ORIGINAL ARTICLE

Presence of coronary collaterals in ST-elevation myocardial infarction patients does not affect long-term outcome

Tomasz Rechciński, Aleksandra Jasińska, Jan Z. Peruga, Jakub Foryś, Maria Krzemińska-Pakuła, Zbigniew Bednarkiewicz, Ewa Trzos, Jarosław D. Kasprzak

Department of Cardiology, Medical University of Lodz, The Bieganski Hospital, Łódź, Poland

KEY WORDS

collateral circulation, myocardial infarction, prognosis

ABSTRACT

INTRODUCTION The significance of coronary collateral circulation in the prognosis of patients after myocardial infarction remains disputable.

OBJECTIVES The aim of the study was to evaluate the effect of coronary collateral circulation, assessed by the Rentrop score, on long-term prognosis in patients treated with primary percutaneous coronary intervention (PCI) in ST-segment elevation myocardial infarction (STEMI).

PATIENTS AND METHODS Coronary collateral flow was assessed by angiography in 330 patients with myocardial infarction using the Rentrop score. Patients were followed up for the mean period of 26 ±12 months with the clinical endpoints of cardiac death, nonfatal reinfarction, and repeat percutaneous or surgical revascularization.

RESULTS Collateral circulation was graded Rentrop 0 in 39%, Rentrop 1 in 36%, Rentrop 2 in 18%, and Rentrop 3 in 7% of the patients. The mortality rate was 8.7%. Reinfarction occurred in 4.7% of the subjects, and repeat coronary revascularization was performed in 10.9% of the patients. These endpoints were not correlated with the degree of collateral circulation. A significant inverse association was observed between the Rentrop score and the infarct-related artery antegrade flow ($P < 0.001$).

CONCLUSIONS The degree of collateral circulation assessed by the Rentrop score during primary PCI is not a useful long-term prognostic factor in the population with STEMI in the current therapeutic approach. This may result from the negative correlation between the Rentrop score and the degree of blood flow in the infarct-related artery. Thus, collateral circulation in a patient with STEMI should not discourage intensive cardiovascular risk factor control in secondary prevention of coronary artery disease.

Correspondence to:
Tomasz Rechciński, MD, PhD, Katedra i Klinika Kardiologii, Uniwersytet Medyczny w Łodzi, Szpital im. Władysława Biegańskiego, ul. Kniaziewicza 1/5, 91-347 Łódź, Poland, phone: +48-42-251-61-12, fax: +48-42-261-60-15, e-mail: tomasz.rechcinski@umed.lodz.pl
Received: September 24, 2012.
Revision accepted: December 7, 2012.
Published online: December 11, 2012.
Conflict of interest: none declared.
Pol Arch Med Wewn. 2013; 123 (1-2): 29-37

INTRODUCTION Coronary collaterals have been the subject of extensive research during the last decades, not only in terms of their prognostic value in acute or chronic ischemic events but also in terms of the biological processes involved in angiogenesis, and even the genetic background. The development of collaterals was shown to be a 2-phase process in patients with acute coronary syndrome.[1] The number and diameter of angiographically detectable collaterals tend to increase with the duration of chronic coronary occlusion after acute myocardial infarction (MI).[1,2] This late recruitment was proved to be associated with better myocardial recovery and improved left ventricular function in patients undergoing late mechanical reperfusion.[3] The presence of collateral blood flow in the acute phase of an ischemic event is a crucial factor for the protection of myocardial viability. Early infarct-related artery collateral flow was confirmed to have beneficial effect on the limitation of infarct size and the recovery of left ventricular function in patients treated with thrombolysis or percutaneous coronary intervention (PCI).[4-7] Other studies, however, showed that the presence of collateral circulation at presentation does not improve long-term survival in patients treated with thrombolysis and primary PCI.[8] The conflicting results of

the above studies derive from different populations varying in morbidity and mortality caused by ischemic heart disease.

The aim of our study was to assess the long-term prognostic value of collateral flow measured by angiography in patients with the acute phase of MI treated with PCI. The Polish population in general, and the population of central Poland in particular, have relatively high total mortality rates, e.g., in 2005, it was 965/100,000 inhabitants. The rate of cardiovascular mortality was 45.7% (441/100,000 inhabitants).[9] In comparison, the rates for the European Community in 2008 were 682/100,000 for total and 272/100,000 for cardiovascular mortality.[10] Despite the substantial progress in the treatment of acute MI in Poland, the achievement of the recommended therapeutic targets, e.g., for dyslipidemia, is not satisfactory.[11,12]

PATIENTS AND METHODS

Study population The study group consisted of 330 consecutive patients with acute ST-segment elevation MI (STEMI) treated with primary angioplasty and hospitalized in our department. Ten patients were lost to follow-up and were excluded from further analysis. The diagnosis of STEMI was established according to the European Society of Cardiology / American College of Cardiology / American Heart Association / World Heart Foundation consensus.[13,14] The study protocol was approved by the local ethics committee. The study complies with the Declaration of Helsinki. An informed consent was obtained from all subjects.

