Kardiologia Polska 2017; 75, 10: 990–996; DOI: 10.5603/KP.a2017.0119

ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

# Association of serglycin levels with isolated coronary artery ectasia

Harun Kundi, Murat Gök, Canan Topçuoğlu, Ender Ornek

Cardiology Department, Numune Education and Research Hospital, Ankara, Turkey

**Abstract**

**Background:** Serglycin plays an important role in the inflammatory status, but the relationship between coronary artery ectasia (CAE) and serglycin is still unknown.

**Aim:** In this study, we aimed to investigate the association of serglycin level with isolated CAE.

**Methods:** Fifty-two patients with isolated CAE and 35 individuals with normal coronary angiography were included into the study. The Markis classification and number of ectatic coronary arteries were recorded. Plasma serglycin levels were measured.

**Results:** Multivariate logistic regression analysis revealed that serglycin and high-sensitivity C-reactive protein were independently associated with the presence of CAE. In receiver operating characteristics curve analysis the cut of serglycin level for the prediction of isolated CAE was 13.5, with a sensitivity of 88.5% and a specificity of 84.8%. However, there was no association between serglycin levels and Markis classification.

**Conclusions:** Serglycin levels are significantly and independently higher in patients with CAE.

**Key words:** coronary artery ectasia, inflammation, serglycin

Kardiol Pol 2017; 75, 10: 990–996

## INTRODUCTION

Coronary artery ectasia (CAE) is defined as dilatation of the coronary artery 1.5-times greater than that of an adjacent normal segment [1]. Its prevalence has been reported from 0.3% to 5% [2]. Isolated CAE, defined as the lack of significant coronary artery stenosis, has rarely been reported (0.1–0.8%) [3, 4]. Acute coronary syndromes can be developed by abnormally dilated coronary arteries as a result of vasospasm, dissection, or thrombus, even in patients without coronary artery disease (CAD) [5]. Therefore, the factors that are associated with the presence of CAE need to be determined. Previous studies have found that atherosclerosis and inflammation play a key role in the development of CAE, although the reasons for the formation of ectasia have not been understood exactly [6, 7].

Serglycin is associated with intracellular proteoglycan and haematopoietic cells. Prior studies demonstrated that some non-haematopoietic cell types also synthesise serglycin. Inflammatory cells synthesise serglycin, which is then stored in granules to react with mediators, for instance cytokines, chemokines, growth factors, and proteases [8–10]. Serglycin can also participate in the mechanism of atheromatous change as well as atherosclerosis. It has been shown that serglycin is up-regulated by lipopolysaccharides in macrophages, tumour necrosis factor (TNF) in endothelial cells, and interleukin 1-beta in smooth muscle cells [10].

Because of these findings, serglycin plays an important role in the inflammatory status and plays a key role in the development of vascular diseases; we hypothesise that serglycin might play a role in CAE. Therefore, we aimed to investigate the association of serglycin level with isolated CAE. To the best of our knowledge, this is the first such study in the literature.

## METHODS

In total, 85 individuals were included in this cross-sectional study. The first group comprised 52 patients with isolated CAE, while the second group consisted of 33 individuals with normal coronary angiography (NCA).

Patients with high troponin due to causes other than acute coronary events including acute coronary syndromes, acute heart failure, pulmonary embolism, active infection or

**Address for correspondence:**
Harun Kundi, MD, Cardiology Department, Numune Education and Research Hospital, 06100, Sıhhiye, Ankara, Turkey, tel: 00905323529393, e-mail: harunkundi@hotmail.com

**Received:** 22.12.2016 **Accepted:** 18.05.2017 **Available as AoP:** 01.06.2017

sepsis, chronic kidney disease, stroke, arrhythmias, or aortic dissection were excluded from the study. Patients with haematological disorders, chronic inflammatory diseases, previous stroke, liver disease, malignancy, rheumatological diseases, previous myocardial infarction, or coronary artery surgery history were also excluded.

