Original article

Kardiologia Polska
2011; 69, 9: 924–930
ISSN 0022–9032

Plasma adiponectin levels in acute myocardial infarction and during the postinfarction recovery period in patients with type 2 diabetes mellitus

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Abstract

Background: Adiponectin is a protein produced by the adipose tissue, exhibits potential antiatherogenic properties and is involved in the pathogenesis of insulin resistance. Adiponectin levels are decreased in patients with cardiovascular diseases and type 2 diabetes (DM).

Aim: To assess the changes in adiponectin levels following acute myocardial infarction (MI) and to evaluate the correlation between adiponectin and C-reactive protein (CRP) in patients with DM.

Methods: Coronary angiography was performed in 56 patients with acute MI — 33 patients with DM (23 men, 10 women, mean age 64.0 ± 11.7 years) and 23 non-diabetic subjects (17 men, 6 women, mean age 58.6 ± 9.9 years). All the patients underwent a medical examination and their body mass indexes and waist-to-hip ratios were calculated. Venous blood samples were collected 24 hours, 5 days and 3 weeks following admission.

Results: Plasma adiponectin levels in non-diabetic patients were significantly higher during the postinfarction recovery period than in the acute phase of MI (7.9 ± 3.5 μg/mL *vs* 7.0 ± 2.7 μg/mL). Plasma adiponectin levels in diabetic patients were significantly lower on Day 21 compared to Day 5 (6.0 ± 2.5 μg/mL *vs* 6.7 ± 3.1 μg/mL). The changes in plasma adiponectin levels (the difference in plasma adiponectin levels between Days 5 and 21) negatively correlated with CRP levels ($r = –0.41$, $p = 0.001$). Adiponectin levels were significantly associated with waist circumference (T2DM: $r = –0.34$, $p = 0.04$; control group: $r = –0.48$, $p = 0.001$).

Conclusions: Plasma adiponectin levels in diabetic patients with acute MI were significantly lower during the postinfarction recovery period. These findings suggest a higher and longer adiponectin utilisation in the regeneration process. A strong inflammatory activity in the atheromatous plaque may decrease plasma adiponectin levels.

Key words: diabetes mellitus, myocardial infarction, adiponectin

Kardiol Pol 2011; 69, 9: 924–930

INTRODUCTION

Adiponectin is a physiologically active protein produced and secreted mainly by the white adipose tissue. Adiponectin suppresses proliferation and migration of vascular smooth muscle cells and expression of adhesion molecules with the resulting inhibition of inflammation within the endothelium [1]. It also inhibits tumour necrosis factor (TNF)-induced expression of endothelial proteins which serve as receptors for monocytes, modulating inflammatory response mounted by endothelial cells [2]. Adiponectin reduces the uptake of low density lipoproteins by macrophages and suppresses transformation of monocytes into macrophages that form foam cells. These effects point to the very important role of this hormone in the pathogenesis of atherosclerosis and cardiovascular

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Received: 06.07.2010 **Accepted:** 27.04.2011

(CV) complications. In addition, adiponectin augments nitric oxide synthase phosphorylation, which leads to the increase of nitric oxide levels in the endothelium.

A negative correlation between adiponectin levels and the following factors has also been observed: visceral adipose tissue content, fasting blood glucose, plasma levels of C-reactive protein (CRP) and cholesterol. Experimental and epidemiological studies have shown that adiponectin increases indirectly inhibiting effects of insulin on gluconeogenesis and reduces insulin resistance [3, 4]. This suggests that adiponectin may increase, in an insulin-independent manner, glucose transport into the cells and decrease plasma glucose levels. This peptide also reduces glucose synthesis in the liver without affecting glycolysis or glycogen synthesis. A positive correlation has been observed between adiponectin levels and the degree of phosphorylation of the insulin receptor thyrosine kinase. It cannot be ruled out that this is one of the mechanisms of action of adiponectin responsible for the beneficial effects of this hormone on carbohydrate metabolism.

Changes in adiponectin levels in the course of myocardial infarction (MI) may therefore lead to numerous biochemical abnormalities, including abnormalities of carbohydrate metabolism. Many clinicians consider adiponectin a valuable marker of CV risk [5–7] and a risk factor for type 2 diabetes mellitus (T2DM). Given the role of adiponectin in the pathogenesis of CV disease and T2DM it seems important to investigate the changes in plasma levels of this hormone in T2DM patients with MI.

