Kardiologia Polska 2017; 75, 1: 65–70; DOI: 10.5603/KP.a2016.0108 ISSN 0022–9032

ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

The relation between vitamin B12 and SYNTAX score

Levent Cerit, Hamza Duygu, Kamil Gulsen, Hatice Kemal, Ozgur Tosun, Barcin Ozcem, Zeynep Cerit, Aziz Gunsel

Near East University, Cyprus

Abstract

Background: Vitamin B12 is required in the metabolism of homocysteine. Vitamin B12 deficiency has been implicated in endothelial dysfunction and cardiovascular disease via hyperhomocysteinaemia. However, the association of vitamin B12 and the severity of coronary artery disease has not been studied to date.

Aim: This study was conducted with the aim of evaluating the relationship between vitamin B12 and SYNTAX score.

Methods: Medical records of consecutive patients who underwent coronary artery bypass grafting surgery were retrospectively reviewed. The study group consisted of 127 patients. Vitamin B12, other biochemical parameters, clinical and echocardiographic parameters, and SYNTAX score were evaluated for all patients.

Results: Patients with vitamin B12 deficiency had a higher prevalence of cardiovascular risk factors such as diabetes mellitus, and history of transient ischaemic attack/stroke and heart failure. The SYNTAX score was significantly higher in patients with vitamin B12 deficiency (29.2 ± 4.9 vs. 22.5 ± 4.5, $p < 0.05$).

Conclusions: In our study, we found a significant relationship between vitamin B12 deficiency and SYNTAX score, demonstrating the severity and complexity of coronary artery disease.

Key words: vitamin B12, SYNTAX score, coronary artery bypass graft surgery

Kardiol Pol 2017; 75, 1: 65–70

INTRODUCTION

Vitamin B12 is required in the metabolism of homocysteine (Hcy), which occurs through remethylation to methionine or transsulphuration to cysteine. Methylation of Hcy, catalysed by methionine synthase produces methionine [1]. The enzyme methylenetetrahydrofolate-reductase (MTHFR) is responsible for the reduction of 5,10-methylene-THF to 5-methyl-THF, where vitamin B12 acts as a cofactor [2]. Hcy-mediated increased lipid peroxidation and generation of free radicals results in inflammation and acute endothelial dysfunction, which accelerates atherosclerotic process predisposing to cardiovascular disease (CVD). Coronary artery disease (CAD) is associated with higher levels of Hcy [3], which plays a permissive role in endothelial damage. Low vitamin B12 concentration and hyperhomocysteinaemia are common in general [4].

Many studies have been undertaken to examine the relation between plasma Hcy and coronary heart disease [5, 6]. The general outcome supports the hypothesis that an elevated plasma Hcy concentration leads to an increased risk of CVD. The SYNTAX score (SS) is an angiographic scoring system and is widely used to evaluate the severity and complexity of CAD. It is used in the estimation of long-term outcomes of CAD and in the selection of the treatment modality [7]. However, there are few studies that showed the association of vitamin B12 with cardiovascular risk factors in patients with known CAD.

The relationship between vitamin B12 and SS has not been studied. In light of this knowledge, we assessed the relationship between vitamin B12 and SS.

METHODS

The study group consisted of 127 consecutive patients who underwent on-pump coronary artery bypass grafting surgery. Data of patients were retrospectively analysed for the demographic features, echocardiographic parameters, biochemical parameters, vitamin B12 level, and SS. The study was approved by the local ethics committee.

Address for correspondence:
Levent Cerit, Assistant Professor, Near East University Hospital, Department of Cardiology, Near East Boulevard, Nicosia, Cyprus, e-mail: drcerit@hotmail.com
Received: 28.01.2016 **Accepted:** 12.05.2016 **Available as AoP:** 08.07.2016

The patient's data including age, gender, history of hypertension, diabetes mellitus (DM), heart failure (HF), transient ischaemic attack (TIA)/stroke, and echocardiographic variables such as left ventricular ejection fraction (LVEF), left atrial diameter, and biochemical and haematological parameters were received from medical records.

