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Risk factors of asymptomatic restenosis in patients with first anterior ST elevation myocardial infarction treated with primary percutaneous coronary intervention

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Abstract

Background: The issue of predicting coronary artery restenosis, especially silent, in patients following primary percutaneous coronary intervention (PCI) has been extensively studied, however, risk factors have not been fully defined.

Aim: To asses the frequency of silent restenosis and its predictors in patients with anterior ST elevation myocardial infarction (STEMI) treated with primary PCI and implantation of bare metal stents (BMS).

Methods: We recruited a cohort of 114 patients with first anterior STEMI treated with primary PCI within 12 hours of the onset of symptoms, and with the left anterior descending coronary artery occlusion (TIMI 0) and successful flow restoration (TIMI 3). A 12-lead ECG was performed before and 60 minutes after PCI. Troponin I and CK-MB were measured on admission and after six, 12 and 24 hours. Transthoracic echocardiography (TTE) was performed at discharge. Resting TTE and coronary angiography were performed after a six month follow-up in asymptomatic patients.

Results: The frequency of silent restenosis in our study group was 23.9%. The best multivariate models in logistic regression of restenosis prediction were: lower end-systolic volume of the left ventricle assessed two days after infarction longer lesion and smaller reference diameter of the stented vessel.

Conclusions: Silent restenosis in patients with first anterior STEMI treated by primary PCI with the use of BMS is still frequent. The best ways to identify patients with silent restenosis at six month follow-up, apart from the lower end systolic volume in the echocardiographic study, are longer narrowing in the infarct-related artery and lower reference diameter of the treated vessel.

Key words: STEMI, primary percutaneous coronary intervention, silent restenosis

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INTRODUCTION

Primary percutaneous coronary intervention (PCI) is, according to the European and American guidelines, an optimal therapy for patients with acute coronary syndrome (ACS) [1, 2]. There has been increased interest that there are more and more patients who, despite adequate pharmacotherapy, experience restenosis at the site of intervention [3, 4–6].

Restenosis is a complex, and as yet not fully understood, process. It is believed that restenosis is long-lasting, with variable rates of progression in different individuals, and it usually takes place three to six months after successful PCI.

Animal models have shown a clear correlation between the level of injury to the artery during PCI, and the frequency of restenosis [7]. The use of bare metal stents (BMS) signifi-

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cantly slows the development of unfavourable processes, reducing the risk of such mechanisms of restenosis as elastic renarrowing, dissection or negative remodelling. The introduction of stents has decreased the number of restenoses mainly because the technique increases the diameter of the artery. Even in cases of relatively large lumen loss, the remaining caliber is adequate to relieve symptoms.

Restenosis may be symptomatic with the recurrence or exacerbation of symptoms. Often, however, it is clinically silent, making it more difficult to diagnose, and so delaying therapy. This is why in our study we aimed to determine the frequency of silent restenosis in patients with first anterior myocardial infarction with ST elevation (STEMI) treated successfully with PCI and implantation of BMS. We attempted to identify the predictors of asymptomatic restenosis in the infarct-related artery (IRA) at six month follow-up.

METHODS

Inclusion criteria

Initially, we included in our study all patients hospitalised in our Medical Centre with first anterior STEMI treated with primary PCI within 12 hours of the onset of symptoms, and with the amputation of left anterior descending (TIMI 0) and successful restoration of flow (TIMI 3) followed by stent implantation. Anterior STEMI was defined as chest pain lasting more than 20 minutes and elevation of ST segment ≥ 2 mm in precordial V1–V3 leads, elevation ≥ 1 mm in other precordial leads, and I, aVL leads, or new left bundle branch block. Finally, we included patients who presented no symptoms over a six-month observation and who agreed to control coronary angiography.

