Association between paraoxonase activity and late saphenous vein graft occlusion in patients with coronary artery bypass grafting

Goksel Cagirci¹, Serkan Cay², Ozlem Karakurt¹, Nuray Yazihan³, Cengiz Aydin⁴, Sadik Acikel¹, Mehmet Dogan¹, Harun Kilic¹, Serkan Topaloglu², Dursun Aras², Ramazan Akdemir¹

¹ Department of Cardiology, Ministry of Health, Dışkapı Yıldırım Beyazıt Research and Educational Hospital, Ankara, Turkey

² Department of Cardiology, Yuksek Ihtisas Heart-Education and Research Hospital, Ankara, Turkey

³ Pathophysiology Department, and Molecular Biology Research and Development Unit, Faculty of Medicine, Ankara University, Ankara, Turkey

⁴ Biochemistry Department, Dogubeyazit Government Hospital, Agri, Turkey

Abstract

Background: Coronary vein graft disease is an important contributor to the morbidity after coronary artery bypass grafting (CABG). Late occlusion of the graft is a serious complication that limits the use of the saphenous vein as a coronary bypass conduit. It is frequently encountered in old, degenerated vein grafts with advanced atherosclerotic plaque formation. Paraoxonase-1 (PON-1) is an HDL-bound enzyme which has anti-atherogenic properties and protects LDL cholesterol from oxidative modification.

Aim: To examine the association between PON-1 activity and late saphenous vein graft occlusion.

Methods: Thirty-eight patients who had at least one occluded saphenous vein graft (group 1; 12 females, 26 males) and 41 patients who had a patent saphenous vein graft (group 2; 7 females, 34 males) were enrolled in this study. Paraoxonase activity was measured spectrophotometrically.

Results: The mean PON-1 activity in group 1 was significantly lower than in group 2 ($74.1 \pm 52.1 \text{ vs.} 114.4 \pm 90.9 \text{ U/l}, \text{ p} = 0.02$). The mean platelet volume was significantly higher in group 1 than group 2 ($8.8 \pm 1.6 \text{ vs.} 8.2 \pm 1.1 \text{ fl}, \text{ p} = 0.04$). Multiple logistic regression analysis showed that only PON-1 activity (beta = 0.011, p = 0.042) was an independent predictor of late occlusion of a saphenous vein graft.

Conclusions: Our results show that PON-1 activity is lower in patients with late saphenous vein graft occlusion. Reduced PON-1 activity may lead to acceleration of saphenous vein graft occlusion.

Key words: paraoxonase, saphenous vein graft

Kardiol Pol 2009; 67: 1063-1068

Introduction

Coronary artery disease (CAD) still remains the major cause of mortality in developed countries. Essential treatment modalities include medical therapy, percutaneous transluminal coronary interventions and coronary artery bypass grafting (CABG). Surgical treatment still remains a valuable option despite the improvements in percutaneous transluminal coronary interventions, especially in threevessel disease and left main CAD.

Although arterial graft conduits are intended to be used in bypass surgery most commonly, saphenous vein grafts are still frequently used. At the end of the first year, 15% of these saphenous grafts restenose, and by 10 years after surgery, approximately 50% of these grafts are occluded [1]. Saphenous vein graft occlusion is the main reason for the majority of reinterventions [2].

Studies have shown that typical atherosclerotic changes have not been observed up to 1 year and very rarely up to 2 years in vein grafts. After 2 years, graft atherosclerosis becomes a major source of mortality and morbidity. Atherosclerosis follows similar pathways in vein grafts as in native artery disease.

An increased level of high density lipoproteins (HDL) has been reported to be associated with decreased risk for

Address for correspondence:

Serkan Cay MD, Yuksek Ihtisas Heart-Education and Research Hospital, Yasamkent Mah. 3222. Cad.2. Blok (Yakut) No: 37 D: 27 Cayyolu, Ankara, Turkey, tel.: +90 312 217 38 62, e-mail: cayserkan@yahoo.com

Received: 14 March 2009. Accepted: 24 June 2009.

