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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Patients with acute myocardial infarction and severe target lesion calcifications undergoing percutaneous coronary intervention have poor long-term prognosis

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Abstract

Background: To assess the influence of severe target lesion calcification (TLC) on the outcomes of patients undergoing percutaneous coronary interventions (PCI) due to acute myocardial infarction (AMI).

Aim: Contemporary data concerning coronary artery calcifications (CAC) are based on pooled analyses from randomised trials with short follow-up. We still lack the knowledge on how CAC in target lesions affect long-term prognosis of patients with AMI in everyday practice.

Methods: We evaluated clinical and laboratory data of 206 consecutive patients who underwent coronary angiography and PCI due to AMI. Primary endpoints were all-cause death and recurrent hospitalisations due to acute coronary syndrome (ACS).

Results: Severe TLC lesions were present in 17% of patients. These patients were older (71 vs. 65 years, $p = 0.02$) and more often diagnosed with non-ST segment elevation myocardial infarction (77% vs. 58%, $p = 0.03$). Patients with severe TLC had lower rates of PCI success (80% vs. 97%, $p < 0.0001$) and less often achieved full revascularisation during index procedure (14% vs. 41%, $p = 0.003$). During 30 months follow-up patients with severe TLC more often suffered from another ACS (37% vs. 13%, $p = 0.0005$) and had higher all-cause mortality (31% vs. 16%, $p = 0.04$). Multivariate Cox regression model showed severe TLC to be an independent predictor of another ACS (HR 2.8; 95% CI 1.4–5.6; $p = 0.004$).

Conclusions: Severe TLC are not uncommon in patients with ACS. The presence of severe TLC is a prognostic factor of another ACS in AMI patients undergoing PCI.

Key words: myocardial infarction, percutaneous coronary intervention, calcification, revascularisation

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INTRODUCTION

Coronary artery calcifications (CAC) are a known predictive factor of poor prognosis in patients undergoing percutaneous coronary intervention (PCI) with either bare metal (BMS) or drug eluting stent (DES) implantation [1–3]. Recent studies showed unfavourable outcomes also in patients with target lesion calcifications (TLC) undergoing PCI due to acute myocardial infarction (AMI) [4]. TLC are also connected with higher rates of PCI failure [5, 6]. Aging and an increasing number of comorbidities in patients undergoing PCI results in a growing number of challenges during PCI, including dealing with severe TLC, even in low-volume centres. Contemporary data concerning the impact of TLC on the prognosis of patients undergoing PCI due to AMI is based on pooled analyses from large randomised trials where follow-up data was limited to one year observation [4]. Moreover, most clinical trials excluded patients with severe TLC. We still lack the knowledge on how severe TLC affects long-term prognosis of patients with AMI undergoing PCI in everyday practice. This study was created to assess the frequency and influence of severe TLC

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on adverse events in patients undergoing PCI due to AMI in a "real-life" scenario.

METHODS

Study design

This was a single-centre prospective observational study. The study protocol was accepted by the local Ethics Committee and was in accordance with the declaration of Helsinki. All patients gave written, informed consent for participation in the study.

Study population

In our study, we included all consecutive patients with the diagnosis of AMI, who underwent coronary angiography and PCI in our centre from May to November 2012. The only exclusion criterion was the lack of written consent from the patient. The definition of AMI was established in accordance with the third universal definition of myocardial infarction (MI) proposed by the European Society of Cardiology [7].

Treatment

In-hospital treatment was conducted according to current standards and was left to the discretion of the physicians in charge of the patients.

Angiographic assessment

Each coronary angiography was separately evaluated by two experienced interventionalists who assessed the presence and extent of CAC. In the case of discordant results the opinion of a third senior operator was definitive. Physicians assessing coronary angiographies were blinded to treatment results and follow-up.

Calcification classification

The most common angiographic classification divides patients with CAC into three groups according to the extent of visible calcium: no/mild calcifications (not visible on coronary angiography), moderate calcifications (radiopaque densities visible during heart motion and affecting one side of the vessel), and severe calcification (densities visible without heart motion and affecting both sides of the vessel) [8]. There is still no consensus regarding the cut-off value for clinically important calcium concentration. Due to the relatively small population the authors decided to divide the patients into two groups only. We decided to focus on patients with severe calcifications because they should be most prone to further adverse events and require different treatment approach. Moreover, the definition of severe calcifications in angiography is the most consistent and least prone to underestimation.

