Kardiologia Polska 2014; 72, 12: 1388–1393; DOI: 10.5603/KP.a2014.0114 ISSN 0022–9032

ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Impact of serum alkaline phosphatase level on coronary collateral circulation

Ahmet Karabulut[1], Irfan Sahin[2], Ilhan Ilker Avci[2], Ertugrul Okuyan[2], Zeki Dogan[3], Bulent Uzunlar[3], Seckin Satilmis[1]

[1]Department of Cardiology, Acibadem Atakent Hospital, Istanbul, Turkey
[2]Department of Cardiology, Bagcilar Public Education and Research Hospital, Turkey
[3]Department of Cardiology, Istanbul Medicine Hospital, Turkey

Abstract

Background: Serum alkaline phosphatase (ALP) level has been shown to be a prognostic factor for myocardial infarction and stroke via its promotion of vascular calcification.

Aim: To investigate for the first time the correlation between serum ALP level and coronary collateral circulation (CCC) development.

Methods: A total of 356 patients with stable angina pectoris were evaluated retrospectively. Patients were classified according to ALP level and CCC grade. Rentrop 0–1 flow was defined as impaired CCC. Serum ALP > 129 mg/dL in men and > 104 mg/dL in women was defined as elevated ALP. All groups were compared statistically according to clinical, laboratory and demographic features.

Results: Impaired CCC was observed in 53.7% of the patients. The mean ALP level was 102.8 ± 57.9 mg/dL, and elevated ALP levels were obtained in 19.4% of cases. There was a significant correlation between ALP and CCC grade, and impaired CCC was associated with relatively higher ALP values (65.2% vs. 50.9%, p = 0.03). Multivariate regression analysis also showed a significant correlation between elevated ALP level and impaired CCC (OR 1.85, with a 95% CI 1.056–3.264; p = 0.03).

Conclusions: Serum ALP is a widely avaliable unfavourable prognostic parameter in coronary heart disease. Elevated ALP levels were associated with inadequate CCC, which supports the previously reported literature concerning the negative prognostic value of ALP levels in cardiovascular settings.

Key words: alkaline phosphatase, coronary collateral circulation, stable angina

Kardiol Pol 2014; 72, 12: 1388–1393

INTRODUCTION

Coronary collateral circulation (CCC) is an adaptive response of coronary vessels and it develops as a result of angiogenesis and/or arteriogenesis. Prolonged myocardial ischaemia plays a pivotal role in the development of CCC, although not all aspects of the mechanisms underlying the development of coronary collaterals are thoroughly understood [1–4]. The variance between patients regarding coronary collateral development has prompted research on the different clinical and laboratory factors that may influence this phenomenon [5–10]. Serum alkaline phosphatase (ALP) level has been shown to be a prognostic factor in myocardial infarction and stroke due to its promotion of vascular calcification [11, 12]. A few recent studies have also shown that elevated ALP levels were associated with mortality and an unfavourable prognosis in coronary artery disease (CAD) [13, 14]. Herein, we investigated the correlation between serum ALP level and coronary collateral development for the first time. Our hypothesis was generated on the basis of vascular calcification, which may affect angiogenesis and the formation of new collateral vessels.

METHODS

Patient selection

From May 2009 to December 2012, a total of 356 patients diagnosed with stable angina pectoris with documented

Address for correspondence:
Ahmet Karabulut, MD, Acibadem Atakent Hospital Department of Cardiology, Halkali Merkez Mah. Turgut Ozal Bulvari, No: 16, 34303 Kucukcekmece, Istanbul, Turkey, tel: +90 50 53577477, e-mail: drkarabulut@yahoo.com
Received: 16.12.2013 **Accepted:** 24.04.2014 **Available as AoP:** 14.05.2014

coronary stenosis > 95% in at least one major epicardial vessel were enrolled in the study. All data was collected from a single regional cardiovascular centre with a high patient turnover. Patients were evaluated retrospectively, and clinical risk factors, medical history, laboratory results, and coronary angiography recordings were entered into a computerised database. Exclusion criteria were: acute coronary syndromes, chronic liver disease, active hepatitis, acute and chronic biliary system disease, active infection, chronic inflammatory diseases including skeletal system, decompensated heart failure, chronic renal disease, and history of cancer.