Exclusion criteria

The exclusion criteria were: previous MI, hypertrophic cardiomyopathy, implanted pacemaker or implantable cardioverter-defibrillator, significant valvular diseases, women of child-bearing potential, unsatisfactory angiographic effect of PCI, with residual value of more than 30%. The presence of angina at six month follow-up was another exclusion criterion.

Electrocardiography

12-lead electrocardiography (ECG) was performed directly before, and 60 minutes after, the PCI procedure. In the first ECG, the following parameters were analysed: maximum ST elevation in single lead (maximum ST elevation), sum of ST segment elevations in the precordial I, and aVL leads (ΣST) measured 60 ms after QRS complex ending. On the basis of a second ECG, patients were qualified to the group with or without normalisation of ST segment, following the criteria from the TIMI 14 study: reduction of sum of elevations of ST segment by at least 50% (ΣST50%).

All patients after the intervention were monitored for 24 hours using the 12-lead ECG system (DASH 4000, GE). The time to normalisation of ST segment, defined as the reduction of elevation at least 50% from the lead with the highest elevation (ΔtST50%) was analysed. The ΔtST50% < 61 min was taken from ROC curve.

Biochemical studies

The levels of myocardial necrosis enzymes — troponin I, CK-MB — were measured on admission and six, 12 and 24 hours after admission to hospital.

Coronary angiography and primary PCI

The study was performed either by transfemoral or transradial approach. The choice of technique of stent implantation, and the use of GP IIb/IIIa inhibitor was left to the operator. The flow in IRA was assessed in TIMI scale before and after PCI. Collaterals were determined with the Rentrop scale. Localisation and presence of thrombus were noted. The QCA parameters described the longitude and diameter of lesion. Following the optimal PCI, myocardial tissue perfusion was assessed using the Myocardial Blush Grade (MBG) scale. The number of balloon pre-dilations, the used pressure and the number of stents as well as their length and diameter, were recorded. The GP IIb/IIIa inhibitor use was noted. All patients received BMS.

Echocardiographic study

On the day of discharge, a transthoracic echocardiographic (TTE) study was performed. The following parameters were measured: wall motion score index, global function of left ventricular end-diastolic volume (LVEDV), LV end-systolic volume (LVESV), and LV ejection fraction, measured by Simpson method as arithmetic mean of values from two- and four-dimensional projections.

Follow-up

Six months after discharge from hospital, in the group of patients without symptoms of angina, resting TTE and coronary angiography were performed. Restenosis was defined as the presence of a lesion narrowing the lumen of the implanted stent by ≥ 70% (in stent restenosis — ISR) and/or the presence of a similar narrowing 5 mm proximally or distally to the implanted stent (segmental restenosis).

Statistical analysis

Statistica 7.1 was used for statistical analysis. Continuous variables are presented as mean ± standard deviation (SD), and dychotomic variables are presented as numbers and percentages. Normal distribution of data was tested with the Kolmogorow-Smirnow, Lillefors and Shapiro-Wilk tests. The uniformity of variances was tested by Levene, Brown and

Forsyth tests. The comparison of values in groups with normal distribution was done with a Student t-test. For non-normal distribution, non-parametric tests for two groups were performed: Kolmogorow-Smirnow, and U-Mann Whitney. The predictive value of parameters was assessed in univariate logistic regression analysis. Following the analysis of correlation, and after exclusion of dependent variables with logistic function and estimation by Rosenbrock and quasi Newton, we constructed the optimal predictive model for isolating the subpopulation of patients in whom we may expect silent restenosis. Next, its value was assessed by checking sensitivity, specificity, accuracy, and positive and negative predictive values. A p value < 0.05 was significant.

RESULTS

Initially, 114 patients were enrolled to the study. During the six month follow-up, three patients died due to MI, and 19 (17%) presented symptoms of angina.

After performing coronary angiography, three patients were selected for urgent surgical revascularisation and the remaining subjects were qualified for angioplasty. Thus, in the final analysis, we included 92 symptom-free patients, whose characteristics are shown in Table 1.