Endpoint definitions

Primary endpoints were all-cause mortality and recurrent hospitalisations due to ACS. Predetermined combined secondary endpoint was the composite of all-cause death and recurrent hospitalisation due to ACS or stroke.

Follow-up

Follow-up data regarding all-cause mortality and recurrent hospitalisations was obtained from the Polish National Health Found database, so no patient was lost to follow up. All patients were observed for 30 months.

Statistical analysis

Continuous variables with normal distribution are presented as mean ± standard deviation. Continuous variables with skewed distribution are presented as median with interquartile range. Categorical variables are presented as numbers and percentages. For continuous variables, intergroup differences were compared using Student's t test or the Mann-Whitney U test, depending on the type of distribution. The χ^2 test was used to compare categorical variables. A p-value < 0.05 was considered statistically significant. Univariate and multivariate Cox proportional hazard models were used to determine the predicting factors of all-cause death, recurrent hospitalisation due to ACS, and composite secondary endpoint. The multivariate model included all variables with $p < 0.05$ in the univariate model. Survival and event-free survival curves were created using the Kaplan-Meier method. Differences in survival and event-free survival rates were compared using the log-rank test. All statistical analyses were performed using Statistica 10.0 (StatSoft, USA) software.

RESULTS

Clinical, demographic, and laboratory characteristics

During the studied period 336 patients were hospitalised in our institution due to MI. Forty patients refused participation in our study or were not able to give informed consent. Of the remaining 296 patients 206 underwent PCI and were included in our study. Severe TLC were present in 35 (17%) patients. Patients with severe TLC were older (71 vs. 65 years, $p = 0.02$) and less often diagnosed with ST-segment elevation MI (STEMI). Higher levels of N-terminal prohormone of B-type natriuretic peptide at admission were also present in these patients (2006 vs. 745 pg/mL, $p = 0.01$). There were no differences in other clinical, demographic, and laboratory characteristics. Complete demographics, comorbidities, and laboratory results are presented in Table 1.

Procedure characteristics

Procedure characteristics are summarised in Table 2. Patients with severe TLC less often underwent complete revascularisation during index procedure (14% vs. 41%, $p = 0.003$). Some of the patients who did not undergo full revascularisation during index hospitalisation were later scheduled for planed procedure. Qualification for further treatment was

Table 1. Baseline clinical and laboratory characteristics

Parameter	All patients	No/mild/moderate TLC	Severe TLC	p
Number	206	171 (83%)	35 (17%)	
Age [years]	66 (60–77)	65 (58–77)	71 (64–79)	0.02
Male	133 (65%)	109 (64%)	24 (69%)	0.59
STEMI	80 (39%)	72 (42%)	8 (23%)	0.03
Diabetes	51 (25%)	38 (22%)	13 (37%)	0.06
Hypertension	169 (82%)	137 (80%)	32 (91%)	0.11
Hyperlipidaemia	155 (75%)	127 (74%)	28 (80%)	0.47
Current smoker	56 (27%)	51 (30%)	5 (14%)	0.09
HFREF	22 (11%)	19 (11%)	3 (9%)	0.66
Previous MI	72 (35%)	57 (33%)	15 (43%)	0.28
Previous stroke/TIA	20 (10%)	14 (8%)	6 (17%)	0.1
Previous diagnosis of renal insufficiency	38 (18%)	30 (18%)	8 (23%)	0.46
Cancer	27 (13%)	23 (13%)	4 (11%)	0.75
LVEF [%]	50 (40–55)	50 (40–55)	45 (40–50)	0.07
Baseline TnI [ng/mL]	1.44 (0.2–7.05)	1.5 (0.22–7.4)	1.08 (0.18–4.43)	0.56
Maximal TnI [ng/mL]	9.38 (1.39–34.95)	9.46 (1.8–44.6)	9.28 (0.78–22.1)	0.22
WBC [$10^3/\mu L$]	9.1 (7.2–11.1)	9.2 (7.4–11.2)	8.4 (6.4–10.6)	0.14
Haemoglobin [g/dL]	13.6 ± 1.7	13.6 ± 1.7	13.3 ± 1.8	0.65
Platelet count [$10^3/\mu L$]	220 (184–253)	218 (186–253)	227 (179–259)	0.43
eGFR [mL/min/1.73 m^2]	75 ± 26.1	77 ± 26	71 ± 25	0.22
NT-proBNP [pg/mL]	941 (263–2870)	745 (234–2477)	2006 (485–5316)	0.01
Hs-CRP [ng/mL]	7.6 (3.0–11.1)	7.6 (2.9–11.1)	8.1 (3.7–11.1)	0.62