Arterial hypertension was defined as blood pressure measuring > 140/90 mm Hg in multiple measurements or use of antihypertensive drugs. Diabetes mellitus was defined as a fasting plasma glucose level ≥ 126 mg/dL in multiple measurements or use of anti-diabetic medications currently. Hyperlipidaemia was defined as use of lipid lowering medication or serum containing total cholesterol > 200 mg/dL.

Coronary angiography was done by clinical indications such as the test results of abnormal stress, positive treadmill test, dobutamine stress echo, typical chest pain, or myocardial perfusion scintigraphy. All the patients were clinically stable. The standard Judkins technique and 6 F or 7 F catheters (Massachusetts, Expo; Boston Scientific Corporation) were used to perform baseline angiography through the radial or femoral artery, using a Siemens Axiom Sensis XP device. Coronary angiographies were evaluated by at least two independent interventional cardiologists. CAE was defined based on the criteria used in the Coronary Artery Surgery Study [11]. According to the angiographic definition used in that study, segmental ectasia was considered when the diameter of the ectatic segment was ≥ 1.5 times that of the adjacent normal segment. When an identifiable normal adjacent segment could not be found, the mean diameter of the corresponding coronary segment in the control group was used as the normal value [1]. CAE without coronary artery stenosis was considered as isolated CAE, and the severity of isolated CAE was determined according to the Markis classification [3]. In decreasing order of severity, diffuse ectasia of two or three vessels was classified as type 1, diffuse disease in one vessel and localised disease in another vessel as type 2, diffuse ectasia of only one vessel as type 3, and localised segmental ectasia as type 4.

Blood samples from the patients after a fasting period of 12 h were collected into plain tubes, and serum was separated after centrifugation at 4000 g for 10 min and stored at –80°C until analysis. Complete blood count and differentials were determined from the peripheral venous blood samples obtained at admission. An automatised analyser was used to measure high-sensitivity C-reactive protein (hsCRP), total cholesterol, triglyceride, creatinine, and low- and high-density lipoprotein cholesterol. Serglycin levels in plasma were determined by previously described enzyme-linked immunosorbent assay method [12].

Transthoracic echocardiography was performed in all individuals. Left ventricular ejection fraction was calculated using Simpson's method.

### *Statistical analysis*

Ankara Numune Education and Research Hospital's Local Ethics Committee approved the study protocol, and all patients provided written, informed consent.

SPSS 22.0 statistical package software was used to perform all data analyses. The distribution pattern of the variables was analysed using the Kolmogorov-Smirnov test. Continuous data are presented as mean ± standard deviation, or median and interquartile range, according to the distribution pattern of the variables. Student's t test was used to compare parametric continuous variables, and the Mann-Whitney U test was used to compare nonparametric continuous variables. Categorical variables were compared using the $\chi^2$ test, the results of which are presented as percentages. The correlation between hsCRP and serglycin was assessed by performing the Spearman rank test. The effects of different variables on CAE were determined with univariate analysis. Variables with unadjusted p values < 0.10 in logistic regression analysis were identified as potential risk factors and were included in the full model. We eliminated potential risk factors by means of likelihood ratio tests with reduced model, using stepwise multivariate logistic regression analysis. A p value < 0.05 was considered statistically significant. The receiver operating characteristics (ROC) curve was used to show the sensitivity and specificity of serglycin, as well as the optimal cut-off value for predicting CAE.

## RESULTS

We performed a total of 1840 angiograms during the study period. CAE was detected in 165 patients. Of the 165 patients with CAE, 101 had also obstructive coronary lesions. Twelve of 64 patients with isolated CAE, who did not give informed consent, were excluded from the study. Finally, 52 patients with isolated CAE, and 35 individuals with NCA as the control group, were included into the study.

Clinical and laboratory characteristics of the study population are shown in Table 1. Current smoker, serglycin, hsCRP, total cholesterol, and platelet count were significantly higher in the CAE group. Lymphocyte counts were significantly lower in the CAE group compared with the control group. The angiographic characteristics of the patients in the coronary ectasia group are also shown in Table 2.