Angiography Coronary angiography was performed via transfemoral approach according to the Seldinger technique. In the case of significant lesions in the femoral arteries, angiography was performed via transradial approach according to the Sones technique. Target lesions were evaluated both visually and with quantitative coronary angiography. After an infarct-related artery was identified, balloon angioplasty and intracoronary stent implantation were performed at the discretion of an operator, according to clinical and angiographic indications.

No less than 3 views for the right coronary artery and no less than 5 views for the left coronary artery were recorded in each patient. An intracoronary infusion of nitroglycerine (Perlinganit, Schwarz Pharma AG, Germany) was used in single contrast administration for both the right and left coronary arteries. Coronary collateral flow to the infarct-related artery was graded on baseline angiograms with the use of a 4-degree qualitative classification by Rentrop and Cohen[15]: 0 – no collateral vessels; 1 – filling of side branches of the occluded artery via collateral channels without visualization of the epicardial segment; 2 – partial filling of the epicardial segment via collateral channels; 3 – complete filling of the epicardial segment of the occluded artery via collateral channels (FIGURE 1). The analysis of collateral circulation was performed retrospectively by 1 researcher (25-year experience in invasive cardiology; 14,000 coronary angiographies; 500 PCI/year). The intraobserver variability was tested in the random sample of 30 patients, reaching 93% agreement for the Rentrop scale and 97% agreement for the TIMI scoring (Thrombolysis in Myocardial Infarction).

The determination of blood flow in the infarct-related artery was performed before and after the procedure according to the angiographic flow criteria of the TIMI study group: 0 – no perfusion; 1 – penetration without perfusion; 2 – partial perfusion; 3 – complete perfusion.[16]

Follow-up Patients were subject to a 2-year clinical follow-up (mean duration, 26 ±12 months; censored at first event). The following clinical events were defined as endpoints: cardiac death, nonfatal reinfarction, and repeat revascularization – percutaneous (PCI) or surgical (coronary artery bypass grafting). A combined endpoint (composite), defined as the occurrence of any endpoint (cardiac death or reinfarction or repeat revascularization) was also analyzed. The follow-up was performed prospectively. Long-term follow-up data were obtained from medical records and by telephone or personal contact with trained medical staff.

Statistical analysis Continuous variables are expressed as mean ± standard deviation, whereas categorical variables are presented as absolute values and percentages. Patients were divided into 4 groups based on the degree of collateral flow. Group differences of categorical variables were analyzed by the χ^2 test with the Yates correction for continuity, while group differences of continuous variables were evaluated by the analysis of variance. Survival rates were calculated by the Kaplan-Meier curve analysis; the curves were summarized with the calculation of the hazard ratio and the 95% confidence interval. The hazard ratio is an approach of the relative risk for the association between collateral circulation and the endpoints. Differences were considered significant at the value of $P < 0.05$ (two-tailed). The MedCalc (Belgium) statistical software version 7.3.0.1 was used to perform all statistical calculations.

RESULTS

Baseline demographic, angiographic, procedural, and clinical data including coronary risk factors and left ventricular ejection fraction evaluated within 2 to 3 days after acute MI are shown in TABLE 1.

In 1.8% of the cases, a transradial approach according to the Sones technique was performed. In 92.2% of the cases treated with a transfemoral approach 6F arterial sheaths were used, less frequently 7F (5%) and 8F (2.8%). The mean prehospital delay was 188 ±108 minutes (range, 20–720 minutes). Sixty-three percent of the patients were admitted within 3 hours from the onset of symptoms, 33% within 6 hours, and 4% within more than 6 hours. Coronary angiography

