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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Clinical significance and determinants of prompt recruitment collaterals during primary percutaneous coronary intervention

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Abstract

Background: Due to ischaemic time delays from the chest pain occurrence in acute ST elevation myocardial infarction (STEMI), prompt recruitment collaterals (PRCCs) to infarct-related artery (IRA) are the major protective structures during this period.

Aim: We aimed to investigate the clinical significance and determinants of PRCCs in acute STEMI patients.

Methods: A total of 1375 consecutive acute STEMI patients were prospectively enrolled in the study. The patients were divided into two groups, according to PRCCs to IRA; Rentrop ≤ 1 were defined as inadequate collateral development (ICD) group and Rentrop ≥ 2 defined as adequate collateral development (ACD) group.

Results: Patients in the ICD group had higher incidence of baseline risk characteristics, including older age, hypertension, and diabetes mellitus; however, pre-infarct angina incidence was lower than in the ACD group ($p < 0.05$ for all). In addition, the ICD group had worse haemodynamic status on admission and 30-day mortality. Compared to the ACD group, the non-IRA chronic total occlusion (CTO), peak troponin-T, N-terminal pro-B-type natriuretic peptide (NT-proBNP), and high sensitivity C-reactive protein (hs-CRP) levels were higher in the ICD group. On multivariate logistic regression analysis, non-IRA CTO ($\beta = 3.114$, 95% CI 1.382–7.017, $p < 0.006$) with pre-infarction angina together with higher values of peak troponin-T, NT-proBNP, and hs-CRP were associated with PRCCs in acute STEMI.

Conclusions: Taking into account that the main message of the study is that if patients have higher cardiac biomarkers and adverse clinical findings (which, of note, may show the extent of myocardial infarction) and have non-IRA CTO, there is a higher chance that they will have inadequate collateralisation.

Key words: arteriogenesis, collateral network, chronic total occlusion, ST elevation myocardial infarction, mortality

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INTRODUCTION

Primary percutaneous coronary intervention (PCI) is the best choice for rapid restoration of ante-grade flow in acute ST elevation myocardial infarction (STEMI) patients [1]. However, time delays from chest pain occurrence to balloon inflation in the culprit artery are often inevitable and cause a prolongation in ischaemic time. During this ischaemic time, the prompt occurrence of coronary collaterals (PRCCs) to infarct-related artery (IRA) plays a pivotal protective role in myocardial salvage, ventricular remodelling, and cardiac outcomes [2, 3].

Acute thrombotic occlusion and chronic total occlusion (CTO) of the coronary artery are two processes that can stimulate collateral development due to pressure gradient. Both occlusion types can only be seen together in STEMI patients who have had a concurrent non-IRA CTO. Despite the beneficial effects of PRCC to IRA, CTO in a non-IRA that has chronic coronary collaterals is related with adverse clinical outcomes in patients with acute STEMI. In particular, non-IRA CTO was found to be an independent predictor of 30-day mortality in these patients [4, 5]. In addition, various factors may contribute to the development of PRCCs. Therefore, we aimed to investigate the clinical significance and determinants of PRCCs in patients with acute STEMI.

METHODS

From June 2014 to December 2015, 1375 acute STEMI patients who underwent primary PCI were prospectively considered eligible for the study. Patients with symptoms of

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ischaemic chest pain (at least for 30 min, and no-longer than 12 h), and an electrocardiography (ECG) showing ST-segment elevation of 0.1 mV in two or more limb leads or 0.2 mV in two or more contiguous precordial leads, or presumed new left bundle branch block, were included into the present study. Angiographic assessment of PRCCs to IRA from patent vessels, Thrombosis in Myocardial Infarction (TIMI) flow, and non-IRA CTO were evaluated on angiograms before antegrade restoration of IRA by two experienced investigators, who were blinded to the study data. Rentrop classification was used for grading PRCCs to IRA territory from patent vessels. Rentrop classification is defined below [6]: grade 0 — no visible filling of any collateral channel; grade 1 — filling of the side branches of the occluded artery, with no dye reaching the epicardial segment; grade 2 — partial filling of the epicardial vessel; and grade 3 — complete filling of epicardial vessel by collateral vessels. After classification of collateral blood flow to IRA, patients who had Rentrop ≤ 1 were defined as the inadequate collateral development (ICD) group, and those who had Rentrop ≥ 2 were defined as the adequate collateral development (ACD) group.