Procedure and protocol

Upon admission, patients were evaluated with anamnesis and physical examination. Standard 12-lead electrocardiogram (ECG) and transthoracic echocardiography were performed for all patients, then blood samples were taken for analysis. Patients with typical angina pectoris and/or patients with a positive exercise treadmill test were transferred to the catheter laboratory and standard coronary angiography was performed via the femoral route. The patients' angiographic data according to the catheter laboratory records was evaluated by three interventional cardiologists and a CCC grade was assigned to each patient. Serum ALP analysis was performed using the automated enzymatic analyser Cobas Integra 400 plus (Roche Diagnostic, Mannheim, Germany), with a normal range of 40–129 U/L for men and 35–104 U/L for women.

Definitions

Stable angina pectoris was defined as typical chest pain or angina equivalent symptoms triggered either by exercise or stressful conditions. A positive exercise treadmill test was defined as typical ischaemic ECG changes upon exercise. The CCC was defined according to the Rentrop classification [15]. Accordingly, grade 0 was classified as no filling; grade 1 was classified as filling of side branches via collateral channels without visualisation of the epicardial segment; grade 2 was classified as partial filling of the epicardial major coronary artery via collateral channels; and grade 3 was classified as complete filling of the epicardial major coronary artery. In patients with more than one coronary lesion, when there was more than one CCC, the CCC with the highest Rentrop grade was used. The patients were classified as having impaired CCC (group 1, Rentrop grades 0–1) or adequate CCC (group 2, Rentrop grades 2–3). Multivessel disease was defined as the presence of > 50% stenosis in two or more major epicardial arteries. Serum ALP > 129 mg/dL in men and > 104 mg/dL in women was defined as elevated ALP.

Statistical analysis and approval of the study

Statistical analyses were performed using SPSS 15.0 (SPSS Inc., Chicago, IL, USA) software. The data is presented as mean ± standard deviation with 95% confidence intervals (CI). The Student t-test was used for continuous variables between groups. Categorical variables were compared using the χ^2 test and one-way ANOVA. Correlation between serum ALP level and CCC was demonstrated with Pearson's correlation analysis and Mann-Whitney U test. In addition, univariate and multivariate binary logistic regression analysis was performed to detect independent factors affecting CCC grade.

Receiver-operating characteristic (ROC) curves for the prediction of CCC were constructed and the area under the curves (AUC) were calculated for ALP levels. Sensitivity and specificity values were estimated for predictive significance of ALP levels.

All p values were two-sided in the tests and p values less than 0.05 were considered to be statistically significant.

The study was approved by the Local Ethics Committee of the hospital.

RESULTS

The baseline demographic and clinical characteristics of the patients are summarised in Table 1. The mean age was 61.10 ± 11.5, ranging from 31 to 87, years; 279 (78.4%) patients were male. The majority of patients had an anamnesis of hypertension and diabetes mellitus. Non-developed collateral circulation (Rentrop 0) was found in only eight (2.2%) patients. Female gender and presence of diabetes mellitus were correlated with CCC grade. The mean ALP level was 102.8 ± 57.9, ranging from 41 U/L to 485 U/L, with percentile values of 70.0/86.0/110.0 mg/dL; elevated ALP levels were observed in 19.4% of cases. Elevated ALP levels were more common in women and in diabetic patients. There was a significant correlation between ALP and CCC grade, and patients with an impaired CCC grade had relatively higher ALP values (65.2% vs. 50.9%; p = 0.03, Fig. 1, Table 2). Although univariate analysis showed a significant correlation between elevated ALP level and impaired CCC (OR 1.81, 95% CI 1.048–3.129; p = 0.03), multivariate binary logistic regression analysis including all demographic, clinical and laboratory variables failed to show a significant correlation between elevated ALP level and CCC (OR 1.30, 95% CI 0.292–5.846; p = 0.72). However, multivariate analysis including significant variables in univariate analysis revealed an independent effect of ALP on impaired CCC (OR 1.85, 95% CI 1.056–3.264; p = 0.03) (Table 3). ROC curve showed a higher specificity and a relatively low sensitivity with the following result: AUC = 0.53, p = 0.30, z-statistic value = 1.20 with 95% CI 0.478–0.594. For an optimal 53 mg/dL ALP cut-off point: sensitivity was 8% and specificity was 96%.