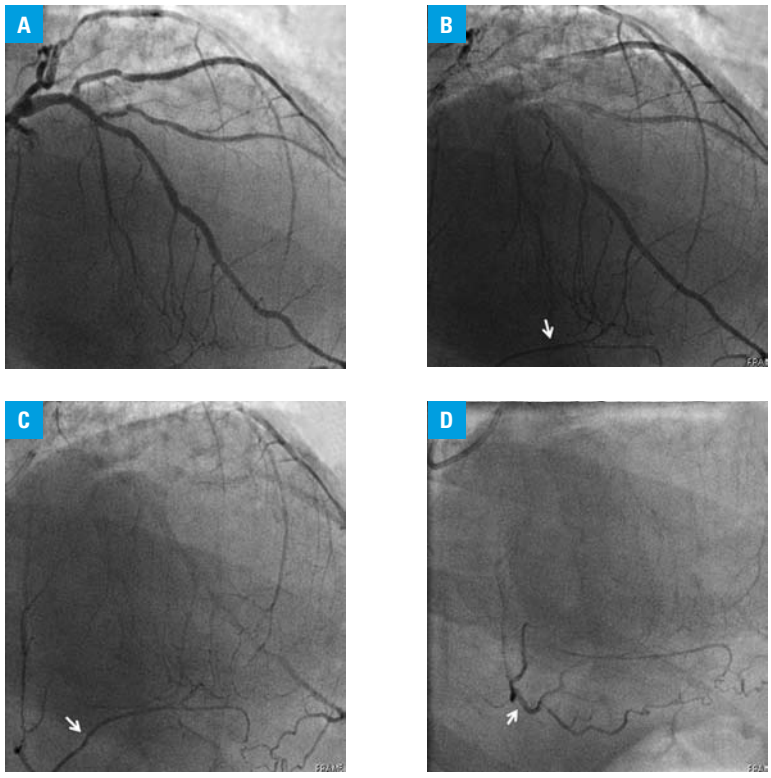

FIGURE 1 Visualization of contrast flow from the left coronary artery (**A**) via collaterals (**B** and **C**) into occluded right coronary artery (**D**) – Rentrop 3

and subsequent angioplasty were performed immediately with the mean door-to-needle time of 37 minutes. Stenting was performed in 314 patients (98.1%), bare-metal stents in 251 (78.4%), and drug-eluting stents in 63 (19.7%). Thrombus in the infarct-related artery was present in 60 cases (18.7%) and aspiration thrombectomy was done in 48 (15.0%). The number of patients with 2- or 3-vessel coronary artery disease was 127 (39.7%) and those with chronic occluded artery – 33 (10.3%).

Among the 320 patients, 126 (39.4%) had no angiographically visible collaterals (Rentrop grade 0), 116 (36.2%) were graded Rentrop 1, 57 (17.8%) Rentrop 2, and 21 (6.6%) Rentrop 3. No statistically significant differences were revealed in the prevalence of risk factors. Similarly, there was no correlation between the extent of collateralization and the time from the onset of symptoms, either expressed in minutes as a continuous variable, or in the usual 3-hour intervals. There was a trend indicating better collateralization in patients with previous MI (χ^2 test for trend, P = 0.1) (TABLE 1).

Angiographically, in 140 patients (43.75%), the right coronary artery (RCA) was recognized as the infarct-related vessel, whereas the left circumflex artery (LCX) and the left anterior descending artery (LAD) were identified as culprit vessels in 44 (13.75%) and 128 (40%) patients, respectively. In 9 patients (2.8%), no definite single vessel responsible for the infarction could be identified. Collateral circulation was developed significantly better in patients with RCA-related infarctions (P <0.001) (TABLE 2).

Before the procedure, in 67.8% of patients (217/320), the infarct-related artery remained totally occluded at the time of angiography (TIMI 0 flow). In 29.0% of the patients (93/320), TIMI 1 was graded, in 1.5% (5/320) TIMI 2, and in 1.5% (5/320) TIMI 3. During PCI, in 88.1% of the patients (282/320) TIMI 3 flow was restored, in 5% (16/320) TIMI 2 flow was achieved, and in 2.2% of the patients (7/320) – TIMI 1. In 4.7% of the cases (15/320), PCI was ineffective (TIMI 0 flow). It cannot be excluded that in some of the cases the presence of chronic total occlusion was responsible for procedural failure. The degree of collateral circulation did not affect PCI results – no statistically significant difference was found between the degree of collateralization and postprocedural