All patients were carefully assessed for pre-infarct angina. Pre-infarct angina defined as at least one episode of typical transient (< 30 min) chest pain (Canadian Cardiovascular Society ≥ 1) in the 24 h before the index event.

The baseline characteristics of patients, including age, gender, body mass index, hypertension, hyperlipidaemia, diabetes mellitus (DM), smoking status, family history, previous PCI, and pre-infarction angina, were recorded from all patients. The left ventricular ejection fraction (LVEF) was measured within the first week of post-myocardial infarction (MI) period by an experienced, blinded sonographer using Simpson's method [7]. All blood samples were drawn at admission including blood glucose, cell blood count, lipid profile, troponin T, N-terminal pro-B-type natriuretic peptide (NT-proBNP), and high-sensitivity C reactive protein (hs-CRP), and troponin T level was obtained before intervention and every 12 h up to 24 h after intervention.

Patients who had TIMI flow > 1 at first contrast injection, patients with chronic stable angina, patients with chronic and active inflammatory diseases, oncological and haematological disorders, severe renal or hepatic failure, asthma, and patients admitted later than 12 h were excluded from the study. The Local Ethics Committee approved the study protocol, and each participant provided written, informed consent.

Statistical analysis

Baseline characteristics of study patients summarised as percentages and frequencies for categorical variables and as the means with standard deviation for continuous variables. Kolmogorov-Smirnov test was used to determine whether they were normally distributed. Categorical variables were compared using the χ^2 test or Fisher exact tests. The effects of variables on collateral development were calculated using univariate analysis. Variables showing an unadjusted $p < 0.10$ in logistic regression analysis were determined as risk markers. All significant parameters in the univariate analysis were selected in the multivariate model. Multivariate logistic regression analysis was performed with a stepwise, backward selection method to determine the independent predictors of collateral development in acute STEMI patients. Statistical analysis was performed using SPSS 17.0 for Windows (SPSS Inc., Chicago, Illinois, USA). A p-value < 0.05 was considered as significant.

RESULTS

Of the 1375 patients who underwent primary PCI, 278 (20.2%) were identified as the ACD group, and 1097 (79.8%) were identified as the ICD group. Patients in the ICD group had higher incidence of baseline risk characteristics, including older age (57.7 ± 11.3 vs. 56.2 ± 10.8 years, $p = 0.040$), hypertension (44.2% vs. 36.3%, $p = 0.021$), and DM (23.7% vs. 18%, $p = 0.045$); however, pre-infarct angina (20.8% vs. 26.6%, $p = 0.042$) incidence was lower when compared with the ACD group. Laboratory analysis showed that the ICD group had higher peak troponin T and NT-proBNP level and lower hs-CRP level, when compared to the ACD group. The other baseline and laboratory findings of the study groups were similar (Table 1).

Non-IRA CTO was obtained in 10.6% of the study patients, who had significantly higher 30-day mortality (14.4% vs. 3.4%; $p < 0.001$) when compared to those without. In addition to poor collateralisation, in patients with co-existing CTO, the CTOs supplied by IRA were all in the ICD group.

ICD group had worse haemodynamic status on admission and were more likely to have presented with higher incidence of heart failure (at least Killip class 2, and lower LVEF), when compared with the ACD group. In addition to higher incidence of multi-vessel disease (14.7% vs. 20.2%, $p = 0.040$) and non-IRA CTO (2.5% vs. 12.7%, $p < 0.001$) (Fig. 1) in the ICD group, the 30-day mortality rate (1.8% vs. 5.3%, $p = 0.010$) was also higher than in the ACD group (Fig. 2, Table 2).

In multivariate logistic regression analysis, we observed that non-IRA CTO ($\beta = 3.114$, 95% CI 1.382–7.017, $p < 0.006$), patients with pre-infarction angina, as well as higher levels of peak troponin T, NT-proBNP, and hs-CRP were independent predictors of PRCCs to IRA in acute STEMI patients (Table 3).

DISCUSSION

Our results showed that non-IRA CTO with pre-infarction angina, as well as higher levels of NT-proBNP, peak troponin T, and hs-CRP were associated with PRCCs to IRA in acute STEMI patients.