DISCUSSION

This clinical investigation is the first report of a correlation between serum ALP level and CCC, a topic that, to the best of our knowledge, has not been studied previously. We clearly demonstrate that elevated ALP levels are associated with impaired CCC in patients with stable angina pectoris. We found that as well as interfering with normal vascular function, an

Table 1. Baseline demographic characteristics, clinical features, laboratory results and angiographic findings of the patients

Variables	(n = 356)
Age [years]	61.1 ± 11.5
Age — range [years]	31–87
Sex	
Male	78.4% (279)
Female	21.6% (77)
Presence of diabetes mellitus	43.5% (155)
Presence of hypertension	69.4% (247)
LDL cholesterol [mg/dL]	124.7 ± 44.3
Triglyceride [mg/dL]	179.4 ± 102.8
Creatinine [mg/dL]	1.01 ± 0.37
HbA1c [%]	6.8 ± 1.6
AST [U/L]	55.0 ± 119.8
ALT [U/L]	34.5 ± 63.8
Calcium [mg/dL]	9.27 ± 0.67
Phosphate [mg/dL]	3.51 ± 0.84
ALP [mg/dL]	102.8 ± 57.9
ALP — range [mg/dL]	41–485
Elevated ALP (> 129 in man, > 104 in women)	19.4% (69)
LVEF [%]	48.4 ± 10.1
Multivessel disease	71.1% (253)
Coronary collateral circulation:	
Rentrop 0	2.2% (8)
Rentrop 1	51.4% (183)
Rentrop 2	36.8% (131)
Rentrop 3	9.6% (34)
Collateral classification:	
Impaired	53.7% (191)
Adequate	46.3% (165)

LDL — low density lipoprotein; HbA1c — glycosylated haemoglobin; ALT — alanine aminotransferase; AST — aspartate aminotransferase; ALP — alkaline phosphatase; LVEF — left ventricular ejection fraction

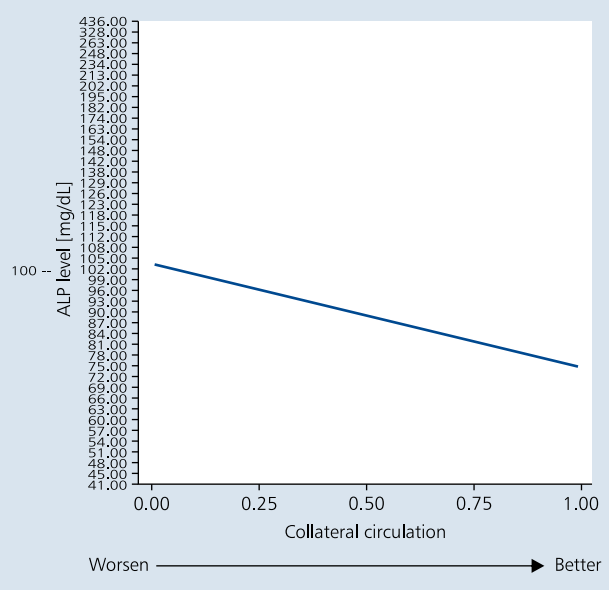

Figure 1. Correlation between serum alkaline phosphatase (ALP) levels and coronary collateral circulation

elevated ALP level may also affect neo-angiogenesis and lead to inadequate coronary collateral formation.