There are three known PON genes: *PON1, PON2* and *PON3*. The PON-1 is synthesised in the liver and transported to the HDL in the plasma [3]. It is a protein of 354 amino acids with a molecular weight of 43 kDa [4]. Studies have shown that HDL-associated PON-1 inhibits lipid peroxidation or degrades biologically active oxidised lipids in low density lipoprotein (LDL) [5]. The PON-1 is recruited with breakdown of lipid peroxides before they can accumulate on LDL [6]. The PON-1 over-expression protects mouse from atherosclerosis [7]. Its activity decreases in many diseases such as slow coronary flow [8], cardiac syndrome X [9], CAD [10, 11], diabetes mellitus, and myocardial infarction [12].

No studies have previously evaluated the effects of PON activity on late saphenous vein graft occlusion. Therefore, the goal of this study was to assess the association between PON activity and late saphenous vein graft occlusion.

Methods

Study population

The study was performed at the Ministry of Health, Diskapi Yildirim Beyazit Research and Educational Hospital, Cardiology Clinic and Yuksek Intisas Education and Research Hospital, Cardiology Clinic in Turkey from June 2007 to May 2008.

The study population included 79 consecutive patients who underwent elective coronary angiography because of recurrence of postoperative symptoms. Two groups of patients were included in the study: 38 patients (12 females, 26 males, mean age 61.6 ± 8.9 years) who had at least one occluded saphenous vein graft on their late control coronary angiography after CABG, and 41 patients (7 females, 34 males, mean age of 61.0 ± 9.7 years) who had patent vein grafts on their last control coronary angiography.

Subjects who had acute or chronic inflammatory diseases, myeloproliferative diseases, malignancies, renal, hepatic and thyroid diseases, immunological diseases, haematocrit < 0.30 or > 0.52, platelet count < 100 000/mm³, patients with acute coronary syndromes during the last 48 h before hospitalisation, and those with severe valvular heart diseases were excluded from the study. Patients who underwent CABG within one year preceding the study were also excluded.

Transthoracic echocardiography was performed using the Vingmed Vivid 3 echocardiograph and a 2.5 MHz transducer to detect underlying structural heart disease. Written consent was obtained from all patients and our local ethical committee approved the study.

Coronary angiography

Coronary angiography was performed using standard angiographic techniques. All cineangiograms were

reviewed by two interventional cardiologists who were blinded to the study protocol. The bypass grafts were examined in multiple projections and the degree of stenosis determined in the projection that showed the most severe narrowing. The stumps of occluded (100% stenosis) grafts were selectively injected or visualised on aortography in the appropriate projection.

Blood sample collection

Blood samples were obtained following an overnight fasting period before angiography. The serum was separated from the cells by centrifugation at 3000 r min⁻¹ for 10 min and stored at -78° C until measurement of PON activity.

Measurement of PON activity

The serum PON activity was measured using the synthetic substrate paraoxon (diethyl-p-nitrophenol, PS610, SUPELCO, USA). The rate of paraoxon hydrolysis was measured by monitoring the increase of absorbency at 415 nm at 37°C. The amount of generated p-nitrophenol was calculated from the molar absorptivity coefficient at pH 8, which was 16 900 M⁻¹cm⁻¹ [13]. Paraoxonase activity was expressed as U/l.

Measurement of lipid parameters

Fasting plasma glucose, total cholesterol, HDL cholesterol, and triglyceride levels were measured enzymatically by the autoanalyzer (Hitachi 911, Japan). The LDL cholesterol levels were determined with the Friedewald formula.

Other laboratory data

Blood samples were collected in an ethylene diamine tetra acetic acid (EDTA) tube for complete blood count, including platelet count and haemoglobin. Mean platelet volume was measured using a Coulter S+ resistive particle counting system.