On the basis of the performed control coronary angiography, the studied population was divided into two groups. The first comprised patients with silent restenosis (SR) — 22 (23.9%) subjects. The second included 70 (76.1%) patients without restenosis (NR). The characteristics of patients with SR and NR are presented in Table 2. Most patients in both groups were male. The NR group was slightly older. The SR population more often (though not statistically significantly) had diabetes, hypertension and smoked cigarettes. We less often noted dyslipidaemia in subjects with SR. In the group of patients with SR, there were lower troponin I levels and the time from the onset of pain to PCI was significantly shorter. The ECG showed differences between the groups in terms of ST segment elevation reduction. Angiographic analysis revealed more frequent use of longer and narrower stents, as well as a larger number of stents, in the patients with SR. Direct stenting was more frequent than stenting with pre-dilatation in this group.

Results of univariate analysis are shown in Table 3. The best multivariate model in logistic regression, predicting SR six months after the event were: lower end-systolic volume of LV (LLVESV) assessed two days after infarction ($p < 0.04$), longer lesion ($p < 0.03$) and smaller reference diameter of stented vessel ($p < 0.01$).

On the basis of multivariate analysis, we managed to construct an equation which may help to select patients with the highest probability of developing SR. This is: Logit (P) = = 5.34 – 0.03*LLVESV + 0.076*longitude of lesion – 2.01*reference diameter of vessel. The constructed multivariate model is characterised by high sensitivity, specificity, positive and negative predictive values and accuracy: 78%, 86%, 59%, 94% and 84%, respectively.

Table 1. Population characteristics

Population (n)	92
Men [%]	77.2
Age [years]	56.1 ± 9.3
Diabetes mellitus [%]	10.9
Hypertension [%]	38.1
Dyslipidaemia [%]	52.2
Smoking [%]	52.2
Angina before infarction [%]	48.9
Time from first pain [min]	240 ± 188
Troponin I maximum [ng/mL]	31.3 ± 19.2
CK-MB maximum [U/L]	261.3 ± 224.8
Creatinine [mg/dL]	1.1 ± 0.3
LVESV [mm^3]	62.7 ± 22.7
LVEDV [mm^3]	104.1 ± 31.8
LVEF [%]	40.6 ± 7.1
WMSI	1.40 ± 0.20
Max. elevation ST in 1 lead [mm]	5.5 ± 2.6
ΣST elevations [mm]	17.3 ± 8.4
ΣST50% [%] YES	55.4
ΔtST50% [%] YES	435 ± 562
Lesion in proximal LAD [%] YES	65.9
Multivessel disease [%]	76.1
Myocardial Blush Grade scale (1–4)	1.76 ± 1.01
Collateral circulation (3–4) [%] YES	6.6
Direct stenting [%]	48.4
Longitude of stented lesion [mm]	19.5 ± 7.44
Number of stents	1.04 ± 0.20
Diameter of stented vessel [mm]	3.20±0.37
Stent diameter [mm]	3.35 ± 0.40
Stent longitude [mm]	22.1 ± 7.69
Beta-blocker [%]	99.4
Statin [%]	98.9
ACE inhibitor [%]	96.7
Acetylsalicylic acid [%]	100
Clopidogrel or ticlopidin [%]	96.7
Abciximab [%]	65.2

CK-MB — creatine kinase MB isoenzyme; LVESV — left ventricular end-systolic volume; LVEDV — left ventricular end-diastolic volume; LVEF — left ventricular ejection fraction; WMSI — wall motion score index; LAD — left anterior descending; ACE — angiotensin converting enzyme

DISCUSSION

The results of many published trials over the last 20 years have indicated that the importance of stress tests in diagnosing SR is low following successful PCI [8]. In our group of 92 patients with anterior STEMI, we showed that the best predictors of SR, besides typical angiographic factors, is LLVESV on the second day of infarction. We believe this simple echocardiographic indicator is clinically important, and