Inflammation plays an important role the development of myocardial injury in the course of MI, and such markers of inflammation as CRP may reflect the extent of myocardial necrosis and the severity of processes occurring in atheromatous plaques. As adiponectin plays a protective role for the myocardium and may be involved in regeneration processes in the course of MI, the changes in the levels of this hormone during the postinfarction recovery period and the correlations between these levels and the levels of CRP are an interesting research topic.

The aim of our study was to analyse the dynamics of adiponectin levels and the correlations between adiponectin levels and CRP levels during the acute phase of MI and during the postinfarction recovery period in patients with T2DM.

METHODS

Study group

A total of 56 patients hospitalised for electrocardiographically, biochemically and angiographically confirmed MI with the duration of symptoms from onset to admission not exceeding 12 hours were enrolled in the study. This group included 33 patients with T2DM (diagnosed at least 6 months earlier). In the remaining 23 patients the history and the oral glucose tolerance test performed on the 21st day post-MI ruled out any abnormalities of carbohydrate metabolism. Patients with cancer, acute and chronic inflammatory diseases, renal failure and liver failure were excluded from the study. The study protocol was approved by the Medical University of Warsaw Bioethics Committee. All the patients provided written informed consent to participate in the study.

All the patients underwent coronary angiography within a few hours from admission, and those with significant occlusion underwent primary coronary angioplasty. Left ventricular ejection fraction (LVEF), full blood counts, levels of glucose, CRP, troponin and fibrinogen, and lipid profile were measured in all patients. Basic biochemical markers were measured by standard methods. Glycated haemoglobin (HbA1c) was measured by high-performance liquid chromatography (HPLC) using Bio-Rad assays.

Blood samples for adiponectin measment were collected between 24 and 48 hours post-MI, after an overnight fast and no earlier than 6 hours following coronary arteriography. Subsequent blood samples were collected on Days 5 and 21 post-MI, and adiponectin levels were determined by radioimmunoassay (RIA) with the use of Human Adiponectin RIA Kit from LINCO Research. All the patients underwent anthropometric measurements (body mass, height, waist circumference, hip circumference) with calculation of body mass index (BMI) and waist-to-hip ratio (WHR).

Statistical analysis

The statistical analysis was carried out with the use of the Polish version of Statistica 7.1.340.0 from StatSoft Inc. under Academic Licence for the Medical University of Warsaw. The results are presented as means ± SD or as absolute and relative frequencies. The differences in adiponectin levels between the study groups were assessed with the U Mann-Whitney test, and correlations were assessed with the Spearman test. The differences between adiponectin levels determined in consecutive stages of the study were assessed with Wilcoxon's test signed-rank test. A correlation between CRP levels and changes in adiponectin levels between Days 5 and 21 was also assessed (the difference in the levels of this hormone between Days 5 and 21). The threshold of statistical significance was adopted at 5%.

RESULTS

The study groups did not differ significantly in terms of age, sex, BMI and LVEF. There were also no significant differences between the two groups in troponin, fibrinogen, triglycerides and white blood cell counts. Patients with T2DM less frequently smoked and were more frequently hypertensive. In patients with T2DM, coronary arteriography revealed multivessel disease more commonly, although this difference was not significant. In both study groups, comparable outcomes of percutaneous transluminal coronary angioplasty were observed and stents were implanted in the same percentage of the cases. No significant differences between the study groups were observed in the drug treatment offered during hospitalisation. Table 1 presents the full characteristics of the study group and the type of vascular changes detected.