Statistical analysis

Results are reported as mean ± standard deviation (SD) and percentages. Student's t-test was used to compare normally distributed continuous variables and the Mann--Whitney U test was used for variables without normal distribution. Categorical variables were compared by using the χ^2 test. In order to determine independent predictors of late saphenous vein graft occlusion, multiple logistic regression analysis was performed by including parameters which were significantly different between group 1 and group 2. A p value of < 0.05 was considered statistically significant. The SPSS-15.0 for Windows statistical software package was used.

Results

The demographic and clinical characteristics of the groups are presented in Table I. There were no significant

	Patients with graft occlusion (n = 38)	Patients with patent grafts (n = 41)	р	
Age [years]	61.6 ± 8.9	61.0 ± 9.7	0.75	
Male/female	26/12	34/7	0.13	
BMI [kg/m ²]	28.9 ± 4.0	28.1 ± 3.4	0.31	
Hypertension [%]	17 (44.7)	20 (48.8)	0.72	
Diabetes mellitus [%]	13 (34.2)	12 (29.3)	0.64	
Smoking [%]	11 (28.9)	9 (22.0)	0.48	
Family history of CAD [%]	7 (18.4)	9 (22.0)	0.70	
Ejection fraction [%]	49.3 ± 10.0	48.0 ± 11.4	0.59	
Aspirin, n (%)	32 (84.2)	34 (82.9)	0.88	
Beta-blockers, n (%)	29 (76.3)	34 (82.9)	0.46	
ACE inhibitors, n (%)	17 (44.7)	14 (34.1)	0.34	
ARB, n (%)	4 (10.5)	4 (9.8)	0.91	
Statin, n (%)	13 (34.2)	24 (58.5)	0.03	
Nitrates, n (%)	5 (13.2)	9 (22)	0.30	
Time from surgery [months]	71.6 ± 45.5	67.5 ± 56.3	0.72	
SBP [mmHg]	132.6 ± 10.4	133.8 ± 11.1	0.64	
DBP [mmHg]	83.9 ± 7.4	85.2 ± 9.6	0.52	

Table I. Demographic and clinical characteristics of the study patients

Abbreviations: BMI – body mass index, ACE – angiotensin-converting enzyme, ARB – angiotensin receptor blocker, SBP – systolic blood pressure, DBP – diastolic blood pressure

	Patients with graft occlusion (n = 38)	Patients with patent grafts (n = 41)	р
Haemoglobin [g/dl]	14.2 ± 2.1	13.9 ± 1.9	0.55
Platelets [x 10 ³ /mm ³]	253.7 ± 75.3	249.9 ± 85.6	0.83
MPV [fl]	8.8 ± 1.6	8.2 ± 1.1	0.04
Fibrinogen [mg/dl]	4.0 ± 1.1	3.8 ± 1.0	0.28
Total cholesterol [mg/dl]	189.4 ± 44.3	174.1 ± 36.1	0.09
LDL cholesterol [mg/dl]	111.2 ± 37.6	97.6 ± 32.1	0.09
HDL cholesterol [mg/dl]	38.8 ± 9.6	38.6 ± 11.9	0.94
Triglyceride [mg/dl]	189.9 ± 111.6	170.8 ± 94.2	0.41
Fasting glucose [mg/dl]	96.3 ± 12.8	92.1 ± 11.8	0.14
PON-1 activity [U/l]	74.1 ± 52.1	114.4 ± 90.9	0.02

Table II. Comparison of laboratory parameters between patients with occluded and patent grafts

Abbreviations: MPV – mean platelet volume, LDL – low-density lipoprotein, HDL – high-density lipoprotein, PON – paraoxonase

differences in age, gender, body mass index (BMI) and presence of typical atherosclerotic risk factors between the groups. The mean period from CABG to last coronary angiogram was similar in both groups (71.6 \pm 45.5 vs. 67.5 \pm 56.3 months, NS). The two groups did not differ with regard to baseline medication taken before entry into the study including aspirin, beta-blockers, angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, nitrates, and lipid-lowering drugs. However, statin use was significantly lower in patients with occluded grafts [13 (34.2%) vs. 24 (58.5%), p = 0.03].