Table 2. Characteristics of patients without restenosis (NR) and with silent restenosis (SR)

Parameter	NR	SR	P
Population (n)	70	22	
Men [%]	77.1	68.2	NS
Age [years]	57.9 ± 10.6	54.6 ± 8.4	NS
Diabetes mellitus [%]	8.6	18.2	NS
Hypertension [%]	35.7	45.5	NS
Dyslipidaemia [%]	55.7	40.9	NS
Smoking [%]	50.0	59.1	NS
Angina before infarction [%]	48.6	50.0	NS
Time from first pain [min]	264 ± 197	162 ± 138	< 0.03
Troponin I maximum [ng/mL]	34.7 ± 17.9	21.6 ± 19.4	< 0.05
CK-MB maximum [U/L]	281.1 ± 201.8	237.5 ± 354.1	NS
Creatinine [mg/dL]	1.04 ± 0.28	0.97 ± 0.19	NS
LVESV [mm^3]	65.6 ± 24.1	53.5 ± 13.9	< 0.01
LVEDV [mm^3]	107.7 ± 33.1	92.6 ± 24.4	NS
LVEF [%]	39.9 ± 7.2	41.4 ± 4.8	NS
WMSI	1.43 ± 0.21	1.31 ± 0.16	< 0.02
Max. elevation ST in 1 lead [mm]	5.6 ± 2.4	5.1 ± 3.4	NS
ΣST elevations [mm]	18.4 ± 9.7	14.9 ± 7.6	NS
ΣST50% [%] YES	55.7	54.5	NS
ΔtST50% [%] YES	47.1	72.7	< 0.03
Lesion in proximal LAD [%] YES	68.1	59.1	NS
Multivessel disease [%]	77.1	72.7	NS
Myocardial Blush Grade scale (1–4)	1.66 ± 1.04	1.96 ± 0.95	NS
Collateral circulation (3–4) [%] YES	2.90	18.2	< 0.03
Direct stenting [%]	28.6	51.4	< 0.03
Longitude of stented lesion [mm]	18.5 ± 5.9	22.7 ± 10.6	< 0.02
Number of stents	1.0 ± 0.1	1.1 ± 0.4	< 0.02
Diameter of stented vessel [mm]	3.3 ± 0.4	3.0 ± 0.3	< 0.02
Stent diameter [mm]	3.4 ± 0.4	3.2 ± 1.1	< 0.02
Stent longitude [mm]	21.1 ± 6.17	25.3 ± 10.9	< 0.02
Beta-blocker [%]	100	98.6	NS
Statin [%]	100	95.5	NS
ACE inhibitor [%]	95.7	100	NS
Acetylsalicylic acid [%]	100	100	NS
Clopidogrel or ticlopidin [%]	98.57	100	NS
Abciximab [%]	64.3	68.2	NS

Abbreviations as in Table 1

may help to identify patients who are at risk although they present no symptoms.

The pathogenesis of restenosis at the site of previous angioplasty has been studied for more than 20 years. It is believed that the percentage of restenosis in the site of angioplasty with the use of BMS remains high (between 10% and 40%), even with intensive pharmacological treatment. The knowledge of potential factors predisposing to restenosis after implantation of a stent may influence the optimisation of pharmacotherapy, as well as indications for implantation, and may indirectly affect the technique of the procedure.

At six month follow-up, the frequency of symptomatic restenosis was 17%. A similar rate was reported in publications concerning stable angina [9–12]. These relatively good results in our study population are probably associated with low frequency of diabetes mellitus and hypertension, as well as young age. A much higher frequency (25–35%) of restenosis was described in the non-STEMI population [9, 13, 14].