Table 1. Characteristics of the study group

Clinical details	Patients with diabetes mellitus (n = 33)	Patients without diabetes mellitus (n = 23)	P
Sex: females/males	10 (30.3%)/23 (69.7%)	6 (26.1%)/17 (73.9%)	0.7
Age [years]	64.0 ± 11.7	58.6 ± 9.9	0.12
Body mass index [kg/m^2]	29.4 ± 3.9	27.9 ± 3.2	0.21
WHR: females/males	0.93 ± 0.03/1.01 ± 0.03	0.89 ± 0.05/0.96 ± 0.04	0.08/< 0.01
Waist circumference [cm]: females/males	92.5 ± 9/100.2 ± 9.9	85.1 ± 5.9/94.6 ± 10.5	0.1/0.08
Hip circumference [cm]: females/males	98.9 ± 8/98.6 ± 8.6	94.8 ± 5.7/98.4 ± 9.3	0.3/0.7
Smoking	7 (21.2%)	14 (60.8%)	< 0.01
Hypertension	26 (78.8%)	12 (52.1%)	0.03
LVEF	49 ± 9.4	52.7±8.4	0.1
Blood pressure [mm Hg]: systolic/diastolic	136.6 ± 15.4/80.9 ± 10.7	127.1 ± 18.9/74.7 ± 11.4	0.02/0.04
STEMI	20 (60.6%)	18 (78.2%)	0.1
Cholesterol [mg/dL]	177.3 ± 48.7	197.5 ± 33.5	0.03
HDL-cholesterol [mg/dL]	40.9 ± 8.3	45.6 ± 11.3	0.04
LDL-cholesterol [mg/dL]	115.7 ± 48.9	132.8 ± 40.3	0.08
Triglycerides [mg/dL]	171 ± 90.1	152.8 ± 87.3	0.53
C-reactive protein	23.2 ± 26.0	11.5 ± 19.6	< 0.01
Troponin	8.3 ± 15.4	10.5 ± 22.2	0.8
Fibrinogen	436 ± 113	426 ± 114	0.7
White blood cell count	10.1 ± 2.1	9.6 ± 2.4	0.4
Platelet count	217 ± 62	220 ± 91	0.8
HbA1c [%]	8.0 ± 1.5	5.8 ± 0.4	< 0.01
Glucose [mg/dL]:			
Day 2	156.9 ± 39.5	115.1 ± 17.8	< 0.01
Day 5	154.5 ± 48.7	112.7 ± 19.1	< 0.01
Day 21	144.4 ± 38.7	111 ± 16.8	< 0.01
No. of vascular changes:			0.2
One-vessel disease	15 (45.4%)	14 (60.9%)	
Two-vessel disease	12 (36.4%)	7 (30.4%)	
Three-vessel disease	6 (18.2%)	2 (8.7%)	
Infarct-related artery:			0.2
RCA	11 (33.3%)	13 (56.6%)	
LAD	12 (36.4%)	7 (30.4%)	
LM	2 (6.1%)	0 (0%)	
Cx	8 (24.2%)	3 (13%)	0.2
Stenting	27 (81.8%)	19 (82.6%)	0.6
Initial TIMI 0–1 flow	27 (82%)	20 (87%)	0.4
Final TIMI 3 flow	30 (91%)	22 (95%)	0.4
Drug therapy during hospitalisation:			
Aspirin	27 (81.8%)	21 (91.3%)	0.2
Beta-blockers	29 (87.9%)	21 (91.3%)	0.5
ACE inhibitors	31 (94%)	22 (95.7%)	0.6
Statins	28 (84.8%)	19 (82.6%)	0.5
Clopidogrel	29 (87.9%)	20 (87%)	0.6

WHR — waist to hip ratio; LVEF — left ventricular ejection fraction; STEMI — ST elevation myocardial infarction; RCA — right coronary artery; LAD — left anterior descending artery; LM — left main coronary artery; Cx — left circumflex artery; TIMI — Thrombolysis in Myocardial Infarction; ACE — angiotensin converting enzyme

Table 2. Comparison of adiponectin levels [μg/mL] between patients with or without diabetes mellitus

	Patients with diabetes mellitus (n = 33)	Patients without diabetes mellitus (n = 23)	P
Day 2	6.34 ± 2.63	6.81 ± 2.71	0.4
Day 5	6.71 ± 3.1	7.0 ± 2.73	0.5
Day 21	6.06 ± 2.5	7.97 ± 3.56	0.07