The laboratory parameters of the groups are summarised in Table II. The values of total cholesterol, LDL cholesterol, HDL cholesterol, triglyceride, haemoglobin, fasting blood glucose, and fibrinogen levels were not different between the groups.

The mean PON-1 activity in patients with graft occlusion was significantly lower than in patients with patent grafts

Table III. Multivariate analysis of para	meters					
predicting saphenous vein graft occlusion						

	Beta	S.E.	р
PON-1 activity [U/l]	0.011	0.005	0.042
MPV [fl]	-0.320	0.203	0.115
Statin use	-0.799	0.535	0.136

Abbreviations: PON – paraoxonase, MPV – mean platelet volume

 $(74.1 \pm 52.1 \text{ vs. } 114.4 \pm 90.9 \text{ U/l, } p = 0.02)$. The mean platelet volume was significantly higher in patients with occluded grafts (8.8 ± 1.6 vs. 8.2 ± 1.1 fl, p = 0.04).

Multiple logistic regression analysis including parameters with significant differences between the two groups showed that only PON-1 activity (β = 0.011, p = 0.042) was an independent predictor of late occlusion of a saphenous vein graft (Table III).

Discussion

To our knowledge, this is the first study to evaluate the effects of PON-1 activity on late saphenous vein graft occlusion. In our study, we found that mean PON-1 activity was significantly lower in patients with occluded saphenous vein graft than in the patent saphenous vein graft group. Also, mean platelet volume was significantly higher in the occluded saphenous vein group than in the patent saphenous vein group. In multiple logistic regression analysis, only PON-1 activity was an independent predictor of late occlusion of a saphenous vein graft.

Coronary artery bypass grafting is one of the commonly used revascularisation techniques and it improves the patient's quality of life unless graft occlusion occurs. Graft occlusion remains the major problem of this procedure. Three consecutive phases of venous graft disease have been described: a phase of thrombotic occlusion (especially during the first month), an intermediate phase of neointimal hyperplasia (up to 1 year), and a phase of atherosclerotic disease (after 1 year). Late vein graft occlusion is considered to be a part of the atherosclerotic process [14-16]. Therefore, this study was performed in patients with a long-term follow-up of a saphenous vein graft. The mean time from surgery was 71.6 \pm 45.5 months in the occluded saphenous vein group and 67.5 \pm 56.3 months in the patent saphenous vein group.

Increased levels of HDL have been reported to be associated with decreased risk for CAD. This protective effect of HDL is largely related to the PON-1 enzyme which is located on the HDL molecule [17]. Several studies have shown that HDL protects LDL from oxidative modification, which is thought to be the principle step of atherosclerosis. The PON-1 can hydrolyse lipid peroxides in oxidised lipoproteins [18]. It has been recently observed that PON-1 hydrolyses and reduces lipid peroxides not only in LDL but also in human coronary and carotid lesions (Michael Aviram, unpublished). Several studies have demonstrated that there is a significant relationship between PON-1 activity and concentration and severity of CAD [19-21]. The reduced PON-1 activity in the occluded saphenous vein graft group revealed in our study supports these studies.

Previous studies have shown that changes of PON-1 activity may occur independently of changes in the HDL cholesterol [22, 23]. Navab et al. demonstrated in patients with CAD that oxidation of LDL was not protected by HDL due to low serum PON activity despite relatively normal HDL concentrations [24]. Similarly, in our study, there was no difference in HDL cholesterol between the groups.

The PON activity decreases in many diseases such as slow coronary flow [8], cardiac syndrome X [9], CAD [10, 11], diabetes mellitus, myocardial infarction [12], and acute phase response [25]. The PON-1 activity is determined genetically; however, several factors can influence its activity. Van Der Gaag et al. have reported that PON activity was increased in subjects with daily moderate alcohol intake [26]. James et al. have shown that smoking decreased serum PON activity [27]. Degraded cooking oil has been reported to lower serum PON-1 levels in humans [28]. In addition, previous studies demonstrated that PON-1 activity was increased with statin use [29-31]. In the present study, statin use was lower in the occluded saphenous vein group than in the patent saphenous vein group. This finding might explain the reduced PON-1 activity of the occluded saphenous vein group. However, this increased statin use did not cause a significant difference in the HDL, LDL and total cholesterol values between the two groups. In addition, in the multiple logistic regression analysis, only PON-1 activity was an independent predictor of late occlusion of a saphenous vein graft.