PRCCs were observed in 10–40% of the patients after onset of acute STEMI, and consisted with our findings [2, 8, 9].

Table 1. Baseline and laboratory features of study groups

Variables	ICD group (n = 1097)	ACD group (n = 278)	p*
Age [years]	57.7 ± 11.3	56.2 ± 10.8	**0.040**
Gender (male)	834 (76%)	214 (77%)	0.813
Body mass index [kg/m^2]	27.2 ± 4.2	27.4 ± 4.0	0.386
Hypertension	484 (44.2%)	101 (36.3%)	**0.021**
Diabetes mellitus	259 (23.7%)	50 (18%)	**0.045**
Hyperlipidaemia	351 (32%)	88 (31.7%)	0.943
Smoking	638 (58.3%)	170 (61.2%)	0.413
Family history	539 (49.2%)	138 (49.6%)	0.946
Previous PCI	160 (14.6%)	46 (16.5%)	0.399
Pre-infarction angina	228 (20.8%)	74 (26.6%)	**0.042**
Glucose [mg/dL]	164 ± 69	156 ± 70	0.086
Total cholesterol [mg/dL]	190 ± 45	192 ± 43	0.864
Triglyceride [mg/dL]	140 ± 128	148 ± 125	0.311
HDL-C [mg/dL]	39.5 ± 9.5	40 ± 9.9	0.257
LDL-C [mg/dL]	134 ± 41	132 ± 38	0.503
WBC count [×1000/μL]	12.2 ± 3.9	11.9 ± 3.3	0.472
Haemoglobin [mg/dL]	13.8 ± 1.7	13.7 ± 1.8	0.641
Platelet count [×1000/μL]	233 ± 75	232 ± 68	0.998
Mean platelet volume	10.3 ± 1.14	10.2 ± 1.22	0.123
Creatinine [mg/dL]	0.88 ± 0.3	0.88 ± 0.4	0.875
Hs-CRP [mg/L]	5.59 ± 2.85	4.87 ± 2.25	**< 0.001**
Peak troponin T [ng/mL]	2194 ± 2513	1712 ± 2096	**0.001**
NT-proBNP [pg/mL]	818 ± 1379	389 ± 465	**< 0.001**

Data are presented as mean ± standard deviation and number (percentage). Significant p values ($p < 0.05$) are indicated in boldface; *t-test for independent variables and chi-square test; ACD — adequate collateral development; HDL-C — high-density lipoprotein cholesterol; Hs-CRP — high-sensitive C-reactive protein; ICD — inadequate collateral development LDL-C — low-density lipoprotein cholesterol; NT-proBNP — N terminal pro-B-type natriuretic peptide; WBC — white blood cell

In several previous studies, it was shown that angiographically visible collateral blood flow to IRA territory has beneficial effects on microvascular circulation, infarct size, procedural success, and cardiac outcomes in STEMI patients [2, 3]. Perez-Castellano et al. [10] demonstrated that the majority of patients who died from cardiogenic shock had no collateral supply to IRA. Likewise, we observed that patients in ICD the group had significantly higher incidences of heart failure (Killip class ≥ 2) and 30-day mortality. In addition, if a patient had adverse clinical findings (which, of note, may show the extent of MI), there was a higher chance that he/she would have inadequate collateralisation.

Angiogenesis and arteriogenesis are the main processes for developing collateral circulation as a response to myocardial ischaemia [11, 12]. Angiogenesis is a highly coordinated process and means formation of new capillary blood vessel sprout from a pre-existing blood vessel, which requires interaction between endothelium, extracellular matrix, and surrounding cells mediated by growth factors [13]. However, arteriogenesis occurs by structural remodelling of preformed collaterals that depends on vessel wall shear stress and blood flow redistribution [14]. This process begins a short time after arterial occlusion by the shear stress which leads to endothelial activation [15]. Arteriogenesis is especially observed in CTO or IRA territory because of the pressure decrease in the occluded artery. Moreover, acute STEMI with a non-IRA CTO is the single disease that includes both types of total occlusion. In HORIZONS-AMI trial, the adverse effects of a non-IRA CTO on procedural success, and 30-day mortality, have been shown in these patients [4]. We demonstrated that as well as being a predictor of 30-day mortality, non-IRA CTO is also an independent predictor of inadequate collateralisation. A possible explanation of this inverse relationship between non-IRA CTO and inadequate collateralisation may be the neutralisation of the pressure gradient in patients with CTO, which is supplied by the IRA. In the present study, all CTOs supplied by the IRA were in the ICD group, which supports this hypothesis. Furthermore, in this situation the myocardial