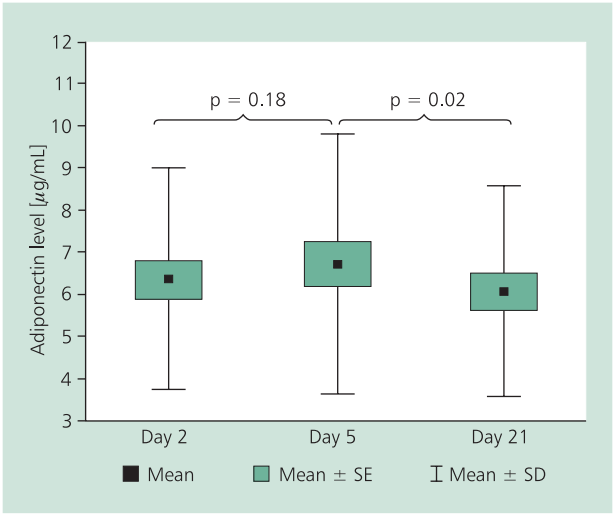

Figure 1. Adiponectin levels in patients with diabetes mellitus

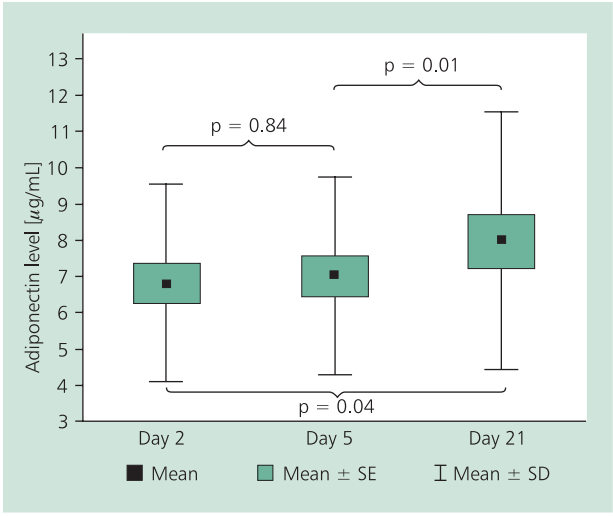

Figure 2. Adiponectin levels in patients without diabetes mellitus

At all three time points (Days 2, 5 and 21), adiponectin levels in patients with T2DM tended to be lower than those in the non-diabetic patients, although the difference at any of the time points did not reach statistical significe (Table 2). In both study groups, there were no significant differences in adiponectin levels between Days 2 and 5 post-MI (Figs. 1, 2). In patients with T2DM, adiponectin levels on Day 21 were significantly lower than those on Day 5 post-MI ($p = 0.02$; Fig. 1), while those in the control group were higher ($p = 0.01$; Fig. 2). This finding was observed in both sexes. Adiponectin levels in women, at each time point, were only slightly higher than those in men, both in patients with T2DM and without T2DM.

When we analysed other factors that might affect adiponectin levels on Day 21 we did not observe any significant differences relative to the type and outcome of treatment or the blood vessel responsible for the MI. Similarly, adiponectin levels in patients with non-ST-elevation MI (NSTEMI) did not differ significantly from adiponectin levels observed in patients with ST-elevation MI (STEMI). There were also no significant correlations between adiponectin levels and the levels of troponin, fibrinogen, cholesterol, glucose and LVEF or BMI (Table 3). In both study groups, however, adiponectin levels negatively correlated with waist circumference and body mass, and in non-diabetic patients, adiponectin levels additionally correlated negatively with hip circumference and triglyceride levels (Table 3). The CRP levels negatively correlated with changes in adiponectin levels between Days 5 and 21 of the study ($r = –0.41$, $p = 0.001$) (Fig. 3).

DISCUSSION

In our study, both in patients with and without T2DM, adiponectin levels on Day 2 post-MI were lower than those on Day 5 of the study. It should be emphasised that these findings are consistent with the results of previous studies investigating this issue. The available literature reports significantly lower adiponectin levels 72 hours after-MI compared to those observed 7 days post-MI [8] as well as lower levels of this hormone 3 days after-MI compared to 1 or 5 days post-MI [9]. Our results suggest that in diabetic patients, adiponectin levels during the postinfarction recovery period are lower than baseline levels, while in the non-diabetic group, there was a significant increase in adiponectin levels compared to those observed during the acute phase of MI. Kojima et al. [8] also observed slightly lower levels of adiponectin in patients with MI 4 weeks after admission compared to the 7th day post-MI. In another study, which investigated men only, there were no significant changes in adiponectin levels during a one-month

Table 3. Correlations between adiponectin and anthropometric as well as biochemical parameters