Study limitations

The most important limitation of our study is the small number of patients. In addition, the PON-1 genotype was not determined in the study population. However, serum PON-1 concentration and activity have been shown to be better predictors of the risk for cardiovascular diseases than PON-1 genotype [32, 33]. In addition, the levels of ox-LDL, homocysteine as well as hs-CRP or IL-6 have not been measured in our study. Therefore, it cannot be ruled out that the basic pathomechanisms of late saphenous vein graft occlusion include the activation of monocytes/macrophages caused by high homocysteine level and the associated oxidative stress. Lastly, although multivariate analysis showed that PON-1 activity was an independent predictor of graft occlusion, this association was of almost borderline significance (p = 0.042) and differences in statin use were striking. Thus, some uncertainty concerning the independent value of PON-1 exists.

Conclusion

Our results showed that PON-1 activity is lower in patients with late saphenous vein graft occlusion than in

those with patent grafts. Reduced PON-1 activity may lead to the acceleration of saphenous vein graft occlusion. These patients require more aggressive medical treatment in order to prevent the atherosclerotic process.

References

- 1. Bourassa MG. Long term vein graft patency. *Curr Opin Cardiol* 1994; 9: 685.
- Holmann WL, McGiffin DC, Kirklin JK. Role of bypass surgery. In: Fuster V, Topol EJ, Nabel EG (eds.). Atherothrombosis and Coronary Artery Disease. 2nd ed. *Lippincott Williams & Wilkins*, 2005.
- Bergmeier C, Siekmeier R, Gross W. Distribution spectrum of paraoxonase activity in HDL fractions. *Clin Chem* 2004; 50: 2309-15.
- 4. Primo-Parma SL, Sorenson RC, Teiber J, et al. The human serum paraoxonase/arylesterase gene (PON1) is one member of a multigene family. *Genomics* 1996; 33: 498-509.
- 5. Barter PJ, Nicholls S, Rye A, et al. Antiinflammatory properties of HDL. *Circ Res* 2004; 95: 764-72.
- 6. Mackness MI, Arrol S, Durrington PN. Paraoxonase prevents accumulation of lipoperoxides in low density lipoprotein. *FEBS Lett* 1991; 286: 152-4.
- 7. Tward A, Xia YR, Wang P, et al. Decreased atherosclerotic lesion formation in human serum paraoxonase transgenic mice. *Circulation* 2002; 106: 484-90.
- Yildiz A, Gur M, Yilmaz R, et al. Association of paraoxonase activity and coronary blood flow. *Atherosclerosis* 2008; 197: 257-63.
- 9. Gur M, Yildiz A, Demirbag R, et al. Paraoxonase and arylesterase activities in patients with cardiac syndrome X, and their relationship with oxidative stress markers. *Coron Artery Dis* 2007; 18: 89-95.
- Azarsiz E, Kayikcioglu M, Payzin S, et al. PON1 activities and oxidative markers of LDL in patients with angiographically proven coronary artery disease. *Int J Cardiol* 2003; 91: 43-51.
- 11. Gur M, Aslan M, Yildiz A, et al. Paraoxonase and arylesterase activities in coronary artery disease. *Eur J Clin Invest* 2006; 36: 779-87.
- Ayub A, Mackness MI, Arrol S, et al. Serum paraoxonase after myocardial infarction. Arterioscler Thromb Vasc Biol 1999; 19: 330-5.
- 13. Eckerson HW, Wyte CM, La Du BN. The human serum paraoxonase/arylesterase polymorphism. *Am J Hum Genet* 1983; 35: 1126-38.
- 14. Fuster V, Chesebro JH. Role of platelets and platelet inhibitors in aortocoronary artery vein-graft disease. *Circulation* 1986; 73: 227-32.
- 15. Walts AE, Fishbein MC, Matloff JM. Thrombosed, ruptured atheromatous plaques in saphenous vein coronary artery bypass grafts: ten years' experience. *Am Heart J* 1987; 114: 718-23.
- Yilmaz MB, Balbay Y, Caldir V, et al. Late saphenous vein graft occlusion in patients with coronary bypass: possible role of aspirin resistance. *Thromb Res* 2005; 115: 25-9.
- Shih DM, Gu L, Hama S, et al. Genetic-dietary regulation of serum paraoxonase expression and its role in atherogenesis in a mouse model. J Clin Invest 1996; 97: 1630-9.
- Aviram M, Billecke S, Sorenson R, et al. Paraoxonase active site required for protection against LDL oxidation involves its free