Vitamin B12 was measured by Ion Capture Microparticle enzyme immunoassay with commercial kits supplied by Abbott Laboratory. Intra assay and inter assay precision was < 10% for above parameters. B12 deficiency was defined as < 200 pg/mL. For comparison we divided all subjects into two groups according to vitamin B12 levels: < 200 pg/mL — group 1; > 200 pg/mL — group 2. SYNTAX score was divided into two groups: ≥ 23 points — high, < 23 points — low.

Echocardiographic examination

All patients underwent transthoracic echocardiography using a Vivid S5 (GE healthcare) echocardiography device and Mass S5 probe (2–4 MHz). Standard two-dimensional and colour flow Doppler views were acquired according to the guidelines of the American Society of Echocardiography and the European Society of Echocardiography [8, 9]. The ejection fraction was measured according to Simpson's method. Left atrial diameter in parasternal long axis view was measured using two-dimensional echocardiography at the end of left ventricular systole.

Coronary angiography and SYNTAX score analysis

Coronary angiography was performed by the Judkins technique. All lesions causing ≥ 50% stenosis in a coronary artery with a diameter ≥ 1.5 mm were included in the SS calculation. For calculation, website software (http://www. SYNTAXcore.com) was used. Scoring was performed for each patient in keeping with the following parameters: coronary dominance, number of lesions, segments included per lesion, the presence of total occlusion, bifurcation, trifurcation, aorto-ostial lesion, severe tortuosity, calcification, thrombus, diffuse/small vessel disease, and lesion length > 20 mm. SYNTAX score was evaluated separately by two interventional cardiologists blinded to the study protocol and patient characteristics. In the case of a contradiction between two results, the opinion of a senior interventional cardiologist was sought and a common consensus was obtained.

Statistical analysis

All descriptive statistics were calculated and shown for each variable. Continuous variables were expressed as mean ± standard deviation (median), and categorical variables were expressed as frequency (n) and percentage (%). The Shapiro-Wilk test of normality was used to evaluate the distribution of continuous variables. Either Student's t-test or Mann-Whitney U-test was used to compare two groups for continuous variables, depending on their distribution characteristics. For categorical variable comparisons, Pearson χ^2 test or Fisher's Exact test were used, where appropriate. The level of significance was accepted as 0.05. Receiver-operator characteristic (ROC) curve analysis was applied to evaluate the diagnostic performance of vitamin B12 for differentiating low and high SS patients. SYNTAX score differences between B12-deficient and normal groups were further analysed with adjustment for age, HF, TIA/stroke, and DM using general linear model-analysis of covariance (ANCOVA). All statistical analysis was performed with the SPSS (version 18.0, SPSS Inc., Chicago, Illinois) software package.

Table 1. Patient characteristics

Demographical characteristics and health conditions		Vitamin B12		P
		Group 1 (< 200 pg/mL)	Group 2 (> 200 pg/mL)	
Age [years]; mean ± SD (median)		68.3 ± 9.3 (69)	64.2 ± 9.4 (63)	**0.043**
Body mass index [kg/m^2]; mean ± SD (median)		27.7 ± 3.8 (26.9)	26.8 ± 3.9 (26.7)	0.831
Smoking	+	15 (57.6%)	45 (45%)	0.068
	–	11 (42.4%)	55 (55%)	
Gender	Male	22 (84.6%)	88 (88%)	0.741
	Female	4 (15.4%)	12 (12%)	
Hypertension	+	26 (100%)	90 (90.%)	0.121
	–	0 (0%)	10 (10%)	
Diabetes mellitus	+	19 (73.1%)	38 (38%)	**0.002**
	–	7 (26.9%)	62 (62%)	
Transient ischaemic attack/stroke	+	4 (15.4%)	2 (2%)	**0.016**
	–	22 (84.6%)	98 (98.1%)	
Heart failure	+	9 (34.6%)	6 (6%)	**< 0.001**
	–	17 (65.4%)	94 (94%)	