Multivariate logistic regression analysis revealed that serglycin and hsCRP levels were independently associated with the presence of CAE (Table 3). However, there was no association between serglycin and Markis classification (Fig. 1). In addition, there was a positive correlation between hsCRP levels and serglycin levels (p < 0.001) (Fig. 2).

In ROC curve analysis, the cut-off level of serglycin for the prediction of the presence of CAE was 13.5, with a sensitivity of 88.5% and a specificity of 84.8% (area under curve: 0.933, p < 0.001) (Fig. 3).

Table 1. Clinical and laboratory characteristics of the study population

| Variables | Control (n = 33; 38.8%) | Coronary artery ectasia (n = 52; 61.2%) | p |
|---|---|---|---|
| Male | 21 (63.6%) | 42 (80.8%) | 0.450 |
| Age [years] | 57 ± 10 | 58 ± 10 | 0.340 |
| Diabetes mellitus | 6 (18.2%) | 14 (26.9%) | 0.360 |
| Current smoker | 4 (7.7%) | 10 (30.4%) | 0.015 |
| Hypertension | 6 (18.2%) | 14 (26.9%) | 0.150 |
| Hyperlipidaemia | 3 (9.1%) | 8 (15.4%) | 0.266 |
| Body mass index [kg/m$^2$] | 24.45 ± 4.30 | 25.34 ± 5.12 | 0.135 |
| White blood cell count [×10$^9$/L] | 7.75 ± 1.34 | 8.17 ± 1.78 | 0.252 |
| Haemoglobin [g/L] | 14.54 ± 1.2 | 13.7 ± 1.5 | 0.190 |
| Neutrophil count [×10$^9$/L] | 4.96 ± 1.48 | 5.21 ± 1.49 | 0.455 |
| Lymphocyte count [×10$^9$/L] | 3.58 ± 0.93 | 3.1 ± 0.77 | 0.048 |
| Monocyte count [×10$^9$/L] | 0.54 ± 0.15 | 0.60 ± 0.21 | 0.196 |
| Platelet count [×10$^9$/L] | 231 ± 63 | 277 ± 74 | 0.003 |
| Total cholesterol [mmol/L] | 4.04 ± 0.65 | 4.77 ± 0.98 | 0.001 |
| Low density lipoprotein [mmol/L] | 2.67 ± 0.67 | 2.93 ± 0.83 | 0.093 |
| High density lipoprotein [mmol/L] | 1.17 ± 0.28 | 1.09 ± 0.26 | 0.239 |
| Triglycerides [mmol/L] | 1.39 (1.14–1.99) | 1.53 (1.19–1.75) | 0.184 |
| Creatinine [mg/dL] | 64.8 ± 17.5 | 70.9 ± 16.1 | 0.102 |
| Left ventricular ejection fraction [%] | 61 ± 4 | 59 ± 5 | 0.629 |
| High sensitive C-reactive protein [nmol/L] | 23.8 (11.4–40.0) | 91.4 (50.0–135.2) | < 0.001 |
| Serglycin [ng/mL] | 12.06 ± 1.55 | 16.63 ± 2.58 | < 0.001 |

Data are presented as mean ± standard deviation, number and percentage (in brackets), or median and interquartile range

Table 2. Angiographic characteristics of patients in the coronary ectasia group

| | |
|---|---|
| **Markis classification** | |
| Type 1 | 7 (13.5%) |
| Type 2 | 7 (13.5%) |
| Type 3 | 4 (7.6%) |
| Type 4 | 34 (65.4%) |
| **Number of ectatic coronary arteries** | |
| 1 | 30 (57.6%) |
| 2 | 9 (17.4%) |
| 3 | 13 (25.0%) |
| **Coronary artery ectasia distribution** | |
| Left anterior descending artery | 25 (48.1%) |
| Left circumflex artery | 20 (38.5%) |
| Right coronary artery | 29 (55.8%) |

Data are presented as numbers and percentages (in brackets)

Finally, the patients were divided into two subgroups based on a serglycin cut-off level of 13.5 ng/mL (Table 4). The number of patients with CAE was significantly higher among the patients with serglycin > 13.5 ng/mL ($p < 0.001$). Lymphocyte count was lower whereas total cholesterol and hsCRP levels were significantly higher, in the high serglycin subgroup.