sulfhydryl group and is different from that required for its arylesterase/paraoxonase activities. *Arterioscler Thromb Vasc Biol* 1998; 18: 1617-24.

- 19. Gur M, Aslan M, Yildiz A, et al. Paraoxonase and arylesterase activities in coronary artery disease. *Eur J Clin Invest* 2006; 36: 779-87.
- 20. Tartan Z, Orhan G, Kasikcioglu H, et al. The role of paraoxonase (PON) enzyme in the extent and severity of the coronary artery disease in type-2 diabetic patients. *Heart Vessels* 2007; 22: 158-64.
- 21. Granér M, James RW, Kahri J, et al. Association o paraoxonase-1 activity and concentration with angiographic severity and extent of coronary artery disease. *J Am Coll Cardiol* 2006; 47: 2429-35.
- 22. Mackness MI, Mackness B, Durrington PN, et al. Paraoxonase: biochemistry, genetics and relationship to plasma lipoproteins. *Curr Opin Lipidol* 1996; 7: 69-76.
- 23. Mackness B, Durrington PN, Mackness MI. Human serum paraoxonase. *Gen Pharmac* 1998; 31: 329-36.
- 24. Navab M, Hama SY, Van Lanten BJ, et al. Mildly oxidised LDL induces an increased apolipoprotein/paraoxonase ratio. *J Clin Invest* 1997; 99: 2005-19.
- 25. Feingold KR, Memon RA, Moser AH, et al. Paraoxonase activity in the serum and hepatic mRNA levels decrease during the acute phase response. *Atherosclerosis* 1998; 139: 307-15.
- 26. Van Der Gaag MS, van Tol A, Scheek LM, et al. Daily moderate alcohol consumption increases serum paraoxonase activity; a diet controlled, randomized intervention study in middle-aged men. *Atherosclerosis* 1999; 147: 405-10.
- 27. James RW, Leviev I, Righetti A. Smoking is associated with reduced serum paraoxonase activity and concentration in coronary artery disease patients. *Circulation* 2000; 101: 2252-7.
- Sutherland WHF, Walker RJ, de Jong SA, et al. Reduced postprandial serum paraoxonase activity after a meal rich in used cooking fat. *Arterioscler Thromb Vasc Biol* 1999; 19: 1340-7.
- 29. Kural BV, Orem C, Uydu HA. The effect of lipid-lowering therapy on paraoxonase activities and their relationships with the oxidant-antioxidant system in patients with dyslipidemia. *Coron Artery Dis* 2004; 15: 277-83.
- 30. Tomàs M, Sentý M, Garcýa-Faria F, et al. Effect of simvastatin therapy on paraoxonase activity and related lipoproteins in familial hypercholesterolemic patients. *Arterioscler Thromb Vasc Biol* 2000; 20: 2113-19.
- Fuhrman B, Koren L, Volkova N, et al. Atorvastatin therapy in hypercholesterolemic patients suppresses cellular uptake of oxidized-LDL by differentiating monocytes. *Atherosclerosis* 2002; 164: 179-85.
- 32. Mackness M, Durrington P, Mackness B. Paraoxonase 1 activity, concentration and genotype in cardiovascular disease. *Curr Opin Lipidol* 2004; 15: 399-404.
- 33. Mackness M, Mackness B. Paraoxonase 1 and atherosclerosis: is the gene or the protein more important? *Free Radical Biol Med* 2004; 37: 1317-23.