Table 3. Predictors of silent restenosis at six month follow-up — univariate analysis

Parameter	Unit odds ratio	–95% confidence interval	+95% confidence interval	P
Time from onset of pain [continuous value]	0.996	0.992	0.999	0.036
Troponin I max [continuous value]	0.965	0.941	0.991	0.01
CK-MB max [continuous value]	0.995	0.990	0.999	0.048
ΔtST50% [YES]	2.99	1.03	8.65	0.04
LVESV 0 [continuous value]	0.972	0.947	0.998	0.04
Collateral circulation 3–4 [YES]	7.44	1.23	45.02	0.03
Flow on the guidewire [YES]	3.97	1.05	14.9	0.04
Direct stenting [YES]	3.0	1.1	8.15	0.02
Longitude of lesion [continuous value]	1.07	1.005	1.114	0.03
Number of stents [continuous value]	10.89	1.04	114.46	0.04
Reference value [continuous value]	0.14	0.029	0.695	0.02
Diameter of stents [continuous value]	0.17	0.04	0.71	0.02

Abbreviations as in Table 1

The results of clinical research in which control angiography was performed six months after PCI have shown that angiographic restenosis is more frequent than symptomatic restenosis [15, 16]. Analysis of the results of these studies has revealed that more than 50% of patients with restenosis have no angina symptoms six months after PCI. We believe this group should be carefully supervised, especially in the light of the SWISSI II trial. The study concluded that prognosis in patients with asymptomatic restenosis after infarction is much better in the group treated interventionally than in the group treated conservatively [17].

The meta-analysis of studies by Ruygrok et al. [18] which included ten trials of patients after acute MI, showed the occurrence of SR only in 12% of patients at six month angiography, less often than in our population. On the other hand, the SOPHOS study included patients with ACS but with only one critical lesion [15].

In our trial, many predictors of SR in univariate analysis are the same as predictors of symptomatic restenosis already reported in literature. Angiographic indicators, such as longer narrowing of the IRA vessel, smaller diameter of the stent or reference vessel, or higher numbers of implanted stents are known indicators of symptomatic restenosis [10, 19, 20]. Surprisingly, the results of our trial did not confirm that diabetes or hypertension play a role. In our population, diabetes and hypertension were less frequent compared to other trials in literature, which was probably due to the selected population I (first STEMI, relatively young).

We also did not confirm the role of renal insufficiency, another widely known indicator of restenosis [21]. Renal insufficiency was not an exclusion criterion, but creatinine levels in both groups were within reference values, also probably as the result of young age and few cases of diabetes mellitus.

An interesting new observation is that well functioning collateral circulation, classified according to the Rentrop scale, may predict SR. We believe that this represents typical restenosis, and lack of stenocardia is due to well developed collaterals.

The best multivariate model for predicting SR consisted of longer amputated vessel, smaller diameter of reference vessel, and LLVESV [17, 18]. Lower values of troponin mean less damage to the myocardium, and potentially better supply of blood to the coronary vessel — media and intima — via the vasa vasorum [22–24]. This may result in less damage to the wall of the vessel and to local rennin–angiotensin–aldosteron complexes, and increased proliferation of smooth muscle cells. Paradoxically, this means pathologic healing and restenosis. The role of the vasa vasorum in the pathological process of atherothrombosis is not yet fully understood. Those vessels originate in response to a reactive stimulus. This process is thought to be the source of disease by influencing the function of the endothelium and opening the 'gate' for the migration of monocytes. This leads to an inflammatory reaction, and as a consequence to the development of restenosis [24].

To the best of our knowledge, no study has indicated such a relationship in symptomatic restenosis. That is why the role of LLVESV in the acute phase of STEMI should undergo further investigation.

Limitations of the study

The limitation of the study is the use of BMS only. Nowadays, widely used drug-coated stents, also in ACS, mainly non-STEMI, seem to show a lower rate of restenosis.

The results of recent trials showed a significant reduction in repeat coronary intervention if a drug-eluting stent is used

in STEMI, although not reducing the risk of thrombosis, repeated infarction or death [25, 26]. The newest ESC guidelines for STEMI advise further trials, with long observations, concerning the safety of using drug-eluting stents in STEMI [2].