Table 2. Laboratory and echocardiography parameters

Laboratory and echocardiographic variables	Vitamin B12		P
	Group 1 (< 200 pg/mL)	Group 2 (> 200 pg/mL)	
Haemoglobin [g/dL]	13.3 ± 1.6 (13.1)	13.7 ± 1.6 (13.7)	0.637
Platelet [$10^3/\mu L$]	225.9 ± 64.9 (219.0)	230.2 ± 65.5 (226.0)	0.924
White blood cell [$10^3/\mu L$]	7.6 ± 1.4 (7.5)	7.7 ± 2.4 (7.4)	0.819
Mean platelet volume [fL]	10.5 ± 1.1 (10.4)	10.4 ± 0.9 (10.4)	0.519
Mean corpuscular volume [fL]	96.2 ± 12.4 (102.6)	86.7 ± 9.2 (91.8)	**< 0.005**
Neutrophil [$10^3/\mu L$]	4.3 ± 1.2 (4.3)	4.5 ± 1.6 (4.2)	0.905
Red blood cell distribution width [%]	14.8 ± 2.3 (15.8)	12.3 ± 1.4 (13.1)	0.002
Lymphocyte [$10^3/\mu L$]	1.9 ± 0.8 (1.9)	2.3 ± 1.4 (2.0)	0.339
Neutrophil to lymphocyte ratio	2.8 ± 21.8 (2.7)	2.2 ± 1.2 (1.9)	0.143
Platelet large cell ratio [%]	30.8 ± 6.1 (28.8)	29.1 ± 11.4 (28.0)	0.121
Sedimentation [mm/h]	32.9 ± 27.3 (25.0)	24.1 ± 19.5 (19.6)	0.306
Urea [mg/dL]	44.6 ± 19.6 (40.0)	38.8 ± 17.2 (34)	0.144
Creatinine [mg/dL]	1.1 ± 0.3 (1.0)	0.95 ± 0.25 (0.9)	**0.011**
Fasting plasma glucose [mg/dL]	119.1 ± 38.9 (102.5)	127.1 ± 45.6 (110.0)	0.399
C-reactive protein [mg/L]	2.5 ±1.7 (0.9)	1.5 ± 0.9 (0.3)	**0.030**
Total cholesterol [mg/dL]	183.7 ± 48.6 (182.0)	182.6 ± 51.6 (178.0)	0.908
High density lipoprotein [mg/dL]	39.1 ± 6.7 (38.0)	39.0 ± 11.3 (37.0)	0.432
Low density lipoprotein [mg/dL]	114.7 ± 38.1 (111.5)	114.6 ± 45.6 (106.0)	0.758
Triglyceride [mg/dL]	198.3 ± 104.2 (191.5)	150.8 ± 60.1 (144.5)	0.098
Albumin [g/dL]	4.1 ± 0.4 (4.1)	4.2 ± 0.3 (4.1)	0.790
Uric acid [mg/dL]	6.2 ± 1.3 (6.1)	5.6 ± 1.3 (5.5)	0.576
Folic acid [ng/mL]	7.9 ± 2.3 (8.5)	8.6 ± 3.1 (9.1)	0.874
Haemoglobin A1c [%]	7.3 ± 2.4 (6.1)	7.3 ± 1.9 (6.5)	0.693
Left atrium [mm]	40.1 ± 3.6 (39.0)	38.5 ± 4.5 (38.0)	0.052
Ejection fraction [%]	50.5 ± 8.2 (50.0)	54.9 ± 7.4 (55.0)	**0.011**
SYNTAX score [points]	29.2 ± 4.9 (28.0)	22.5 ± 4.5 (22.2)	**< 0.001**
SYNTAX score — low (< 23 points)	1 (3.8%)	51 (51%)	**< 0.001**
SYNTAX score — high (≥ 23 points)	25 (96.2%)	49 (49%)	