TABLE 1 Baseline characteristics of the patients

	Rentrop 0 (n = 126)	Rentrop 1 (n = 116)	Rentrop 2 (n = 57)	Rentrop 3 (n = 21)	*P*
age, y	59 ±10	58 ±10	56 ±10	57 ±10	0.385
male sex, n (%)	96 (76)	78 (67)	43 (75)	16 (76.2)	0.398
diabetes, n (%)	9 (7)	5 (4.3)	6 (10.5)	1 (4.7)	0.487
hypertension, n (%)	34 (27)	43 (37)	18 (31.6)	5 (23.8)	0.248
smoking, n (%)	37 (29)	39 (34)	19 (33.3)	6 (28.6)	0.839
previous myocardial infarction, n (%)	8 (6)	12 (10)	6 (10.5)	5 (23.8)	0.100
time from symptom onset, min	187 ±125	187 ±100	177 ±94	231 ±107	0.508
serum cholesterol level, mmol/l	5.19 ±1.21	5.56 ±1.24	5.56 ±1.4	5.53 ±1.27	0.224
low-density lipoprotein, mmol/l	3.28 ±1.01	3.64 ±1.09	3.67 ±1.25	3.48 ±1.12	0.126
high-density lipoprotein, mmol/l	1.04 ±0.29	1.04 ±0.29	1.04 ±0.2	1.07 ±0.23	0.994
serum triglyceride level, mmol/l	1.8 ±1.06	1.93 ±1.34	1.71 ±0.84	2.31 ±1.82	0.303
hemoglobin, mmol/l	8.47 ±0.99	8.53 ±1.05	8.47 ±1.17	8.59 ±0.68	0.953
white blood count, 10^9/l	9.84 ±3	10.23 ±3.75	11.03 ±3.4	9.82 ±2.84	0.219
platelet count, 10^9/l	239 ±164	226 ±78	238 ±82	238.4 ±52	0.873
peak creatine kinase myocardial band, U/l	245 ±233	184 ±139	368 ±803	245 ±177	0.440
left ventricular ejection fraction, %	46 ±9.3	46.8 ±9.5	44.4 ±10.9	48.3 ±10.8	0.802

TABLE 2 Distribution of the Rentrop score according to the infarct-related artery

	Rentrop 0	Rentrop 1	Rentrop 2	Rentrop 3	Total	P (χ^2)
any IRA, n (%)	122 (39.2)	114 (36.7)	55 (17.7)	20 (6.4)	311 (100)	
RCA, n (%)	39 (27.8)	51 (36.4)	39 (27.9)	11 (7.9)	140 (100)	<0.001
non-RCA (%)	83 (48.6)	63 (36.8)	16 (9.3)	9 (5.3)	171 (100)	
LAD, n (%)	61 (50)	43 (35.2)	11 (9)	7 (5.8)	122 (100)	0.003
non-LAD, n (%)	61 (32.3)	71 (37.6)	44 (23.3)	13 (6.8)	189 (100)	
Cx, n (%)	22 (44.9)	20 (40.8)	5 (10.2)	2 (4.1)	49 (100)	0.68
non-Cx, n (%)	100 (38.2)	94 (35.9)	50 (19.1)	18 (6.8)	262 (100)	

Abbreviations: Cx – circumflex artery, IRA – infarct-related artery, LAD – left anterior descending artery, RCA – right coronary artery