## DISCUSSION

In the present study, we demonstrated that serglycin provided relevant information regarding the presence of isolated CAE. We found that patients with isolated CAE have significantly greater serglycin compared to control subjects with NCA groups. Serglycin was also statistically significantly correlated with hsCRP levels, showing its strong relation with systemic inflammation. However, the serglycin level did not change according to the group of Markis classification.

With the increase in the number of patients undergoing coronary angiography, we identify CAE in more patients. CAE is an independent predictor of mortality, and the mortality rate of patients with non-obstructive coronary artery aneurysms is similar to that of patients with multivessel disease [13]. In a large cohort study on CAE, the five-year mortality in patients with coronary artery aneurysm was reported as 26% in 1983 [1]. On the other hand, two recent studies found significantly lower mortality rates in CAE patients. In the first

Table 3. Multivariate logistic regression analysis showing independent predictors of coronary artery ectasia

| Variables | Univariate analysis | | Multivariate analysis | |
|---|---|---|---|---|
| | p | OR (95% CI) | p | Adjusted OR (95% CI) |
| Low-density lipoprotein | 0.290 | 1.352 (0.910–1.792) | | |
| Total cholesterol | 0.005 | 1.015 (1.005–1.020) | 0.523 | 1.003 (0.910–1.096) |
| Platelet count | 0.085 | 1.050 (0.715–1.385) | | |
| Lymphocyte count | 0.155 | 0.903 (0.713–1.093) | | |
| Current smoker | 0.002 | 1.150 (1.018–1.282) | 0.370 | 1.040 (0.980–1.100) |
| Serglycin | < 0.001 | 1.930 (1.310–2.550) | < 0.001 | 1.755 (1.340–2.170) |
| High-sensitivity C-reactive protein | < 0.001 | 1.585 (1.380–1.790) | 0.040 | 1.290 (1.040–1.540) |

CI — confidence interval; OR — odds ratio

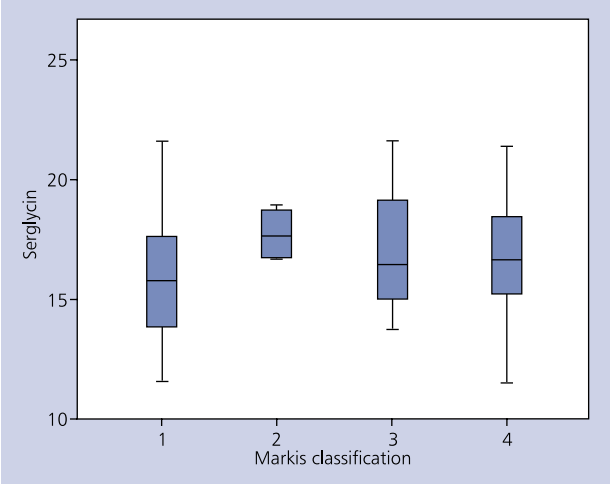


Figure 1. Serglycin levels according to number of Markis classification groups

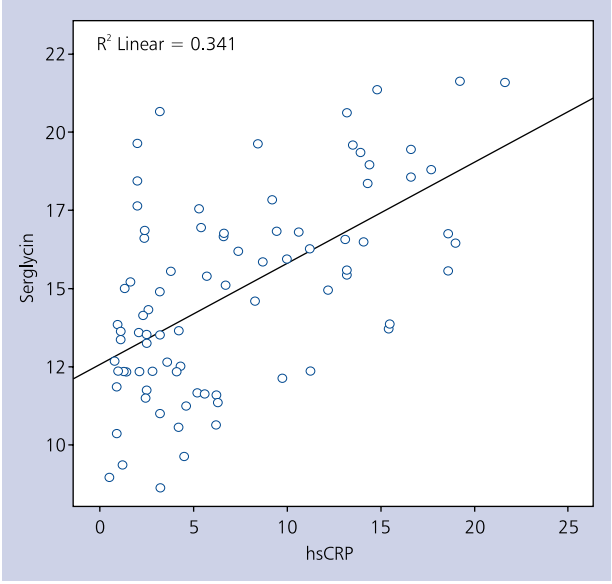


Figure 2. Correlation of serglycin and high-sensitivity C-reactive protein (hsCRP) levels