RESULTS

The present study included 126 consecutive patients, and 26 (20.6%) patients had vitamin B12 deficiency, 52 (41.2%) patients had low SS, and 74 (58.7%) had high SS. The main characteristics of patients with vitamin B12 deficiency (group 1) and normal vitamin B12 level (group 2) are shown in Table 1. Group 1 patients were older and had higher prevalence of cardiovascular risk factors such as DM, TIA/stroke, and HF (Table 1). Laboratory and echocardiographic parameters are presented in Table 2. Creatinine, C-reactive protein (CRP), red blood cell distribution width (RDW), and mean corpuscular volume (MCV) level were higher in group 1 (1.1 ± 0.3 mg/dL vs. 0.95 ± 0.25 mg/dL, $p = 0.011$, 2.5 ± 1.7 mg/dL vs. 1.5 ± 0.9 mg/dL, $p = 0.03$, 14.8 ± 2.3% vs. 12.3 ± 1.4%, $p = 0.002$, 96.2 ± 12.4 fL vs. 86.7 ± 9.2 fL, $p < 0.005$, respectively) (Table 2). LVEF was lower in group 1 (50.5 ± 8.2 vs. 54.9 ± 7.4, $p = 0.011$) (Table 2). Folic acid levels were similar in both groups (7.9 ± 2.3 vs. 8.6 ± 3.1, $p = 0.874$) (Table 2). There was a significant negative correlation between SS and vitamin B12 (R: –0.402, $p < 0.005$). SYNTAX score was significantly increased in patients with vitamin B12 deficiency (29.2 ± 4.9 vs. 22.5 ± 4.5, $p < 0.05$). SYNTAX score level of vitamin B12-deficient and normal groups was further analysed with adjustment for age, HF, TIA/stroke, and DM. The difference was found to be statistically significant ($p < 0.001$).

In ROC analysis, a cut-off value was determined for vitamin B12 in high SS. The cut-off value was 294.5 pg/mL (AUC = 0.702; 84.6% sensitivity, 55% specificity, 95% CI 0.613–0.792; Fig. 1).

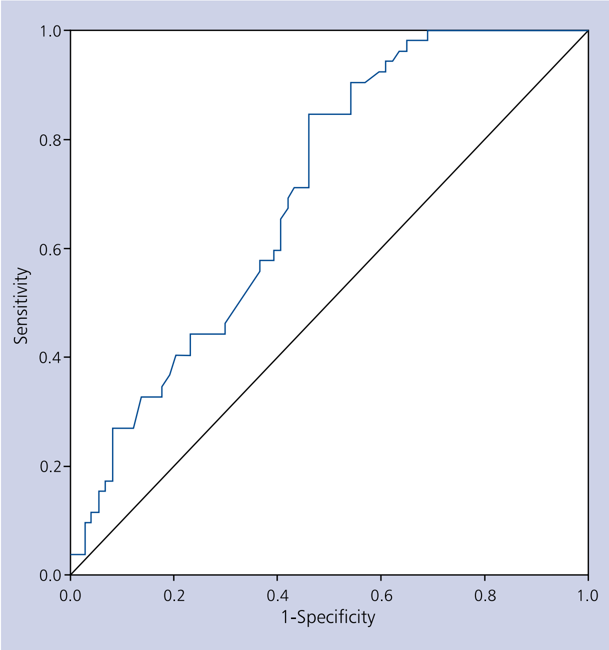

Figure 1. Receiver-operator characteristic curve for vitamin B12 for predicting high SYNTAX score