Because of the uniform population of patients confined to first STEMI of anterior wall, the studied group consisted of young patients (mean age 56.1 ± 9.3). Few patients were aged over 75, a group which normally has more complex lesions in vessels as well as more significant concomitant diseases, something which may determine the frequency of symptomatic and asymptomatic restenosis.

CONCLUSIONS

The frequency of silent restenosis in patients with first anterior wall STEMI treated by primary PCI with the use of BMS is still high, reaching 23.9%. The best indicators identifying patients with SR at six month follow-up after PCI, except for LLVESV in the echocardiographic study, were longer narrowing in the IRA and a lower reference diameter of the treated vessel. Because of the hidden process of unfavourable remodelling and the potential danger of another ischaemic event, it is important to identify patients predisposed to remodelling as early as possible. The model presented in our trial may be of use.

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Czynniki ryzyka rozwoju bezobjawowej restenozy u chorych z zawałem serca z uniesieniem odcinka ST ściany przedniej leczonych pierwotną angioplastyką wieńcową

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Streszczenie

Wstęp: Ze względu na fakt, że wciąż zwiększa się populacja pacjentów z nawrotem zwężenia po pierwotnej angioplastyce wieńcowej (PCI), mimo adekwatnej farmakoterapii, problem określenia czynników ryzyka restenozy po skutecznej pierwotnej PCI jest w ostatnim czasie intensywnie badany. Nawrót zwężenia może powodować objawy kliniczne, jednak u wielu pacjentów jest bezobjawowy, co znacznie utrudnia diagnostykę i opóźnia wdrożenie właściwego leczenia.

Cel: W niniejszej pracy w 6-miesięcznej obserwacji oceniano częstość i czynniki ryzyka wystąpienia bezobjawowej restenozy u chorych z pierwszym w życiu zawałem z uniesieniem odcinka ST (STEMI) ściany przedniej, leczonych pierwotną PCI z implantacją stentów nieuwalniających leku (BMS).

Metody: Do badania włączono 114 osób z pierwszym w życiu STEMI ściany przedniej, z zamknięciem tętnicy przedniej zstępującej (TIMI 0), leczonych pierwotną PCI w ciągu 12 godzin od początku objawów, skutecznym udrożnieniu tętnicy (TIMI 3) i implantacją BMS. Badanie EKG wykonywano przed i 60 minut po PCI, wartości troponiny I i CK-MB oznaczano przy przyjęciu, a następnie po 6, 12 i 24 godzinach. Przezklatkowe badanie echokardiograficzne wykonano 2 dni po zawale — przed wypisem ze szpitala. Po 6-miesięcznej obserwacji u pacjentów bez objawów klinicznych ponownie wykonano spoczynkowe przezklatkowe badanie echokardiograficzne i angiografię tętnic wieńcowych.

Wyniki: Częstość występowania bezobjawowej restenozy w badanej grupie pacjentów wynosiła 23,9%. W analizie wielo-czynnikowej metodą logistycznej regresji najlepiej przewidywały nawrót zwężenia w tętnicy dozawałowej (IRA) — niska końcowoskurczowa objętość lewej komory oceniana 2 dni po zawale, dłuższa blaszka miażdżycowa i mniejsza średnica IRA.

Wnioski: Częstość występowania bezobjawowego nawrotu zwężenia u pacjentów po pierwszym w życiu STEMI ściany przedniej leczonych skuteczną PCI z użyciem BMS jest wciąż wysoka. Najlepszymi czynnikami predykcyjnymi identyfikującymi chorych z nawrotem zwężenia, poza niską objętością końcowoskurczową lewej komory w badaniu echokardiograficznym, jest dłuższe zwężenie i mała średnica IRA.

Słowa kluczowe: zawał ściany przedniej, pierwotna angioplastyka wieńcowa, bezobjawowa restenoza

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