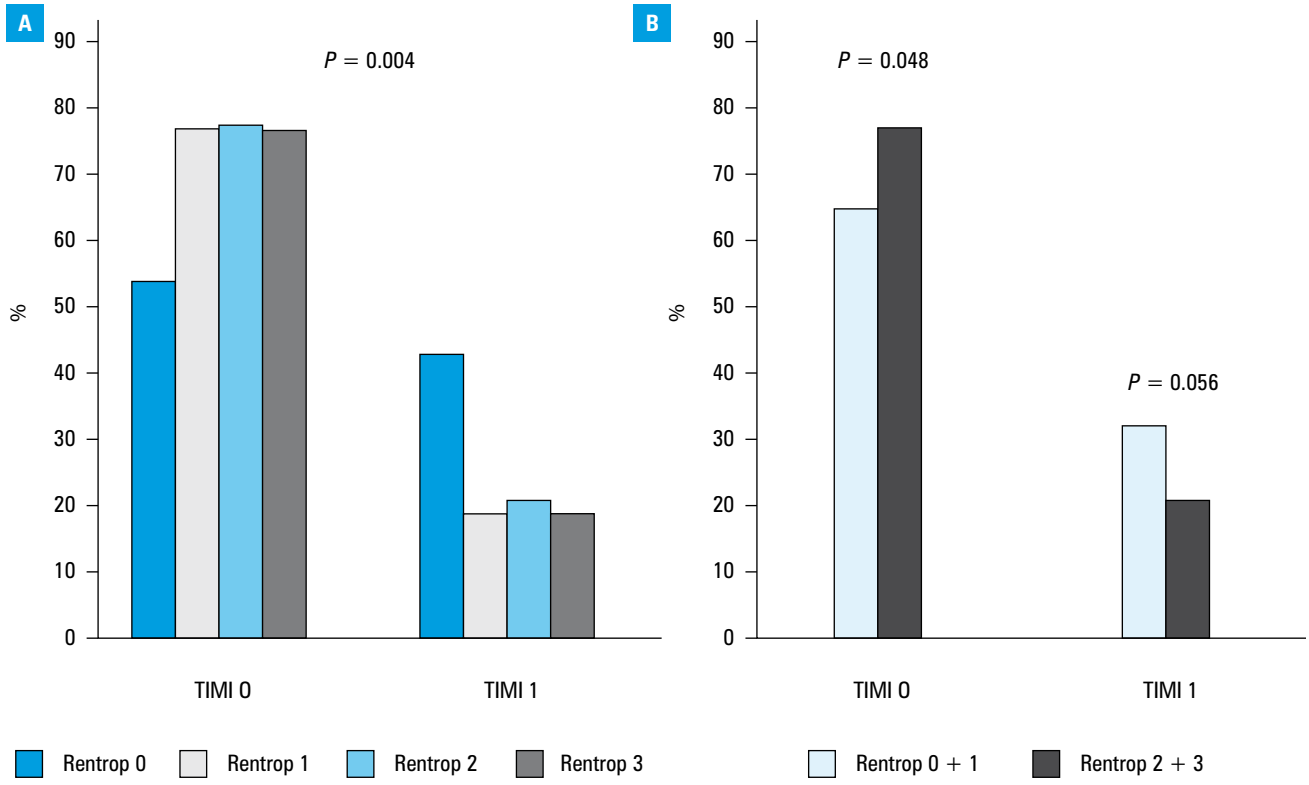

FIGURE 2 Relationship between anterograde (TIMI) and collateral flow degrees (Rentrop scale); distribution of Rentrop 0, 1, 2, and 3 (**A**) and Rentrop 0 + 1 and Rentrop 2 + 3 grades (**B**)

TIMI flow (P = 0.694). The analysis of the relationship between anterograde and collateral flow in the infarct-related arteries showed that higher grades of TIMI flow scores at presentation correlated with weaker collateral circulation. In patients with totally occluded culprit vessels (preprocedural TIMI 0 flow), collaterals were better developed. The distribution of patients with particular grades of the Rentrop score was significantly different between the groups with TIMI 0 and TIMI 1 flow (P = 0.0004, FIGURE 2A). In patients with TIMI 1 flow, collaterals were significantly better developed compared with those with TIMI 1 flow. Better collateralization (Rentrop 2 and 3) was more common than Rentrop 0 and 1 (P = 0.048, FIGURE 2B).

Follow-up data The mortality rate in the follow-up period was 8.7% (28/320). The prevalence of nonfatal reinfarction reached 4.7% (15/320). Repeat coronary revascularization, either percutaneous or surgical, was performed in 10.9% of the patients (35/320). The composite endpoint, defined as the occurrence of any unfavorable outcome: death, reinfarction, or need for another revascularization procedure, was observed in 20.3% (65/320) of the patients.

None of the predefined endpoints correlated with the degree of collateral circulation (TABLE 3). Even the analysis of the results for the 2 subgroups of patients: those with absent (Rentrop 0) or present (Rentrop 1, 2, or 3) collateral circulation did not show any significant correlation with the endpoints, as shown in FIGURE 3.

We then focused on the 242 patients with the totally occluded infarct-related artery (TIMI 0 flow) at the time of angiography. Again, in this subgroup, the extent of collateralization expressed

TABLE 3 Extent of baseline collateralization (Rentrop classification) and incidence of clinical endpoints at the end of the follow-up period

Endpoint	Rentrop 0 (n = 126)	Rentrop 1 (n = 116)	Rentrop 2 (n = 57)	Rentrop 3 (n = 21)	P (χ^2)
death, n (%)	12 (9.5)	9 (7.8)	6 (10.5)	1 (4.8)	0.854
reinfarction, n (%)	3 (2.3)	8 (6.9)	1 (1.8)	3 (14.3)	0.158
repeat revascularization, n (%)	15 (11.9)	13 (11.2)	6 (10.5)	1 (4.8)	0.367
composite[a], n (%)	27 (21.4)	22 (19)	12 (21.1)	4 (19)	0.716
Endpoint	Rentrop 0 + 1 (n = 242)		Rentrop 2 + 3 (n = 78)		P (χ^2)
death, n (%)	21 (8.6)		7 (8.9)		0.936
reinfarction, n (%)	11 (4.5)		4 (5.1)		0.832
repeat revascularization, n (%)	28 (11.6)		7 (8.9)		0.523
composite[a], n (%)	47 (19.4)		16 (20.5)		0.833

a composite endpoint: death or reinfarction or revascularization

with the Rentrop grades did not affect long-term survival (P = 0.904) nor the incidence or other predefined endpoints (P = 0.405 for reinfarction, P = 0.898 for recurrent revascularization, and P = 0.590 for composite endpoint).

DISCUSSION A recently published meta-analysis of about 6500 patients in 12 studies revealed that collateral circulation has a protective effect on mortality in unselected population of patients with ischemic heart disease.[17] Our main finding is the absence of such a relation in the local group of MI survivors.