Serum ALP is a membrane-bound metallo enzyme that catalyses the hydrolysis of organic pyrophosphate. Phosphate was shown to be a protective factor for vascular integrity, and elevated ALP levels may promote vascular calcification via the pyrophosphate pathway [16–18]. Catalytic activity of ALP leads to inactivation of inorganic pyrophosphate, a potent inhibitor of hydroxyapatite crystal growth and potential local and circulating inhibitor of vascular calcification. The final result is chondrogenic conversion of vascular smooth muscle cells and vascular mineralisation which yield endothelial dysfunction [17]. The vasculopathic effect of ALP was initially shown in haemodialysis patients, which encouraged further investigation regarding cardiovascular prognosis [18]. Vascular calcification is one of the major contributors to atherosclerosis, yielding vascular hardening, ageing and eventually, significant vascular events [19]. Up-regulation of serum ALP levels was observed in vessels with medial calcification, which supports the mediating role of ALP [20]. After revealing the vasculopathic effect of ALP, the authors focused on the prognostic effect of ALP in several cardiovascular settings. An initial study by Regidor et al. [18] reported that serum ALP level was linked to coronary calcification in haemodialysis patients. Subsequent studies also showed the significant impact of ALP regarding cardiovascular mortality and all-cause mortality [11, 12]. Park et al. [13] later showed that elevated ALP level was a poor prognostic factor for myocardial infarction, stent thrombosis and mortality in a large patient group. Wannamethee et al. [14] supported this correlation and reported that ALP level predicted major adverse cardiovascular events and overall mortality in the elderly. We have also demonstrated the poor prognostic effect of elevated ALP level in patients with stable angina pectoris, consistent with the current literature. The basic mechanism of the unfavourable effect of ALP on CCC may be linked to endothelial dysfunction and microvascular damage, since the endothelium is the most important element in the process of collateral growth. The proliferation of coronary collaterals is dependent on physical forces that lead to the expression of adhesion molecules by the endothelium and growth factors secreted by active platelets and the vascular endothelium. These in turn lead to endothelial and smooth muscle proliferation, resulting in the development of a collateral network [21]. Elevated ALP levels may interfere with this process and thus lead to impaired collateral circulation. Although our multivariate regression analysis only included three significant

Table 2. Distribution of clinical and demographic characteristics of the patients according to coronary collateral circulation and serum alkaline phosphatase levels

Variables	Impaired collaterals	Adequate collaterals	P	Elevated ALP	Normal ALP	P
Age [years]	60.9 ± 11.8	61.3 ± 11.2	0.73	62.0 ± 12.6	60.9 ± 11.3	0.48
Sex:			**0.08**			**0.001**
Male	74.9% (143)	82.4% (136)		63.8% (44)	81.9% (235)	
Female	25.1% (48)	17.6% (29)		36.2 % (25)	18.1% (52)	
Presence of diabetes mellitus	49.7% (95)	36.4% (60)	**0.01**	34.8% (24)	45.6% (131)	0.06
Presence of hypertension	72.3% (138)	66.1% (109)	0.20	68.1% (47)	69.7% (200)	0.79
LDL cholesterol [mg/dL]	123.8 ± 42.6	125.8 ± 46.2	0.70	132.2 ± 51.5	122.8 ± 42.0	0.13
Triglyceride [mg/dL]	180.3 ± 100.9	182.8 ± 105.9	0.83	172.4 ± 90.8	183.9 ± 106.2	0.43
Creatinine [mg/dL]	1.04 ± 0.42	0.98 ± 0.30	0.15	1.00 ± 0.36	1.03 ± 0.41	0.53
HbA1c [%]	6.9 ± 1.6	6.8 ± 1.5	0.73	7.0 ± 1.9	6.8 ± 1.5	0.51
AST [U/L]	55.9 ± 134.8	53.9 ± 100.2	0.87	56.3 ± 103.3	54.6 ± 123.6	0.91
ALT [U/L]	35.8 ± 76.6	33.0 ± 45.0	0.67	30.7 ± 26.2	35.4 ± 69.9	0.58
Calcium [g/dL]	9.2 ± 0.6	9.3 ± 0.6	0.49	9.3 ± 0.6	9.2 ± 0.6	0.52
Phosphate [mg/dL]	3.4 ± 0.8	3.5 ± 0.8	0.34	3.4 ± 0.7	3.5 ± 0.8	0.71
ALP [mg/dL]	108.3 ± 64.8	96.4 ± 48.1	**0.05**	191.5 ± 77.0	81.4 ± 19.8	
Elevated ALP [%]	23.6% (45)	14.5% (24)	**0.03**			
LVEF [%]	47.4 ± 9.6	48.4 ± 10.1	0.13	47.1 ± 8.5	48.6 ± 10.4	0,35
Multivessel disease	72.3% (138)	69.7% (115)	0.46	71.0% (49)	71.1% (204)	0.99
Coronary collateral circulation:						**0.05**
Rentrop 0	4.2% (8)			1.4% (1)	02.4% (7)	
Rentrop 1	95.8% (183)			63.8% (44)	48.4% (139)	
Rentrop 2		79.4% (131)		27.5% (19)	39.0% (112)	
Rentrop 3		20.6% (34)		7.2% (5)	10.1% (29)	
Collateral classification:						**0.03**
Impaired				65.2% (45)	50.9% (146)	
Developed				34.8% (24)	49.1% (141)	
Total	53.7% (191)	46.3% (165)		19.4% (69)	80.6% (287)	