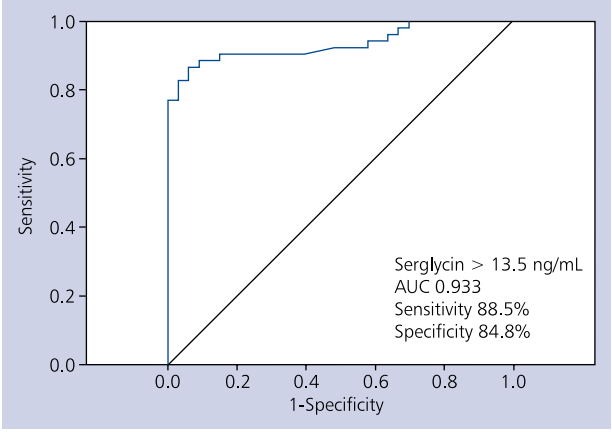


Figure 3. Receiver operating characteristics analysis for serglycin level to predict isolated coronary artery ectasia; AUC — area under curve

study, the cardiovascular mortality rate was 2% after a mean follow-up of 49 ± 21 months in 258 patients with CAE [14]. The second study, which included 540 patients with CAE, reported 2.22% mortality after 36 months of follow-up [15]. The decrease in mortality over time may be due to better management of CAE and improvements in medicine. The pathophysiological mechanism of CAE has also become an important research topic because of the high mortality rate. Although the aetiology and pathophysiology of CAE are still unclear, some pathological mechanisms have been proposed. CAE is considered to be a large positive remodelling of the atherosclerotic coronary artery [16]. The most emphasised mechanisms of this remodelling are the enzymatic degradation of the extracellular matrix and the thinning of the tunica media layer of the vessel due to severe chronic inflammation [17]. Recently, we also demonstrated that novel inflammatory markers such as platelet-to-lymphocyte and monocyte-to-high–density lipoprotein cholesterol ratios

Table 4. Study population according to serglycin level

| Variables | Serglycin ≤ 13.5 ng/mL (n = 34; 40.0%) | Serglycin > 13.5 ng/mL (n = 51; 60.0%) | p |
|---|---|---|---|
| Male | 25 (73.5%) | 38 (74.5%) | 0.850 |
| Age [years] | 58 ± 10 | 57 ± 11 | 0.721 |
| Diabetes mellitus | 9 (26.5%) | 11 (21.5%) | 0.371 |
| Current smoker | 4 (11.8%) | 10 (19.6%) | 0.105 |
| Hypertension | 8 (23.5%) | 12 (23.5%) | 1.000 |
| Hyperlipidaemia | 4 (11.7%) | 7 (13.7%) | 0.481 |
| White blood cell count [$\times 10^9$/L] | 7.77 ± 1.37 | 8.14 ± 1.75 | 0.322 |
| Haemoglobin [g/L] | 14.1 ± 1.3 | 14.0 ± 1.5 | 0.825 |
| Neutrophil count [$\times 10^9$/L] | 4.98 ± 1.38 | 5.18 ± 1.53 | 0.543 |
| Lymphocyte count [$\times 10^9$/L] | 4.30 ± 1.63 | 3.36 ± 1.19 | 0.035 |
| Monocyte count [$\times 10^9$/L] | 0.53 ± 0.14 | 0.61 ± 0.21 | 0.082 |
| Platelet count [$\times 10^9$/L] | 238 ± 65 | 267 ± 77 | 0.065 |
| Total cholesterol [mg/dL] | 4.01 ± 0.60 | 4.71 ± 0.98 | < 0.001 |
| Low density lipoprotein [mmol/L] | 2.67 ± 0.67 | 2.95 ± 0.83 | 0.128 |
| High density lipoprotein [mmol/L] | 1.14 ± 0.28 | 1.09 ± 0.23 | 0.453 |
| Triglycerides [mmol/L] | 3.37 (2.72–4.01) | 3.48 (2.72–4.64) | 0.079 |
| Creatinine [mmol/L] | 67.1 ± 19.8 | 69.4 ± 14.5 | 0.554 |
| Left ventricular ejection fraction [%] | 61 ± 5 | 60 ± 6 | 0.377 |
| High-sensitivity C-reactive protein [mg/L] | 30.4 (11.4-49.5) | 80.9 (11.4-134.3) | < 0.001 |
| Isolated coronary artery ectasia | 6 (17.6%) | 46 (90.2%) | < 0.001 |