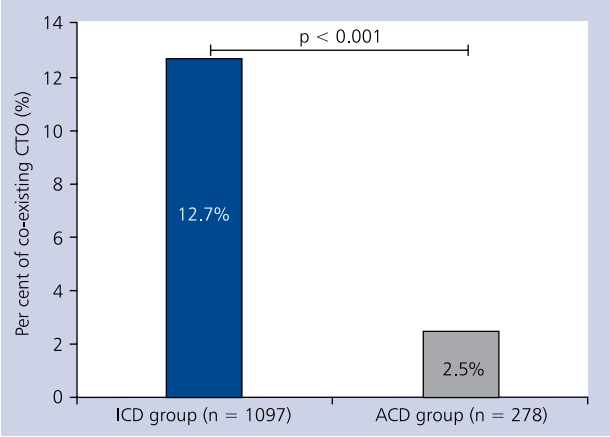

Figure 1. Demonstration of non-infarct-related artery chronic total occlusion (CTO) rates within groups (12.7% vs. 2.5%, p < 0.001); ICD — inadequate collateral development; ACD — adequate collateral development

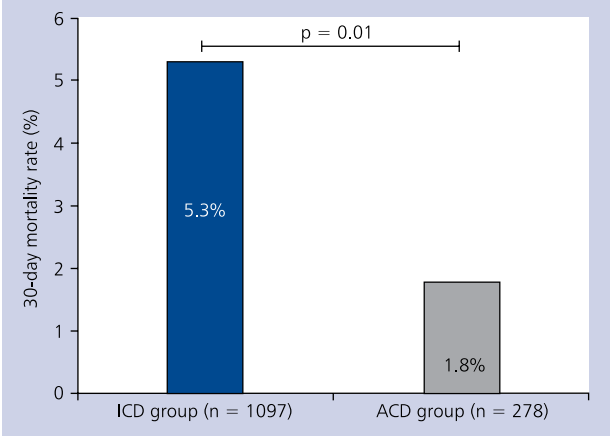

Figure 2. 30-day mortality rate following primary percutaneous coronary intervention for ST segment elevation myocardial infarction in patients with inadequate collateral development (ICD) group compared with adequate collateral development (ACD) group (5.3% vs. 1.8%, p = 0.01)

Table 2. Clinical and angiographic characteristics of study groups

Variables	ICD group (n = 1097)	ACD group (n = 278)	p*
Killip class ≥ 2	88 (8%)	9 (3.2%)	**0.004**
Chest pain duration [h]	2.28 ± 2.2	2.06 ± 2.1	0.143
Heart rate [bpm]	81.5 ± 19.4	78.4 ± 16.4	**0.007**
Systolic BP [mm Hg]	121.8 ± 24.5	127 ± 25.1	**0.002**
Diastolic BP [mm Hg]	73.5 ± 14.4	75.4 ± 14.8	**0.038**
LVEF [%]	49.7 ± 9.0	51.4 ± 8.5	**0.004**
Culprit artery, LAD	458 (41.8%)	115 (41.4%)	0.535
Sullivian Score	38.2 ± 13	36.7 ± 12.7	0.075
Multivessel disease	221 (20.2%)	41 (14.7%)	**0.040**
Patients with non-IRA CTO	138 (12.7%)	7 (2.5%)	**< 0.001**
30-day mortality	58 (5.3%)	5 (1.8%)	**0.010**

Data are presented as mean ± standard deviation and number (percentage). Significant p values (p < 0.05) are indicated in boldface;
*t-test for independent variables and chi-square test; ADC — adequate collateral development; BP — blood pressure; CTO — chronic total occlusion; ICD — inadequate collateral development; IRA — infarct related artery; LVFF — left ventricular ejection fraction

region at ischaemic risk would extend to the IRA and CTO territory, which leads to higher peak troponin T and NT-proBNP level, as obtained in the ICD group. It should be underlined that higher levels of peak troponin T and NT-proBNP are the results of inadequate collateralisation rather than a predictor of PACCs.