	Patients with diabetes mellitus (n = 33)		Patients without diabetes mellitus (n = 23)	
	r	p	r	p
Troponin	–0.04	0.8	–0.21	0.32
Fibrinogen	–0.18	0.29	–0.02	0.8
Left ventricular ejection fraction	–0.07	0.66	0.24	0.25
Body mass index	–0.25	0.15	–0.37	0.07
Waist to hip ratio	–0.31	0.07	–0.19	0.36
Waist circumference	–0.34	0.04	–0.48	0.01
Hip circumference	–0.2	0.25	–0.43	0.03
Body mass	–0.38	0.02	–0.48	0.02
Cholesterol	–0.11	0.52	–0.31	0.14
Triglycerides	–0.2	0.25	–0.5	0.01
HDL-cholesterol	–0.18	0.31	0.38	0.07
LDL-cholesterol	–0.06	0.7	–0.28	0.18
Glucose:				
Day 2	0.17	0.32	–0.01	0.94
Day 5	–0.17	0.32	0.23	0.27
Day 21	0.02	0.88	0.14	0.51

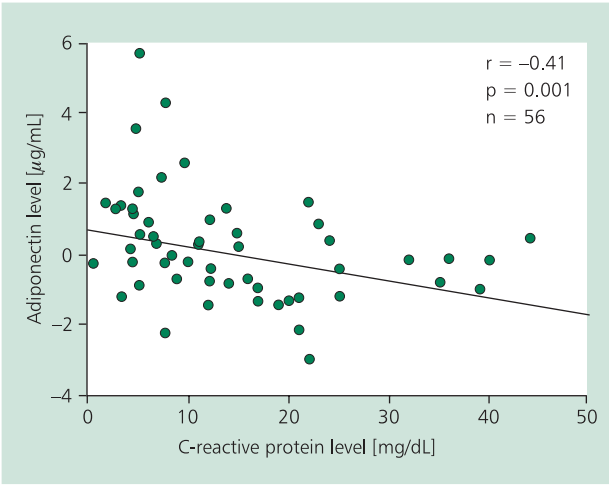

Figure 3. Correlation between C-reactive protein levels and the changes in adiponectin levels between Days 5 and 21 of the study

follow-up after MI [9]. In contrast to our study, the authors did not, however, analyse adiponectin levels separately for patients with or without T2DM, which makes it impossible to perform an accurate comparison of both studies. In the same study, however, the degree of decrease in adiponectin levels within the month after admission negatively correlated with CRP levels. Also in our study, changes in adiponectin levels between Days 5 and 21 showed a significant negative correlation with CRP levels during the acute phase of MI.

The exact mechanisms responsible for the fluctuations in the adiponectin levels in the course of MI are unclear. Okamaoto et al. [10] showed that adiponectin binds with type I, III and V collagen, a component of the vascular wall, which might affect the fluctuations of adiponectin levels in the course of MI, particularly in patients with advanced vascular changes. While Okamaoto et al. [10] observed the presence of this protein in injured vascular walls, they did not demonstrate it in normal blood vessels. It was also shown that during myocardial injury adiponectin accumulates in the myocardium [11]. These data suggest that adiponectin is a protein which may accumulate in the vascular wall when the endothelial barrier is injured. Adiponectin might reach ruptured atheromatous plaques, participating in reparative and regenerative processes and limiting the MI area. It could therefore be assumed that accumulation of adiponectin in the vascular subendothelial space is the main factor responsible for fluctuations of this hormone in the course of MI. Adiponectin also protects cardiac myocytes from inflammatory and proapoptotic factors. Additionally, the persistently decreased adiponectin levels in T2DM patients during the postinfarction recovery period may suggest a higher consumption of this hormone in the myocardium in this group of patients.

There have also been reports of adiponectin synthesis in the cytoplasm of cardiac myocytes and of its secretion outside the cell. Results of the studies by Pineiro et al. [12] suggest that local formation of adiponectin in cardiac myocytes may be an inseparable mechanism that regulates myocardial function and metabolism. Therefore, the significantly lower levels

of adiponectin during the postinfarction recovery period in patients with T2DM may also suggest a limited production of adiponectin during myocardial ischaemia and necrosis.