DISCUSSION

Vascular studies have illustrated impaired arterial endothelial function and increased carotid intima–media thickness (IMT) in vitamin B12-deficient populations. Su et al. [10, 11] have found significantly lower vitamin B12, higher Hcy, and lower brachial artery resistance in Chinese vegetarians. Kwok et al. [12] demonstrated vascular dysfunction in Chinese non-smoking vegetarians with higher plasma Hcy, and lower vitamin B12 levels. Abnormal vascular function and structure were also found. In association with metabolic vitamin B12 deficiency, their carotid IMT and brachial flow-mediated dilation (FMD) were significantly worse than in patients with normal vitamin B12 levels [13, 14]. Waldman et al. [15] showed increased cardiovascular risk factors among German vegans and found that vitamin B12 was the only predictor of Hcy concentration. Hermann et al. [16] reported similar findings. Karabudak et al. [17] reported a similar correlation between vitamin B12 and serum Hcy concentration among Turkish women. Vitamin B12 level was the most significant predictor of Hcy among vegans and vegetarians [18].

Studies haves illustrated several mechanisms for the role of hyperhomocysteinaemia in atherogenesis, including auto-oxidation of Hcy, which leads to synthesis of compounds related to the initiation of atherogenic process, such as superoxide, hydrogen peroxide, and superoxide anion. These compounds have been implicated in atherogenesis through oxidation of low-density lipoprotein, suppression of nitric oxide synthesis, arterial stiffness, endothelial inflammation, and foam cell formation [19, 20]. Chronic hyperhomocysteinaemia reduces the activity of superoxide dismutase, which is important in the dismutation of superoxide radicals [21]. Van Campenhout et al. [22] found a positive association between total Hcy concentration and severity of aortic calcification. Mahalle et al. [23] reported a negative association between vitamin B12 and a positive association between Hcy and triglyceride, very-low-density lipoprotein, and CRP, respectively. In our study, higher CRP and creatinine levels were found in patients with vitamin B12 deficiency.

Elevated MCV and RDW levels point out vitamin B12 deficiency. Tonelli et al. [24] reported a significantly increased risk of symptomatic HF in patients with higher RDW level. In our study, we found raised RDW and MCV level and decreased LVEF in patients with vitamin B12 deficiency.

Kwok et al. [25] reported the impact of vitamin B12 supplementation on vascular surrogates. Vitamin B12 supplementation significantly increased serum vitamin B12 level and lowered plasma Hcy, associated with significant improvement of brachial FMD and carotid IMT. Woo et al. [13] reported that vitamin B12 supplementation improved arterial stiffness, endothelial function, and carotid IMT, suggested because of the protective effect against atherosclerosis in subjects with subnormal vitamin B12 status. In our study, the cut-off vitamin B12 value predictive of high SS was 294.5 pg/mL. The relation between vitamin B12 and CAD is well known. Regarding our results, for a vitamin B12 level below 294.5 pg/mL in patients with CAD the need of replacement should be questioned. To determine the lower limit of vitamin B12 for replacement in patients with CAD further randomised trials are needed.

In light of this knowledge, we aim to evaluate the relation between SS and vitamin B12 status. The present study was the first to evaluate the relationship between vitamin B12 and SS. In our study there was a significant relation between vitamin B12 deficiency and increased SS. Further prospective and randomised studies with a larger number of patients are needed on this topic.

Limitations of the study

Our study has some limitations: First, the retrospective study design; second, the small sample size; third, the single measurement of vitamin B12 level was used; and fourth, the lack of Hcy levels.

CONCLUSIONS

To the best or our knowledge, this study is the first to evaluate the relation between vitamin B12 and SS. In our study there was a strong negative correlation between vitamin B12 and SS. Further prospective and randomised studies with a larger number of patients are required due to the relatively small number of patients in the present study.