LDL — low density lipoprotein, HbA1c — glycosylsated haemoglobin; ALP — alkaline phosphatase, Elevated ALP > 129 in men, > 104 in women; LVEF — left ventricular ejection fraction; ALT — alanine aminotransferase; AST — aspartate aminotransferase; $p < 0.05$ is indicated as significant

variables, correlation analysis clearly revealed the unfavourable effect of ALP on coronary heart disease ($p = 0.03$).

Our study design and exclusion criteria make our findings reliable. We measured the tissue-non-specific ALP, which is mainly concentrated in the bone, liver and kidney, and excluded patients with chronic diseases of the liver, bone and kidney, which could have interfered with our results. We also excluded patients with inflammatory diseases in which ALP levels tend to increase. Therefore, we propose that serum ALP level is an important prognostic factor in the formation of coronary collaterals and can be considered in the clinical implications regarding CAD. Moreover, an elevated ALP level may show a stronger association with either the presence (Rentrop 1-2-3) or absence (Rentrop 0) of CCC. Due to our strict inclusion criteria (>95 % stenosis in at least one major epicardial vessel), there were only eight patients with Rentrop 0 CCC. Further studies with larger samples may be required to show a significant correlation, as in our sample elevated ALP levels were only observed in 69 patients.

Limitations of the study

The retrospective study design and relatively small sample size (only eight patients in the Rentrop 0 group) are the major limitations of the study. We included patients with coronary stenosis of at least 95% in one major epicardial coronary vessel in order to obtain more patients with collateral circulation. However, this strict inclusion criterion led to a small patient sample with non-developed CCC (Rentrop 0). We recommend that further investigations regarding CCC should have a standard inclusion criterion of > 70% coronary stenosis in at least one major epicardial coronary vessel, which may solve this prob-

Table 3. Univariate and multivariate logistic regression analysis to reveal the determinants of coronary collateral circulation (CCC)

Dependant variable: CCC	Univariate analysis		Multivariate analysis	
	95% CI	P	95% CI	P
Age [years]	0.985–1.021	0.73		
Sex (male/female)	0.379–1.066	0.08	0.437–1.272	0.28
Presence of diabetes mellitus	1.132–2.650	0.01	1.153–2.750	0.009
Presence of hypertension	0.851–2.102	0.20		
LDL cholesterol	0.996–1.006	0.70		
Triglyceride	0.998–1.002	0.83		
Creatinine	0.352–1.180	0.15		
HbA1c	0.812–1.159	0.73		
Multivessel disease	0.715–1.791	0.59		
Elevated ALP	1.048–3.129	0.03	1.056–3.264	0.03
LVEF [%]	0.994–1.44	0.13		
Calcium	0.816–1.522	0.49		
Phosphate	0.844–1.620	0.34		

CI — confidence interval; LDL — low density lipoprotein; HbA1c — glycosylated haemoglobin; ALP — alkaline phosphatase; LVEF — left ventricular ejection fraction; $p < 0.10$ is indicated as significant in univariate analysis; $p < 0.05$ is indicated as significant in multivariate analysis

lem. Moreover, we assessed CCC with Rentrop classification and we did not perform other methods such as collateral flow grade, collateral frame count or recipient filling grade which may yield different results. In addition, other inflammatory and neurohormonal mediators including C-reactive protein, B-type natriuretic peptide, erythropoietin, nitric oxide, other proinflammatory cytokines and markers of oxidative stress have the potential to affect CCC development, but they were not measured herein. We also did not measure the parathyroid hormone and vitamin D levels, which may affect ALP levels. We individually measured the tissue-non-specific ALP level. Measurement of bone-specific ALP may yield a more accurate result. Serum ALP was defined as a marker of vascular calcification. However, we did not check the coronary calcium score or perform specific techniques such as intravascular ultrasound to determine the extent and severity of the CAD. We only evaluated the collateral circulation. On the other hand, numerous studies have already shown that CCC is a strong prognostic factor in the progression of CAD disease, and elevated ALP level may be accepted as an unfavourable factor in the progression of CAD.