Data are presented as mean ± standard deviation, number and percentage (in brackets), or median and interquartile range

were significantly elevated in the isolated CAE group when compared to the obstructive CAD and NCAs [18, 19]. As shown in various studies, inflammation is a well-known mechanism during the development and progression of atherosclerosis.

Serglycin is a dominant intracellular proteoglycan expressed by immune cells, where it interacts with numerous inflammatory mediators, such as proteases, chemokines, cytokines, and growth factors [8, 9]. In a recent study serglycin was found to be among the most abundantly expressed proteins in adiposities of epicardial adipose tissue in patients with CAD. It was also demonstrated that tumour necrosis factor-alpha (TNF-$\alpha$) induces expression and secretion of serglycin in adipocytes. These observations suggest that serglycin and TNF-$\alpha$ probably contribute to the development and progression of CAD through cross-talk between macrophages and adiposities [20].

Circulating serglycin might also participate in the mechanism of systemic vascular insult and atheromatous change [21, 22]. The biosynthesis of serglycin is up-regulated by lipopolysaccharides in macrophages [21, 22], TNF in endothelial cells, and interleukin 1-beta in smooth muscle cells [10].

On the basis of these findings and the pathophysiological role of inflammation in isolated CAE, we hypothesised that serglycin might be associated with isolated CAE. We found that serglycin was significantly higher in patients with CAE, but there was no difference in types of Markis classification. Thus, there is an inflammatory status in patients with CAE, but additional factors affecting the severity or extensiveness of CAE might also be present. Another finding of our study is that there was a positive correlation between hsCRP level and serglycin, which also supports the systemic inflammation role in our study. Finally, our findings indicated that serglycin > 13.5 ng/mL was significantly and independently related with isolated CAE compared to NCA groups.

### *Limitations of the study*

Our study has some limitations. First, it was a cross-sectional study; therefore, we did not analyse the follow-up data adequately. Second, inflammatory markers other than hsCRP, such as TNF-$\alpha$ matrix metal proteinases and interleukin-6, were not analysed, and therefore we did not compare them with serglycin level. In addition, we did not have a control group that included obstructive CAD. A further limitation is

that the evaluation of coronary angiography was visual; we did not use intravascular ultrasound or optical coherence tomography. Another limitation is the relatively small number of patients that was included in the study. The final limitation of our study is the lack of information about taking drugs such as nitrates, angiotensin (AT) converting enzyme, and AT-II inhibitors, which can affect the occurrence of CAE.

## CONCLUSIONS

In conclusion, serglycin level is significantly and independently higher in patients with CAE, but there might be some additional factors determining the severity and extensiveness of CAE. Our findings suggest that inflammation may play a role in the development of CAE. Serglycin may achieve prediction of isolated CAE in the time of clinical practice. We believe that further, larger studies are needed to clarify the relationship between serglycin and CAE.