Data are presented as numbers and percentages for categorical variables, mean ± standard deviation for continuous variables with normal distribution, and median with interquartile range for continuous variables with skewed distribution. STEMI — ST segment elevation myocardial infarction; HFREF — heart failure with reduced ejection fraction; MI — myocardial infarction; TIA — transient ischaemic attack; TLC — target lesion calcification; LVEF — left ventricular ejection fraction; TnI — troponin I; WBC — white blood count; eGFR — estimated glomerular filtration rate; NT-proBNP — N-terminal prohormone of B-type natriuretic peptide; hs-CRP — high-sensitivity C reactive protein

Table 2. Procedure characteristics

Parameter	All patients	No/mild/moderate TLC	Severe TLC	p
Stents implanted	1.04	1.06	0.94	0.22
Drug eluting stent	60 (29%)	50 (29%)	10 (29%)	0.94
Bare metal stent	123 (60%)	105 (61%)	18 (51%)	0.27
POBA only	23 (11%)	16 (9%)	7 (20%)	0.07
Complete revascularisation (index procedure)	75 (36%)	70 (41%)	5 (14%)	0.003
Complete revascularisation (all procedures)	118 (57%)	106 (62%)	12 (34%)	0.003
Procedural success	194 (94%)	166 (97%)	28 (80%)	< 0.0001
Radial approach	187 (90%)	156 (91%)	31 (89%)	0.62
Manual thrombectomy	73 (35%)	65 (38%)	8 (23%)	0.09
Rotational atherectomy	1 (0.4%)	0 (0%)	1 (3%)	0.38
Infarct-related artery:				
LMCA	1 (0.4%)	1 (0.6%)	0 (0%)	0.65
LAD	76 (37%)	54 (32%)	22 (63%)	0.0005
Circumflex artery	19 (9%)	19 (11%)	0 (0%)	0.08
Right coronary artery	67 (33%)	60 (35%)	7 (20%)	0.08
Graft	6 (3%)	6 (4%)	0 (0%)	0.26
Other	37 (18%)	31 (18%)	6 (17%)	0.89

Data are presented as numbers and percentages. POBA — plain old balloon angioplasty; LMCA — left main coronary artery; LAD — left anterior descending artery; TLC — target lesion calcification

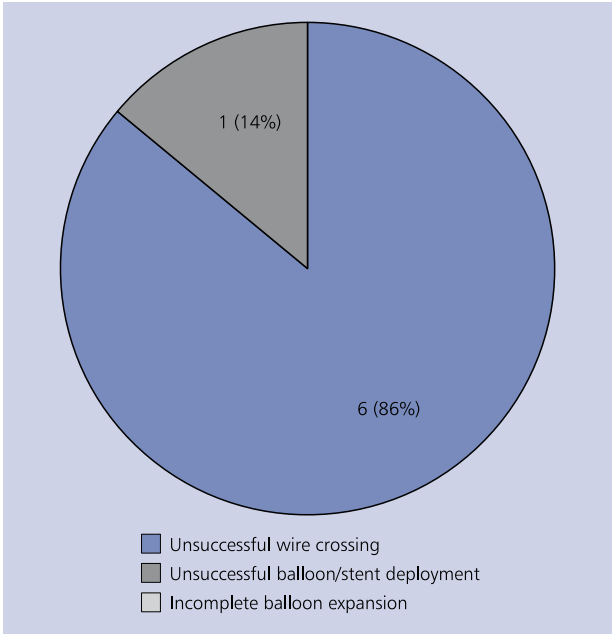

Figure 1. Causes of percutaneous coronary intervention failure in patients with severe target lesion calcification

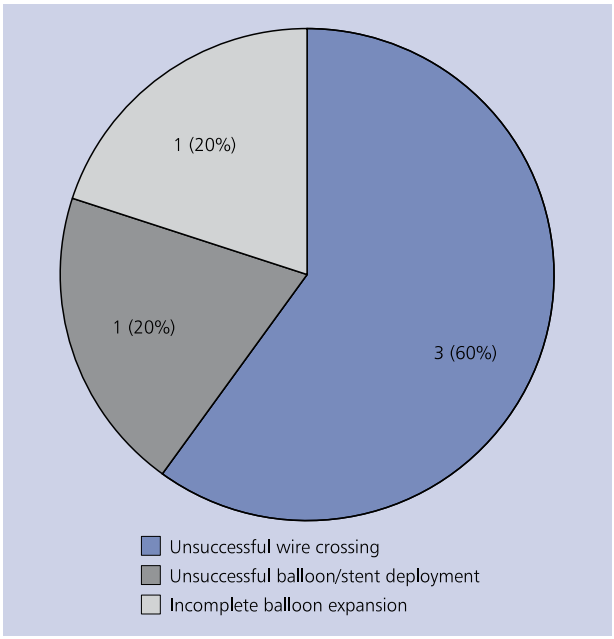

Figure 2. Causes of percutaneous coronary intervention failure in patients without severe target lesion calcification