CONCLUSIONS

Serum ALP is a widely avaliable vasculopathic prognostic parameter in coronary heart disease. Our results support the previously proposed unfavourable prognostic effect of ALP in cardiovascular disease. We showed that elevated ALP levels were associated with inadequate CCC, which supports the reported literature concerning the negative prognostic value of ALP levels in cardiovascular settings.

Conflict of interest: *none declared*

References

1. Charney R, Cohen M. The role of the coronary collateral circulation in limiting myocardial ischemia and infarct size. Am Heart J, 1993; 126: 937–945.
2. Antoniucci D, Valenti R, Moschi G et al. Relation between preintervention angiographic evidence of coronary collateral circulation and clinical and angiographic outcomes after primary angioplasty or stenting for acute myocardial infarction. Am J Cardiol, 2002; 89: 121–125.
3. Chilian WM, Mass HJ, Williams S et al. Microvascular occlusions promote coronary collateral growth. Am J Physiol, 1990; 258: H1103–H11011.
4. Ishihara M, Inoue I, Kawagoe T et al. Comparison of the cardioprotective effect of prodromal angina pectoris and collateral circulation in patients with a first anterior wall acute myocardial infarction. Am J Cardiol, 2005; 95: 622–625.
5. Sabri MN, DiSciascio G, Cowley MJ et al. Coronary collateral recruitment: functional significance and relation to rate of vessel closure. Am Heart J, 1991; 121: 876–880.
6. Sasayama S, Fujita M. Recent insights into coronary collateral circulation. Circulation, 1992; 85: 1197–1204.
7. Tanboga IH, Topcu S, Nacar T et al. Relation of coronary collateral circulation with red cell distribution width in patients with non-ST elevation myocardial ınfarction. Clin Appl Thromb Hemost, 2013; Jan 15, doi:10.1177/1076029612470490.
8. Ege MR, Acıkgoz S, Zorlu A et al. Mean platelet volume: an important predictor of coronary collateral development. Platelets, 2013; 24: 200–204.
9. Sarli B, Baktir AO, Saglam H et al. The relation of serum gamma-glutamyl transferase levels and coronary collateral circulation in patients with chronic coronary total occlusion. Coron Artery Dis, 2013; 24: 298–302.
10. Kasapkara HA, Topsakal R, Yarlioglues M et al. Effects of serum uric acid levels on coronary collateral circulation in patients with non-ST elevation acute coronary syndrome. Coron Artery Dis, 2012; 23: 421–425.
11. Ryu WS, Lee SH, Kim CK et al. Increased serum alkaline phosphatase as a predictor of long-term mortality after stroke. Neurology, 2010; 75: 1995–2002.

12. Tonelli M, Curhan G, Pfeffer M et al. Relation between alkaline phosphatase, serum phosphate, and allcause or cardiovascular mortality. Circulation, 2009; 120: 1784–1792.
13. Park JB, Kang DY, Yang HM et al. Serum alkaline phosphatase is a predictor of mortality, myocardial infarction, or stent thrombosis after implantation of coronary drug-eluting stent. Eur Heart J, 2013; 34: 920–931.
14. Wannamethee SG, Sattar N, Papcosta O et al. Alkaline phosphatase, serum phosphate, and incident cardiovascular disease and total mortality in older men. Arterioscler Thromb Vasc Biol, 2013; 33: 1070–1076.
15. Rentrop KP, Thornton JC, Feit F, Van Buskirk M. Determinants and protective potential of coronary arterial collaterals as assessed by an angioplasty model. Am J Cardiol, 1988; 61: 677–684.
16. Johnson RC, Leopold JA, Loscalzo J. Vascular calcification: pathobiological mechanisms and clinical implications. Circ Res, 2006; 99: 1044–1059.
17. Schoppet M, Shanahan CM. Role for alkaline phosphatase as an inducer of vascular calcification in renal failure? Kidney Int, 2008; 73: 989–991.
18. Regidor DL, Kovesdy CP, Mehrotra R et al. Serum alkaline phosphatase predicts mortality among maintenance hemodialysis patients. J Am Soc Nephrol, 2008; 19: 2193–2203.
19. Detrano R, Guerci AD, Carr JJ et al. Coronary calcium as a predictor of coronary events in four racial or ethnic groups. N Engl J Med, 2008; 358: 1336–1345.
20. Shanahan CM, Cary NR, Salisbury JR et al. Medial localization of mineralization-regulating proteins in association with Monckeberg's sclerosis: evidence for smooth muscle cell-mediated vascular calcification. Circulation, 1999; 100: 2168–2176.
21. Duran M, Ornek E, Murat SN et al. High Levels of Serum uric acid impair development of coronary collaterals in patients with acute coronary syndrome. Angiology, 2012; 63: 472–475.