It is known that microvascular reperfusion is a major factor of procedural success and cardiac consequences following primary PCI [16]. In previous studies, patients with post-procedural lower rates of TIMI 2/3 flow, myocardial blush grade 3, and ST resolution, which are the determinants of microvascular dysfunction, have been found in non-IRA CTO patients [5]. A possible explanation of microvascular dysfunction in the presence of a CTO in another myocardial region may be due to the impairment of collateral flow to IRA territory as a result of decreased collateral network reserve. Thus, we found inadequate collateralisation in patients with concurrent non-IRA CTO.

The effects of pre-existing non-IRA CTO together with coronary disease extension are critical factors for collateral presence. Hence, we calculated coronary extension score by using Sullivan score scoring system [17] and found similar scores within groups. In this regard, we can conclude why the presence of previous coronary advanced atherosclerosis was not different within the two groups. In this study the ICD group had more coronary risk factors (age, hypertension, DM),

Table 3. Significant predictors of prompt recruitment collaterals to infarct-related artery in univariable and multivariable logistic regression analysis

	Univariate			Multivariate		
	OR	95% CI	p	OR	95% CI	p
Age	1.002	0.989–1.015	0.800			
Hypertension	0.797	0.599–1.061	0.120			
Diabetes mellitus	0.878	0.601–1.281	0.499			
Killip class ≥ 2	1.084	0.501–2.344	0.837			
Systolic BP	1.006	1.000–1.011	**0.044**	1.005	1000–1.011	0.058
LVEF	1.000	0.983–1.018	0.994			
Multivessel disease	1.192	0.793–1.790	0.399			
Pre-infarction angina	0.700	0.512–0.959	**0.026**	0.693	0.507–0.948	**0.022**
Hs-CRP [mg/L]	0.934	0.885–0.985	**0.012**	0.932	0.883–0.982	**0.009**
Peak troponin T (per 100 ng/mL increase)	0.994	0.987–1.000	**0.041**	0.994	0.988–1.000	**0.036**
NT-proBNP (per 100 pg/mL increase)	0.972	0.951–0.992	**0.007**	0.970	0.951–0.989	**0.003**
Non-IRA CTO	2.963	1.304–6.733	**0.009**	3.114	1.382–7.017	**0.006**

Significant p values ($p < 0.05$) are indicated in boldface; BP — blood pressure; CI — confidence interval; CTO — chronic total occlusion; Hs-CRP — high sensitive C-reactive protein; IRA — infarct related artery; NT-proBNP — N terminal pro-B-type natriuretic peptide; OR — odds ratio

which leads to induced coronary narrowing and collateral supplying; however, previous clinical and experimental studies on collateral development, particularly in diabetic patients, have reported conflicting results. It is known that endothelial dysfunction, decreased nitric oxide (NO) synthesis, decreased endothelial progenitor cell (EPC) proliferation, and impaired vascular endothelial growth factor (VEGF)-induced EPC proliferation are well-known characteristics of DM [8, 18, 19]. In contrast, these factors are crucial for collateral development.

Previous primary PCI studies demonstrated that the duration of angina, and the level of antegrade TIMI flow and pre-infarction angina are predictors of collateral supply [20, 21]. The cardio-protective mechanism of pre-infarction angina is not fully determined. However, ischaemic preconditioning and collateral development have been suggested as possible explanations [22]. Accordingly, the pressure gradient may occur before the index event for stimulating shear stress. Similarly, we found that pre-infarction angina is an independent predictor of adequate collateralisation in patients undergoing primary PCI.

Increased blood level of hs-CRP, a biomarker of low grade inflammation, is related with unfavourable outcomes in patients with cardiovascular diseases [23]. Increased hs-CRP levels resulted in impaired endothelial function and lead to decrease in expression of endothelial-mediated NO and prostacyclin [24]. In addition, previous evidence suggests that NO has a positive modulation over both angiogenesis and arteriogenesis, which are the main contributors of coronary collateral circulation (CCC) development [10, 11]. Previously, increased hs-CRP and inadequate collateralisation have been shown in CTO patients [25]. In the present study, we also found an inverse correlation between PRCC to IRA and hs-CRP levels in acute STEMI patients.

