enzymes such as myeloperoxidase increase CCC [18, 19]. In a study conducted by Kobusiak-Prokopowicz et al. [20], the researchers investigated the role of proinflammatory factors, proangiogenic factors, and endostatin in the development of heart failure and differentiated CCC. This inflammatory process at the molecular level can be demonstrated by the determination of hsCRP in the peripheral blood. The association of hsCRP levels with atherosclerosis and good CCC has been demonstrated in several previous publications [21, 22], and the impact that hsCRP has on collateral development involves the reduction of nitric oxide production and the inhibition of angiogenesis [10]. Our findings are consistent with the literature showing that the hsCRP level is lower in patients with good CCC.

Ischaemia-modified albumin is detected as elevated in all cardiac and extracardiac events caused by oxidative stress resulting from ischaemia reperfusion injury [23–25]. It has been investigated in internal and surgical diseases where high oxidative stress has been found to be significant, including ischaemic bowel disease and acute appendicitis [26, 27]. Its role in atherosclerotic heart disease has been determined previously, where Zhong et al. [28] notably found a positive correlation between the number of atherosclerotic levels and the IMA level. Multivessel CAD disease is associated with an increase in mortality, and IMA can be used indirectly as a mortality indicator. In addition, a study conducted in 2009 showed that the IMA level may serve as an indicator of atherosclerotic disease burden before hsCRP and pro-B-type natriuretic peptide elevation, thereby holding promise as a risk classification factor [29]. Other studies have indicated a possible role for IMA testing in the early triage of patients with chest pain [30, 31]. IMA is known as a marker of ischaemia but IMA is a product of albumin which is exposed to oxidative products [12]. The underlying cause of IMA production is the increase of oxidative products. Previous studies evaluating the

relationship between oxidative status and CCC demonstrated that there is an association between poorly developed collaterals and increased antioxidant capacity. Demirbag et al. [32] showed that good CCC is associated with increased oxidative stress markers. As serum IMA levels are increased in oxidative stress, it is expected that IMA levels are increased in patients with good CCC, and our findings support this hypothesis.

Limitations of the study

Several limitations should be addressed in this study, the first of which relates to the small sample size. Furthermore, participants in this study had not undergone intracoronary haemodynamic assessment with pressure or Doppler sensors to measure coronary collateralisation, and it is notable that the gold standard for measuring collateralisation is intravascular haemodynamic assessment (coronary flow index). We did not perform any perfusion imaging to show the amount of ischaemic myocardium that can be related with IMA levels. Finally, although small microvascular calibre vessels may not be visualised angiographically, the Rentrop scoring system was used for collateral grading.

CONCLUSIONS

The results of this study show that a high IMA level could serve as an effective predictor of good CCC in patients with stable CAD. However, larger prospective studies are needed to support our study.

Conflict of interest: *none declared*

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