***Conflict of interest:*** *none declared*

## *References*


1. Swaye PS, Fisher LD, Litwin P, et al. Aneurysmal coronary artery disease. Circulation. 1983; 67(1): 134–138, doi: 10.1161/01.cir.67.1.134, indexed in Pubmed: 6847792.
2. Sarli B, Baktir AO, Saglam H, et al. Neutrophil-to-lymphocyte ratio is associated with severity of coronary artery ectasia. Angiology. 2014; 65(2): 147–151, doi: 10.1177/0003319713488932, indexed in Pubmed: 23657176.
3. Markis JE, Joffe CD, Cohn PF, et al. Clinical significance of coronary arterial ectasia. Am J Cardiol. 1976; 37(2): 217–222, doi: 10.1016/0002-9149(76)90315-5, indexed in Pubmed: 1108631.
4. al-Harthi SS, Nouh MS, Arafa M, et al. Aneurysmal dilatation of the coronary arteries: diagnostic patterns and clinical significance. Int J Cardiol. 1991; 30(2): 191–194, indexed in Pubmed: 2010241.
5. Krüger D, Stierle U, Herrmann G, et al. Exercise-induced myocardial ischemia in isolated coronary artery ectasias and aneurysms ("dilated coronaropathy"). J Am Coll Cardiol. 1999; 34(5): 1461–1470, doi: 10.1016/s0735-1097(99)00375-7.
6. Mavrogeni S. Coronary artery ectasia: from diagnosis to treatment. Hellenic J Cardiol. 2010; 51(2): 158–163, indexed in Pubmed: 20378518.
7. Turhan H, Erbay AR, Yasar AS, et al. Comparison of C-reactive protein levels in patients with coronary artery ectasia versus patients with obstructive coronary artery disease. Am J Cardiol. 2004; 94(10): 1303–1306, doi: 10.1016/j.amjcard.2004.07.120, indexed in Pubmed: 15541253.
8. Kolset SO, Pejler G. Serglycin: a structural and functional chameleon with wide impact on immune cells. J Immunol. 2011; 187(10): 4927–4933, doi: 10.4049/jimmunol.1100806, indexed in Pubmed: 22049227.
9. Niemann CU, Abrink M, Pejler G, et al. Neutrophil elastase depends on serglycin proteoglycan for localization in granules. Blood. 2007; 109(10): 4478–4486, doi: 10.1182/blood-2006-02-001719, indexed in Pubmed: 17272511.
10. Korpetinou A, Skandalis SS, Labropoulou VT, et al. Serglycin: at the crossroad of inflammation and malignancy. Front Oncol. 2014; 3: 327, doi: 10.3389/fonc.2013.00327, indexed in Pubmed: 24455486.
11. Investigators CP. The national heart, lung, and blood institute coronary artery surgery study. Circulation. 1981; 63(suppl): 1–81.
12. Reine TM, Vuong TT, Jenssen TG, et al. Serglycin secretion is part of the inflammatory response in activated primary human endothelial cells in vitro. Biochim Biophys Acta. 2014; 1840(8): 2498–2505, doi: 10.1016/j.bbagen.2014.02.002, indexed in Pubmed: 24513305.
13. Baman TS, Cole JH, Devireddy CM, et al. Risk factors and outcomes in patients with coronary artery aneurysms. Am J Cardiol. 2004; 93(12): 1549–1551, doi: 10.1016/j.amjcard.2004.03.011, indexed in Pubmed: 15194034.
14. Turan T, Akyuz AR, Aykan AC, et al. Plasma endocan levels in patients with isolated coronary artery ectasia. Angiology. 2016; 67(10): 932–936, doi: 10.1177/0003319716637789, indexed in Pubmed: 26980772.
15. Varol E, Uysal BA, Dogan A, et al. Mean platelet volume has a prognostic value in patients with coronary artery ectasia. Clin Appl Thromb Hemost. 2012; 18(4): 387–392, doi: 10.1177/1076029611427441, indexed in Pubmed: 22146577.
16. Paik DC, Ramey WG, Dillon J, et al. The nitrite/elastin reaction: implications for in vivo degenerative effects. Connect Tissue Res. 1997; 36(3): 241–251, indexed in Pubmed: 9512892.
17. Antoniadis AP, Chatzizisis YS, Giannoglou GD. Pathogenetic mechanisms of coronary ectasia. Int J Cardiol. 2008; 130(3): 335–343, doi: 10.1016/j.ijcard.2008.05.071, indexed in Pubmed: 18694609.
18. Kundi H, Gök M, Çetin M, et al. Relationship between platelet-to-lymphocyte ratio and the presence and severity of coronary artery ectasia. Anatol J Cardiol. 2016; 16(11): 857–862, doi: 10.14744/AnatolJCardiol.2015.6639, indexed in Pubmed: 27109243.
19. Kundi H, Gok M, Kiziltunc E, et al. Relation Between Monocyte to High-Density Lipoprotein Cholesterol Ratio With Presence and Severity of Isolated Coronary Artery Ectasia. Am J Cardiol. 2015; 116(11): 1685–1689, doi: 10.1016/j.amjcard.2015.08.036, indexed in Pubmed: 26434515.
20. Imoto-Tsubakimoto H, Takahashi T, Ueyama T, et al. Serglycin is a novel adipocytokine highly expressed in epicardial adipose tissue. Biochem Biophys Res Commun. 2013; 432(1): 105–110, doi: 10.1016/j.bbrc.2013.01.078, indexed in Pubmed: 23376071.
21. Zernichow L, Abrink M, Hallgren J, et al. Serglycin is the major secreted proteoglycan in macrophages and has a role in the regulation of macrophage tumor necrosis factor-alpha secretion in response to lipopolysaccharide. J Biol Chem. 2006; 281(37): 26792–26801, doi: 10.1074/jbc.M512889200, indexed in Pubmed: 16807245.
22. Kolseth IB, Reine TM, Vuong TT, et al. Serglycin is part of the secretory repertoire of LPS-activated monocytes. Immun Inflamm Dis. 2015; 3(1): 23–31, doi: 10.1002/iid3.47, indexed in Pubmed: 25866637.