Wpływ aktywności fosfatazy zasadowej w surowicy na wieńcowe krążenie oboczne

Ahmet Karabulut[1], Irfan Sahin[2], Ilhan Ilker Avci[2], Ertugrul Okuyan[2], Zeki Dogan[3], Bulent Uzunlar[3], Seckin Satilmis[1]

[1]Department of Cardiology, Acibadem Atakent Hospital, Istanbul, Turcja
[2]Department of Cardiology, Bagcilar Public Education and Research Hospital, Turcja
[3]Department of Cardiology, Istanbul Medicine Hospital, Turcja

Streszczenie

Wstęp: Jak wskazują badania, aktywność fosfatazy zasadowej (ALP) w surowicy jest czynnikiem prognostycznym zawału serca i udaru mózgu, ponieważ enzym ten wpływa na zwiększenie uwapnienia naczyń krwionośnych.

Cel: Celem pracy było zbadanie po raz pierwszy korelacji między aktywnością ALP w surowicy a rozwojem wieńcowego krążenia obocznego (CCC).

Metody: W badaniu retrospektywnie oceniono 356 chorych ze stabilną dławicą piersiową. Osoby te podzielono na grupy w zależności od aktywności ALP i stopnia CCC. Upośledzenie CCC definiowano jako przepływ stopnia 0–1 wg klasyfikacji Rentropa. Aktywność ALP w surowicy wynoszącą > 129 mg/dl u mężczyzn i > 104 mg/dl u kobiet uznawano za podwyższenie aktywności ALP. Przeprowadzono porównania statystyczne parametrów klinicznych, laboratoryjnych i demograficznych między wszystkimi grupami.

Wyniki: Niedostateczne CCC zaobserwowano u 53,7% chorych. Średnia aktywność ALP wynosiła 102,8 ± 57,9 mg/dl, a podwyższoną aktywność ALP wykryto u 19,4% pacjentów. Wykazano istotną korelację między aktywnością ALP a stopniem CCC; słabsze CCC wiązało się z proporcjonalnie wyższymi aktywnościami ALP (65,2% *vs*. 50,9%; p = 0,03). Również wieloczynnikowa analiza regresji wykazała istotną zależność między wyższymi aktywnościami ALP a słabszym CCC (OR 1,85; 95% CI 1,056–3,264; p = 0,03).

Wnioski: Aktywność ALP w surowicy jest powszechnie dostępnym parametrem rokowniczym w chorobie wieńcowej. Podwyższone aktywności ALP wiązały się z niedostatecznym CCC, co potwierdza wyniki wcześniejszych doniesień dotyczących znaczenia tego parametru jako wskaźnika niekorzystnego rokowania u pacjentów z chorobami sercowo-naczyniowymi.

Słowa kluczowe: fosfataza zasadowa, wieńcowe krążenie oboczne, stabilna dławica

Kardiol Pol 2014; 72, 12: 1388–1393

Adres do korespondencji:
Ahmet Karabulut, MD, Acibadem Atakent Hospital Department of Cardiology, Halkali Merkez Mah. Turgut Ozal Bulvari, No: 16, 34303 Kucukcekmece, Istanbul, Turkey, tel: +90 50 53577477, e-mail: drkarabulut@yahoo.com
Praca wpłynęła: 16.12.2013 r. **Zaakceptowana do druku:** 24.04.2014r. **Data publikacji AoP:** 14.05.2014 r.