Związek pomiędzy aktywnością paraoksonazy a późną niedrożnością pomostów żylnych u chorych poddawanych chirurgicznej rewaskularyzacji wieńcowej

Goksel Cagirci¹, Serkan Cay², Ozlem Karakurt¹, Nuray Yazihan³, Cengiz Aydin⁴, Sadik Acikel¹, Mehmet Dogan¹, Harun Kilic¹, Serkan Topaloglu², Dursun Aras², Ramazan Akdemir¹

¹ Klinika Kardiologii, Szpital Dışkapı Yıldırım Beyazıt, Ankara, Turcja

² Klinika Kardiologii, Szpital Yuksek Ihtisas, Ankara, Turcja

³ Klinika Patofizjologii, Biologii Molekularnej i Rozwoju, Uniwersytet w Ankarze, Turcja

⁴ Klinika Biochemii, Szpital Rządowy Dogu Beyazit, Agri, Turcja

Streszczenie

Wstęp: Zwężenie żylnego pomostu aortalno-wieńcowego jest istotnym powikłaniem chirurgicznej rewaskularyzacji wieńcowej (CABG). Późne zamknięcie pomostu jest poważnym powikłaniem, ograniczającym szerokie stosowanie żylnych pomostów u wszystkich chorych. Najczęściej dochodzi do niego w żylnych pomostach wszczepionych wiele lat temu, w których rozwijają się blaszki miażdżycowe. Paraoksonaza 1 (PON-1) jest enzymem wiążącym HDL, który działa przeciwmiażdżycowo, ochraniając LDL-cholesterol przed utlenianiem.

Cel: Zbadanie związku pomiędzy aktywnością PON-1 a późnym zamknięciem pomostu żylnego.

Metody: Do badania włączono 38 chorych z co najmniej jednym zamkniętym pomostem żylnym (grupa 1 – 12 kobiet, 26 mężczyzn)
 i 41 chorych z drożnym pomostem żylnym (grupa 2 – 7 kobiet, 34 mężczyzn). Aktywność PON-1 mierzono metodą spektrofotometryczną.
 Wyniki: Średnia aktywność PON-1 w grupie 1 była istotnie niższa niż w grupie 2 (74,1 ± 52,1 vs 114,4 ± 90,9 U/l, p = 0,02), natomiast

Wyniki: Srednia aktywność PON-1 w grupie 1 była istotnie nizsza niz w grupie 2 (/4,1 ± 52,1 vs 114,4 ± 90,9 U/l, p = 0,02), natomiast średnia objętość płytek krwi była istotnie wyższa w grupie 1 niż 2 (8,8 ± 1,6 vs 8,2 ± 1,1 fl, p = 0,04). Wieloczynnikowa analiza regresji wykazała, że aktywność PON-1 była jedynym niezależnym czynnikiem związanym z późnym zamknięciem żylnego pomostu aortalnowieńcowego (β = 0,011, p = 0,042).

Wnioski: Aktywność PON-1 jest mniejsza u chorych z zamkniętym pomostem żylnym, co może wskazywać, że zmniejszona aktywność tego enzymu przyspiesza zamknięcie żylnych pomostów aortalno-wieńcowych.

Słowa kluczowe: paraoksonaza, żylny pomost aortalno-wieńcowy, okluzja

Kardiol Pol 2009; 67: 1063-1068

Adres do korespondencji:

lek. Serkan Cay, Department of Cardiology, Yuksek Ihtisas Heart-Education and Research Hospital, Yasamkent Mah. 3222. Cad.2. Blok (Yakut) No: 37 D: 27 Cayyolu, Ankara, Turkey, tel.: +90 312 217 38 62, e-mail: cayserkan@yahoo.com **Praca wpłynęła:** 14.03.2009. **Zaakceptowana do druku:** 24.06.2009.