Conflict of interest: *none declared*

References

1. Ng KC, Yong QW, Chan SP, Cheng A. Homocysteine, folate and vitamin B12 as risk factors for acute myocardial infarction in a southeast Asian population. Ann Acad Med Singapore, 2002; 31: 636–640.
2. Sadeghian S, Fallahi F, Salarifar M et al. Tehran Heart Center, homocysteine, vitamin B12 and folate levels in premature coronary artery disease. BMC Cardiovasc Disord, 2006; 6: 38. doi: 10.1186/1471-2261-6-38.
3. Wilcken DE, Wilcken B. The pathogenesis of coronary artery disease. A possible role for methionine metabolism. J Clin Invest, 1976; 57: 1079–1082. doi: 10.1172/JCI108350.
4. Yajnik CS, Deshpande SS, Lubree HG et al. Vitamin B12 deficiency and hyperhomocysteinemia in rural and urban Indians. J Assoc Physicians India, 2006; 54: 775–782.
5. Whincup PH, Refsum H, Perry IJ et al. Serum total homocysteine and coronary heart disease: prospective study in middle aged men. Heart, 1999; 82: 448–454.
6. Boushey CJ, Beresford SAA, Omenn GS, Motulsky AG. A quantitative assessment of plasma homocysteine as a risk factor for vascular disease. JAMA, 1995; 274: 1049–1057.
7. Aksakal E, Tanboğa IH, Kurt M. Predictors of coronary lesions complexity in patients with stable coronary artery disease. Angiology, 2013; 64: 304–309. doi: 10.1177/0003319712464815.
8. Lang RM, Bierig M, Devereux RB et al. Recommendations for chamber quantification. Eur J Echocardiogr, 2006; 7: 79–108.
9. Appleton CP, Jensen JL, Hatle L, Oh JK. Doppler evaluation of left and right ventricular diastolic function: a technical guide for obtaining optimal flow velocity recordings. J Am Soc Echocardiogr, 1997; 10: 271–292.
10. Su TC, Jeng JS, Wang JD et al. Homocysteine, circulating vascular cell adhesion molecule and carotid atherosclerosis in postmenopausal vegetarian women and omnivores. Atherosclerosis, 2006; 184: 356–362. doi: 10.1016/j.atherosclerosis.2005.04.022.
11. Su TC, Torng PL, Jeng JS et al. Arterial function of carotid and brachial arteries in postmenopausal vegetarians. Vasc Health Risk Manag, 2011; 7: 517–523. doi: 10.2147/VHRM.S18881.
12. Kwok T, Chook P, Tam L et al. Vascular dysfunction in Chinese vegetarians: an apparent paradox? J Am Coll Cardiol, 2005; 46: 1957–1958. doi: 10.1016/j.jacc.2005.07.054.
13. Woo KS, Chook P, Yip TWC et al. Folic acid and vitamin B-12 supplementation improves arterial function and structure in subjects with subnormal intake. Heart Lung Circ, 2008; 17: 201–202.
14. Woo KS, Chook P, Chiu ML et al. Vitamins B12 and C supplementation improves arterial reactivity and structure in passive smokers. Heart Lung Circ, 2012; 21: 524. doi: 10.1016/j.hlc.2012.03.118.
15. Waldman A, Koschizke JW, Leitzmann C, Hahn A. German vegan study; diet, life-style factors, and cardiovascular risk profile. Ann Nutr Metab, 2005; 49: 366–372. doi: 10.1159/000088888.
16. Herrmann W, Schorr H, Purschwitz K et al. Total homocysteine, vitamin B(12), and total antioxidant status in vegetarians. Clin Chem, 2001; 47: 1094–1101.
17. Karabudak E, Kiziltan G, Cigerim N. A comparison of some of the cardiovascular risk factors in vegetarian and omnivorous Turkish females. J Hum Nutr Diet, 2008; 21: 13–22. doi: 10.1111/j.1365-277X.2007.00831.x.
18. Bissoli L, Di Francesco V, Ballarin A et al. Effect of vegetarian diet on homocysteine levels. Ann Nutr Metab, 2002; 46: 73–79. doi: 10.1159/000057644.
19. Welch GN, Loscalzo J. Homocysteine and atherothrombosis. N Engl J Med, 1998; 338: 1042–1050. doi: 10.1056/NEJM199804093381507.
20. Shargorodsky M, Boaz M, Pasternak S et al. Serum homocysteine, folate, vitamin B12 levels and arterial stiffness in diabetic patients: which of them is really important in atherogenesis? Diabetes Metab Res Rev, 2009; 25: 70–75. doi: 10.1002/dmrr.902.
21. Dayal S, Arning E, Bottiglieri T et al. Cerebral vascular dysfunction mediated by superoxide in hyperhomocysteinemic mice. Stroke, 2004; 35: 1957–1962. doi: 10.1161/01.STR.0000131749.81508.18.
22. Van Campenhout A, Moran C.S, Parr A et al. Role of homocysteine in aortic calcification and osteogenic cell differentiation. Atherosclerosis, 2009; 202: 557–566. doi: 10.1016/j.atherosclerosis.2008.05.031.
23. Mahalle N, Kulkarni MV, Garg M.K, Naik SS. Vitamin B_{12} deficiency and hyperhomocysteinemia as correlates of cardiovascular risk factors in Indian subjects with coronary artery disease. J Cardiol, 2013; 61: 289–294. doi: 10.1016/j.jjcc.2012.11.009.
24. Tonelli M, Sacks F, Arnold M et al. Relation between red blood cell distribution width and cardiovascular event rate in people with coronary disease. Circulation, 2008; 117: 163–168.
25. Kwok T, Chook P, Qiao M et al. Vitamin B-12 supplementation improves arterial function in vegetarians with subnormal vitamin B-12 status. J Nutr Health Aging, 2012; 16: 569–573.