In the present analysis, the study group was well-balanced in terms of the factors that may affect patients' prognosis after acute MI. The prevalence of classic risk factors and the mean lipid levels or body mass index were comparable with those in other Polish studies.[18-20] There were no sex-related differences in the pharmacological treatment of patients in acute MI (data not shown).[21] No statistically significant relationships were reported between the presence of risk factors and the degree of collateral flow. No correlation was revealed between collateral flow and the presence of diabetes mellitus. This stands in contradiction to numerous previous studies, which showed that collateral circulation was less developed in diabetic patients.[22] However, the absence of such correlation was also noted in the study by Elsman et al.[6] Considering the small percent of diabetic patients in the present study, it has not enough power to resolve the doubts.

Coronary collaterals were significantly better developed in patients with RCA and non-LAD-related infarctions, which is consistent with the previous findings.[6] This may imply better collateralization from the LAD than from the other vessels, possibly due to a larger size of the artery.

Clinical outcomes The beneficial effect of coronary collateral circulation on short-term prognosis in patients with acute MI is well-documented. The presence of well-developed collaterals was proved to limit the size of enzymatically assessed infarct[4,6] and result in faster reperfusion.[8] It was also showed to exert a beneficial effect on myocardial viability after acute MI and on the left ventricular function.[3,4,6] This positive correlation, however, cannot be extrapolated to the long-term analysis. In our study, no correlation was found between collateral flow and infarct size measured with the left ventricular ejection fraction or with the peak values of creatine kinase isoenzyme myocardial band. Moreover, the present study showed no relationship between the degree of collateral circulation and long-term prognosis in patients after acute MI. The results are partly in agreement with the previous studies.[8,23,24] Antoniucci et al.[23] showed that the presence of angiographically detectable collaterals before primary PCI was not a predictor of lower reinfarction, restenosis, or revascularization rate at 6 months. On the other hand, the mortality rate in their study was significantly lower (4% vs. 9%) in patients with pre-PCI collaterals.

Cigarroa et al.[25] reported that anterograde flow in the infarct-related artery improves long-term prognosis after acute MI. In our study, the presence of anterograde flow measured before the procedure according to the TIMI flow scale was negatively correlated with the development of coronary collaterals; the better the collateral, the worse the anterograde flow. This helps explain the results of our study, as the protective roles of collateral and anterograde flow seem to compensate each other – the lack of anterograde flow appears to be balanced by a better collateral supply.

Previous history of MI may serve as an evidence of severity of ischemic heart disease. A positive trend for the development of collateral circulation according to the history of previous MI observed in our study may suggest that severe ischemic heart disease promotes the development of collaterals. Therefore, the potential benefit from the ample development of collateral circulation may be offset by detrimental prognostic consequences of more severe ischemic heart disease. Making a general assumption that collaterals reflect a longer duration of ischemic heart disease, our patients may present a higher-risk population compared with patients assessed in