The results of our study do not allow to compare the observed changes in adiponectin levels with baseline levels, e.g. pre-MI. Studies conducted so far have reported decreased adiponectin levels in MI patients compared to those without MI or patients with stable ischaemic heart disease [13, 14]. In patients with MI, lower levels of adiponectin in the first 2 or 3 days of the disease have been reported compared to the levels observed on admission [8, 9]. Adiponectin exhibits antiatherogenic properties and plays a protective role in the pathogenesis of CV complications. For this reason, the lower levels of adiponectin on Day 21 in T2DM patients could be an additional factors adversely affecting the outcomes in this group of patients.

As regards adiponectin levels during the acute phase of MI, it should be mentioned that levels of this hormone correlate with the activity of the insulin receptor and with insulin sensitivity, as shown in animal models and human studies [15, 16]. For this reason adiponectin is attributed a regulatory role in glucose and lipid metabolism in insulin-sensitive tissues. It has been also suggested that reducing insulin sensitivity (at the level of the liver) under conditions of hypoadiponectinaemia may lead to increased production of glucose in the liver [17]. Lower adiponectin levels during the postinfarction recovery period in T2DM patients may therefore be an additional factor adversely affecting the prognosis, both in terms of ischaemic risk and further course of T2DM itself. Further research is, however, necessary into the role of adiponectin levels during the postinfarction recovery period as a prognostic factor in T2DM patients following MI.

CONCLUSIONS

Diabetic patients with MI have significantly lower adiponectin levels during the postinfarction recovery period, which may suggest a higher and longer utilisation of adiponectin in reparative and regenerative processes in this patient group. Active inflammation within the atherosclerotic plaque may be one of the factors that reduces the level of adiponectin.

Conflict of interest: *none declared*

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Analiza stężenia adiponektyny u chorych na cukrzycę typu 2 w ostrej fazie zawału serca oraz w okresie rekonwalescencji

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Streszczenie

Wstęp: Adiponektyna uczestniczy w regulacji insulinowrażliwości i metabolizmu lipidów; ma również właściwości przeciwzapalne i przeciwmiażdżycowe.

Cel: Celem pracy była analiza dynamiki zmian stężenia adiponektyny i ich korelacji ze stężeniem białka C-reaktywnego (CRP) w ostrej fazie zawału serca (MI) oraz w okresie rekonwalescencji u chorych na cukrzycę typu 2 (DM2).

Metody: Do badania zakwalifikowano 56 pacjentów z MI, w tym 33 z DM2 (10 kobiet, 23 mężczyzn; wiek 64,0 ± 11,7 roku) i 23 osób bez cukrzycy (17 mężczyzn, 6 kobiet; wiek 58,6 ± 9,9 roku — grupa kontrolna). Stężenia adiponektyny oznaczano metodą radioimmunoenzymatyczną w 2., 5. i 21. dobie MI. U wszystkich pacjentów oznaczono stężenia glukozy, CRP, troponiny i HbA_{1c}. Podczas analizy statystycznej wykorzystano testy Spearmana i Wilcoxona.

Wyniki: Nie zaobserwowano istotnych statystycznie różnic między stężeniem adiponektyny w 2. i 5. dobie MI w obydwu badanych grupach. U chorych z DM2 stężenia adiponektyny w 21. dobie badania były istotnie niższe niż w 5. dniu MI (6,0 ± 2,5 *v.* 6,7 ± 3,1 μg/ml; p = 0,02), natomiast w grupie kontrolnej wyższe (7,9 ± 3,5 *v.* 7,0 ± 2,7 μg/ml; p = 0,01). Stężenia CRP istotnie ujemnie korelowały ze zmianami stężenia adiponektyny między 5. i 21. dobą badania (r = –0,41; p = 0,001). Ponadto stężenia adiponektyny ujemnie korelowały z obwodem talii zarówno u chorych na cukrzycę (r = –0,34; p = 0,04), jak i w grupie kontrolnej (r = –0,48; p = 0,01).

Wnioski: Niższe stężenia adiponektyny w okresie rekonwalescencji wśród pacjentów z MI i DM2 mogą świadczyć o większym i dłuższym wykorzystywaniu adiponektyny w procesach naprawczych i regeneracyjnych. Aktywny proces zapalny w obrębie blaszki miażdżycowej może być jednym z czynników redukujących stężenia adiponektyny.

Słowa kluczowe: cukrzyca, zawał serca, adiponektyna

Kardiol Pol 2011; 69, 9: 924–930

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Praca wpłynęła: 06.07.2010 r. **Zaakceptowana do druku:** 27.04.2011 r.