Cite this article as: Cerit L, Duygu H, Gulsen K et al. The relation between vitamin B12 and SYNTAX score. Kardiol Pol, 2017; 75: 65–70. doi: 10.5603/KP.a2016.0108.

Zależność między stężeniem witaminy B12 a oceną w skali SYNTAX

Levent Cerit, Hamza Duygu, Kamil Gulsen, Hatice Kemal, Ozgur Tosun, Barcin Ozcem, Zeynep Cerit, Aziz Gunsel

Near East University, Cypr

Streszczenie

Wstęp: Witamina B12 jest potrzebna do metabolizowania homocysteiny. Jej niedobór wiąże się z dysfunkcją śródbłonka i chorobami układu sercowo-naczyniowego poprzez hiperhomocysteinemię. Jednak dotychczas nie zbadano związku stężenia witaminy B12 ze stopniem ciężkości choroby wieńcowej.

Cel: Celem niniejszego badania była ocena zależności między stężeniem witaminy B12 a oceną w skali SYNTAX.

Metody: Przeprowadzono retrospektywną analizę danych medycznych chorych poddanych zabiegowi pomostowania aortalno-wieńcowego. Badana grupa liczyła 127 osób. U wszystkich pacjentów oceniano stężenie witaminy B12, inne parametry biochemiczne, objawy kliniczne, parametry echokardiograficzne oraz liczbę punktów wg skali SYNTAX.

Wyniki: U chorych z niedoborem witaminy B12 częściej występowały czynniki ryzyka sercowo-naczyniowego, takie jak cukrzyca, przebyte przemijające niedokrwienie mózgu/przebyty udar, niewydolność serca. Liczba punktów wg skali SYNTAX była istotnie większa u pacjentów z niedoborem witaminy B12 (29,2 ± 4,9 *vs*. 22,5 ± 4,5; $p < 0,05$).

Wnioski: W badaniu stwierdzono istotną zależność między niedoborem witaminy B12 a oceną w skali SYNTAX odzwierciedlającą stopień ciężkości i złożoność choroby wieńcowej.

Słowa kluczowe: witamina B12, skala SYNTAX, pomostowanie aortalno-wieńcowe

Kardiol Pol 2017; 75, 1: 65–70

Adres do korespondencji:
Levent Cerit, Assistant Professor, Near East University Hospital, Department of Cardiology, Near East Boulevard, Nicosia, Cyprus, e-mail: drcerit@hotmail.com

Praca wpłynęła: 28.01.2016 r. **Zaakceptowana do druku:** 12.05.2016 r. **Data publikacji AoP:** 08.07.2016 r.