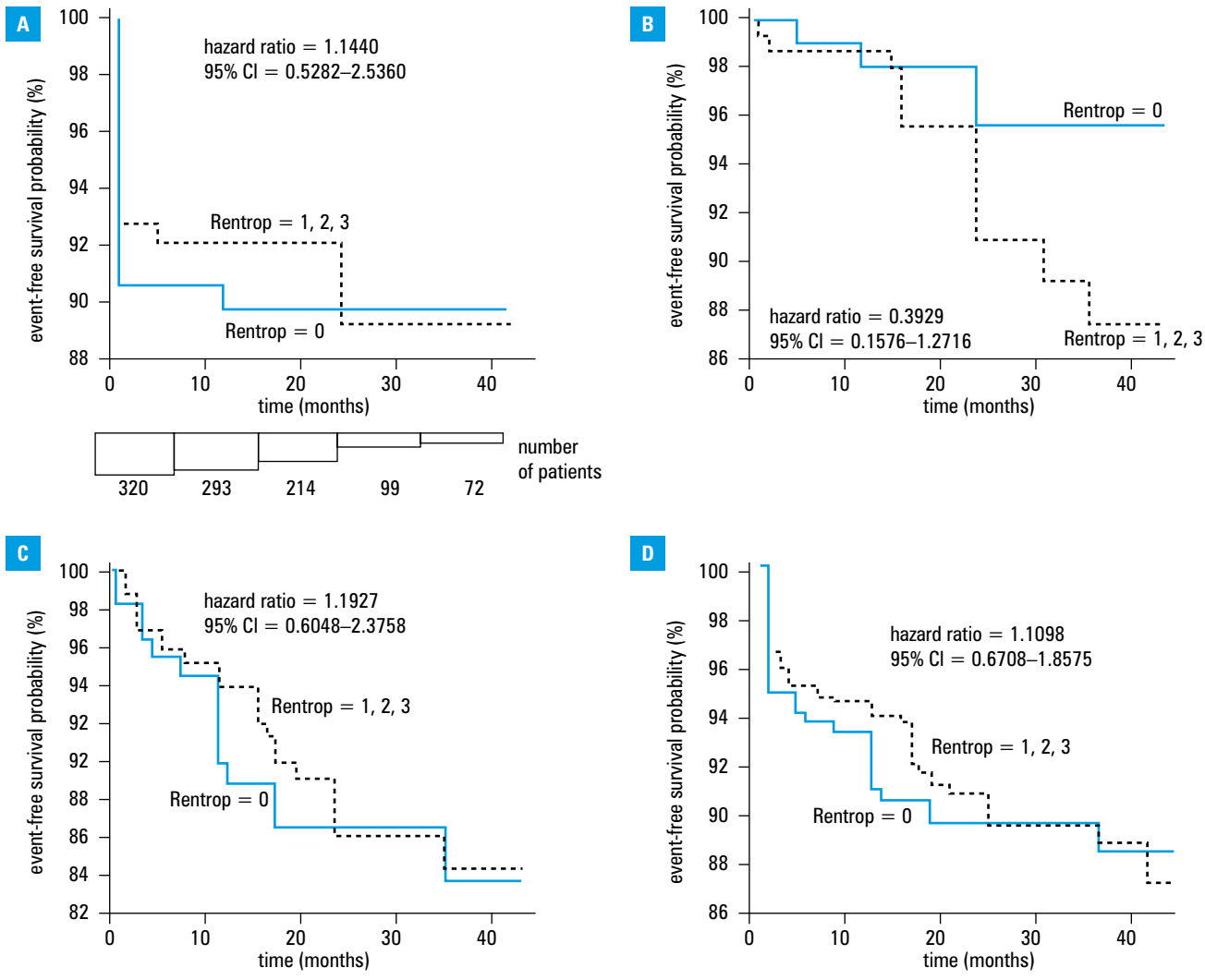

FIGURE 3 Degree of coronary collateral circulation development and incidence of endpoints: **A** – cardiac death, **B** – nonfatal reinfarction, **C** – need for another revascularization procedure, **D** – composite endpoint (occurrence of any event: death or infarction or revascularization)

other studies. For instance, a study by Antoniucci et al.[23] reports a similar rate of collateral circulation (Rentrop >1) in their patients (23% vs. 24% in our study), but the mean age of our patients is about a decade younger. Also in a study by Desch et al.,[26] the mean age of patients with the Rentrop grade 0 or 1 or those with Rentrop 2 or 3 was 8 years higher than in our group. The same researchers have recently concluded that well-developed collaterals have the beneficial effect only in patients with 6-hour or longer symptom-onset-to-reperfusion time. The analysis of our data did not show such a relationship.

Limitations of the study Studies of patients with acute MI evaluating hard clinical endpoints require thousands of patients and long-lasting follow-up. The present study may not have enough power, especially to definitely exclude the impact of coronary collateral circulation on postinfarction prognosis. The lack of the correlation between preprocedural coronary collateral flow levels and long-term outcome may be related to a type 2 error, as all confidence intervals are very wide.

Moreover, a number of clinical variables promoting the development of collateral circulation have been reported. These are mainly duration of angina and use of nitrates.[27,28] Both of them are strong indicators of the severity of ischemic heart disease. However, these factors were not included in this study and their effect on the long-term prognosis could not be measured.

An interplay between the left ventricular mean diastolic pressure, aortic pressure, and distal coronary pressure is a novel factor affecting the efficacy of collateral circulation, not assessed in this study. Meisel et al.[29] proved that during STEMI, high left ventricular mean diastolic pressure (e.g., 40 mmHg) is the most important pressure limiting collateral flow. They used it together with aortic pressure and distal coronary pressure to calculate the acute collateral flow index. Patients with the lowest index, even in the presence of collateral circulation, had significantly higher release of myonecrosis markers during STEMI compared

with those having collateral circulation at STEMI and high acute collateral index.

The dynamics of collaterals recruitment and their effect on microvascular obstruction in our patients were not assessed by imaging techniques during the follow-up period. As demonstrated by Perera et al.,[30] collateral circulation function tends to decrease over a 6-month period after PCI, so that the possible protective role of the circulation might decrease over time. In another study, even though in patients with STEMI early microvascular obstruction quantified by magnetic resonance imaging was alleviated in the group with well-developed collaterals, their presence did not improve clinical outcomes either at 30 days (death, nonfatal reinfarction) or at 6 months.[26] From the practical point of view, a question arises whether the presence of collaterals may influence decision-making during the procedure of PCI. Meier et al.,[31] in the meta-analysis of 7 studies, concluded that the presence of collaterals may help predict restenosis after PCI. Since the presence of collaterals (Rentrop 2 and 3) is associated with increased risk of restenosis, it should be used for risk stratification and for the choice of aggressive antiproliferative therapy, such as drug-eluting stent instead bare-metal stent or cilostazol. In a study by Chen et al.[32] on clinical outcomes after recanalization of chronic totally occluded vessels with bifurcation lesions, it was found that stenting of proximal lesions followed by sudden occlusion of side branch led to an increased rate of periprocedural acute MI in patients without collateral circulation compared with those who had developed collaterals (Rentrop ≥2). Thus, these authors suggest that safety wire placed inside branches with Rentrop <2 should be mandatory to improve the safety of the procedure and to reduce the risk of periprocedural acute MI.[32]