Limitations of the study

This was a single-centre, observational study. Coronary flow index analysis could not be used to evaluate the CCC due to unavailability of intracoronary velocity or pressure techniques. In addition, the diameter of collateral vessels under 100 μm could not be observed due to angiographic limitations. We measured the necrotic myocardial volume by peak troponin T levels due to lack of cardiac magnetic resonance evaluation for quantitative analysis, which is an important limitation of this study. Further studies are warranted to either confirm or contradict these findings.

CONCLUSIONS

Taking that into account that the main message of the study is that a lack of proper collateral supply may lead to higher cardiac biomarkers and adverse clinical findings (which, of note, may show the extent of MI) and non-IRA CTO, there is higher chance that these patients will have inadequate collateralisation. The adverse effects of non-IRA CTO on PRCC may be a major promoter of early mortality in these patients. In addition, CTO-PCI in a stable setting may provide a standby collateral network for preventing adverse outcomes of future STEMI.

Conflict of interest: *none declared*

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Znaczenie kliniczne i uwarunkowania szybkiej dostępności krążenia obocznego w pierwotnej przezskórnej interwencji wieńcowej

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Streszczenie

Wstęp: Ze względu na opóźnienie między wystąpieniem niedokrwienia a pojawieniem się bólu w klatce piersiowej w ostrym zawale serca z uniesieniem odcinka ST (STEMI) szybko dostępne naczynia oboczne (PRCC) w obszarze unaczynienia tętnicy odpowiedzialnej za zawał (IRA) są strukturami mającymi w tym okresie najważniejsze znaczenie ochronne.

Cel: Badanie przeprowadzono w celu oceny znaczenia klinicznego i czynników determinujących PRCC u chorych z ostrym STEMI.

Metody: Do badania włączono prospektywnie 1375 kolejnych pacjentów z ostrym STEMI. Chorych podzielono na dwie grupy w zależności od PRCC w obszarze IRA; osoby z oceną w skali Rentropa wynoszącą ≤ 1 przydzielano do grupy z niedostatecznie rozwiniętym krążeniem obocznym (ICD), a osoby z oceaną w skali Rentropa wynoszącą ≥ 2 — do grupy z wystarczająco rozwiniętym krążeniem obocznym (ACD).

Wyniki: U chorych z grupy ICD stwierdzono częstsze występowanie czynników ryzyka w ocenie wyjściowej, w tym starszy wiek, nadciśnienie tętnicze i cukrzycę, jednak przedzawałowe objawy dławicowe występowały u tych pacjentów rzadziej niż w grupie ACD ($p < 0,05$ dla wszystkich porównań). Ponadto grupa ICD charakteryzowała się gorszymi parametrami hemodynamicznymi przy przyjęciu do szpitala oraz wyższą śmiertelnością w okresie 30 dni. W tej grupie częściej stwierdzano obecność przewlekle niedrożnej tętnicy innej niż IRA (non-IRA CTO) oraz wyższe wartości takich parametrów, jak: maksymalne stężenie troponiny T, stężenie N-końcowego propeptydu natriuretycznego typu B (NT-proBNP) i białka C-reaktywnego o wysokiej czułości (hs-CRP) niż w grupie ACD. W wieloczynnikowej analizie regresji logistycznej wykazano, że obecność non-IRA CTO ($\beta = 3,114$; 95% CI 1,382–7,017; $p < 0,006$) z występowaniem przedzawałowych bólów dławicowych, a także wyższymi stężeniami troponiny T, NT-proBNP i hs-CRP wiązały się z PRCC w ostrym STEMI.

Wnioski: Głównym wnioskiem z badania jest stwierdzenie, że u chorych z wysokimi stężeniami biomarkerów sercowych oraz niekorzystnymi wynikami badania klinicznego (które mogą wskazywać na wielkość zawału), a także z non-IRA CTO, występuje większe ryzyko tego, że mają niedostatecznie rozwinięte krążenie oboczne.

Słowa kluczowe: arteriogeneza, sieć naczyń obocznych, przewlekła całkowita niedrożność, STEMI, śmiertelność

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