**Cite this article as:** Kundi H, Gök M, Topçuoğlu C, Ornek E. Association of serglycin levels with isolated coronary artery ectasia. Kardiol Pol. 2017; 75(10): 990–996, doi: 10.5603/KP.a2017.0119.

# Związek stężenia serglicyny z izolowanym tętniakowatym poszerzeniem tętnic wieńcowych

Harun Kundi, Murat Gök, Canan Topçuoğlu, Ender Ornek

Numune Education and Research Hospital Cardiology Department, Ankara, Turcja

**Streszczenie**

**Wstęp:** Wiadomo, że serglicyna odgrywa istotną rolę w stanie zapalnym, lecz zależności między tętniakowatym poszerzeniem tętnic wieńcowych (CAE) a serglicyną nadal nie są znane.

**Cel:** Celem badania była analiza związku między stężeniem serglicyny a izolowanym CAE.

**Metody:** Do badania włączono 52 chorych z izolowanym CAE i 35 osób z prawidłowym obrazem tętnic wieńcowych w koronarografii. Określono klasyfikację tętniaków wg Markisa oraz liczbę poszerzonych tętnic wieńcowych. Oznaczono stężenie serglicyny.

**Wyniki:** W analizie regresji logistycznej wykazano, że stężenia serglicyny i białka C-reaktywnego były niezależnie związane z obecnością CAE. W analizie krzywych ROC punkt odcięcia dla prognozowania izolowanego CAE wynosił 13,5, a czułość i swoistość metody — odpowiednio 88,5% i 84,8%. Nie stwierdzono jednak żadnych zależności między stężeniem serglicyny a klasyfikacją wg Markisa.

**Wnioski:** Stężenie serglicyny jest istotnie wyższe u osób z CAE i stanowi niezależny czynnik predykcyjny tego zaburzenia.

**Słowa kluczowe:** tętniakowate poszerzenie tętnic wieńcowych, zapalenie, serglicyna

Kardiol Pol 2017; 75, 10: 990–996

**Adres do korespondencji:**
Harun Kundi, MD, Cardiology Department, Numune Education and Research Hospital, 06100, Sıhhiye, Ankara, Turkey, tel: 00905323529393, e-mail: harunkundi@hotmail.com

**Praca wpłynęła:** 22.12.2016 r. **Zaakceptowana do druku:** 18.05.2017 r. **Data publikacji as AoP:** 01.06.2017 r.