Conclusions Coronary collateral circulation assessed with the angiographic Rentrop scale does not seem to play a protective role in the acute phase of MI and cannot serve as a predictor of better long-term clinical outcome in patients after STEMI treated with primary PCI according to the current standards with aggressive adjunctive pharmacotherapy and medical secondary prevention. Therefore, coronary collateral circulation in a patient should not discourage careful postinfarction monitoring or risk stratification aimed at efficient long-term secondary prevention.

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Obecność krążenia obocznego u pacjentów po zawale serca nie poprawia rokowania odległego

Tomasz Rechciński, Aleksandra Jasińska, Jan Z. Peruga, Jakub Foryś, Maria Krzemińska-Pakuła, Zbigniew Bednarkiewicz, Ewa Trzos, Jarosław D. Kasprzak

Katedra i Klinika Kardiologii, Uniwersytet Medyczny w Łodzi, Szpital im. Biegańskiego, Łódź

SŁOWA KLUCZOWE

krążenie oboczne, rokowanie, zawał serca

STRESZCZENIE

WPROWADZENIE Znaczenie wieńcowego krążenia obocznego w rokowaniu pacjentów po zawale serca pozostaje przedmiotem dyskusji.

CELE Celem badania była ocena wpływu wieńcowego krążenia obocznego ocenianego wg klasyfikacji Rentropa na długoterminowe rokowanie u pacjentów leczonych pierwotną angioplastyką wieńcową z powodu zawału serca z uniesieniem odcinka ST (*ST-elevation myocardial infarction* – STEMI).

PACJENCI I METODY Krążenie oboczne oceniano za pomocą skali Rentropa na podstawie koronarografii u 330 chorych z zawałem serca. Następnie pacjentów obserwowano przez średnio 26 ±12 miesięcy z uwzględnieniem wystąpienia zgonu, ponownych zawałów niezakończonych zgonem oraz ponownej przezskórnej lub chirurgicznej rewaskularyzacji.

WYNIKI Krążenie oboczne oceniono w skali Rentropa na 0 u 39% pacjentów, 1 u 36%, 2 u 18% i 3 u 7%. Wskaźnik śmiertelności wyniósł 8,7%. Ponowny zawał serca wystąpił u 4,7% badanych, a kolejną rewaskularyzację przeprowadzono u 10,9% pacjentów. Wymienione punkty końcowe nie korelowały ze stopniem rozwoju wieńcowego krążenia obocznego. Wyraźną odwrotną zależność stwierdzono natomiast między wynikiem oceny krążenia obocznego w skali Rentropa a napływem do tętnicy odpowiedzialnej za zawał ($p < 0{,}001$).

WNIOSKI Stopień rozwoju wieńcowego krążenia obocznego ocenianego podczas pierwotnej angioplastyki za pomocą skali Rentropa nie jest przydatnym długoterminowym czynnikiem rokowniczym w populacji leczonej z powodu STEMI zgodnie z aktualnymi zasadami terapii. Może to wynikać z ujemnej korelacji między wynikiem oceny w skali Rentropa a stopniem napływu krwi do tętnicy odpowiedzialnej za zawał. Stąd obecność krążenia obocznego u pacjenta ze STEMI nie powinna zniechęcać do intensywnej kontroli czynników ryzyka miażdżycy w ramach wtórnej prewencji choroby wieńcowej.

Adres do korespondencji:
dr med. Tomasz Rechciński, Katedra i Klinika Kardiologii, Uniwersytet Medyczny w Łodzi, Szpital im. Władysława Biegańskiego, ul. Kniaziewicza 1/5, 91-347 Łódź, tel.: 42-251-61-12, fax: 42-251-60-15, e-mail: tomasz.rechcinski@umed.lodz.pl

Praca wpłynęła: 24.09.2012.
Przyjęta do druku: 07.12.2012.
Publikacja *online*: 11.12.2012.
Nie zgłoszono sprzeczności interesów.
Pol Arch Med Wewn. 2013; 123 (1-2): 29-37