at the discretion of the physician in charge of the patient at time of hospital discharge. The percentage of complete revascularisation after all planed procedures remained lower in patients with severe TLC (34% vs. 62%, p = 0.003). The PCI success rate was lower in patients with severe TLC (80% vs. 97%, p < 0.0001). Causes of PCI failure in both groups are presented in Figures 1 and 2. The most common cause of PCI failure was unsuccessful wire crossing. Treatment and target lesion selection was at the operator's discretion; therefore, the authors cannot exclude the possibility of erroneous classification of chronic total occlusion as the infarct related artery. In patients with severe TLC left anterior descending artery was more often the culprit lesion (63% vs. 32%, p = 0.0005). Use of rotational atherectomy (RA) was at the operator's decision. Because RA is contraindicated in thrombus-containing lesions its use in MI patients is limited. In our population only one patient with severe TLC underwent RA. No other plaque modifying techniques (cutting balloon etc.) were used. There were no differences in the remaining procedural characteristics.

Long-term follow-up

The results of one-year and long-term clinical observation are presented in Table 3. There was no difference in all-cause mortality between the groups after 12 months (17% vs. 11%, p = 0.32); however, after longer follow-up the mortality in patients with severe TLC was higher (31% vs. 16%, p = 0.04). Kaplan-Meier survival curves are presented in Figure 3. Recurrent hospitalisation due to ACS occurred more often in patients with severe TLC after 12 months (26% vs. 8%, p = 0.003) as well as after 30 months (37% vs. 13%, p = 0.0005). Kaplan-Meier event-free survival curves are presented in Figure 4. Pre-defined combined secondary endpoint was also more frequent in patients with severe TLC (37% vs. 19%, p = 0.02 after 12 months and 60% vs. 28%, p = 0.0003 after 30 months). Rates of recurrent hospitalisations due to stroke were very low and similar in both groups (0% vs. 2%, p = 0.43 after 12 months and 6% vs. 3%, p = 0.4 after 30 months).

Predictors of adverse events in long-term follow-up

Table 4 presents results of univariate and multivariate Cox regression models showing predicting factors of death, recurrent hospitalisation due to ACS, and pre-defined composite end point. Severe TLC (HR 2,8; 95% CI 1.4–5.6; p = 0.004) and age (HR 1.21; CI 1.03–1.44; p = 0.02) were the only independent predicting factors of recurrent hospitalisation due to ACS. Severe TLC were also one of the predicting factors of composite end point (HR 1.7; CI 1.0–2.94, p = 0.05) in 30 months of observation. Independent predictors of all-cause death were age (HR 1.29; 1.07–1.55; p = 0.009) and left ventricular ejection fraction (HR 0.95; CI 0.92–0.98; p = 0.003).

DISCUSSION

Previous studies showed unfavourable prognosis in patients with CAC in short term; however, most of these reports were meta-analyses or pooled analyses from randomised trials with one-year follow-up [3, 4, 9]. The present study is the first, to our knowledge, examining clinical outcomes of patients with

Table 3. Adverse events during one-year and long-term follow-up

Parameter	All patients	No/mild TLC	Severe TLC	p
12 months				
All-cause death	25 (12%)	19 (11%)	6 (17%)	0.32
ACS	23 (11%)	14 (8%)	9 (26%)	0.003
Stroke	3 (1%)	3 (2%)	0 (0%)	0.99
Combined endpoint (stroke, all-cause death, ACS)	46 (22%)	33 (19%)	13 (37%)	0.02
30 months				
All cause death	39 (19%)	28 (16%)	11 (31%)	0.04
ACS	35 (17%)	22 (13%)	13 (37%)	0.0005
Stroke	7 (3%)	5 (3%)	2 (6%)	0.4
Combined endpoint (stroke, all-cause death, ACS)	69 (33%)	48 (28%)	21 (60%)	0.0003

Data are presented as numbers and percentages. ACS — acute coronary syndrome; TLC — target lesion calcification

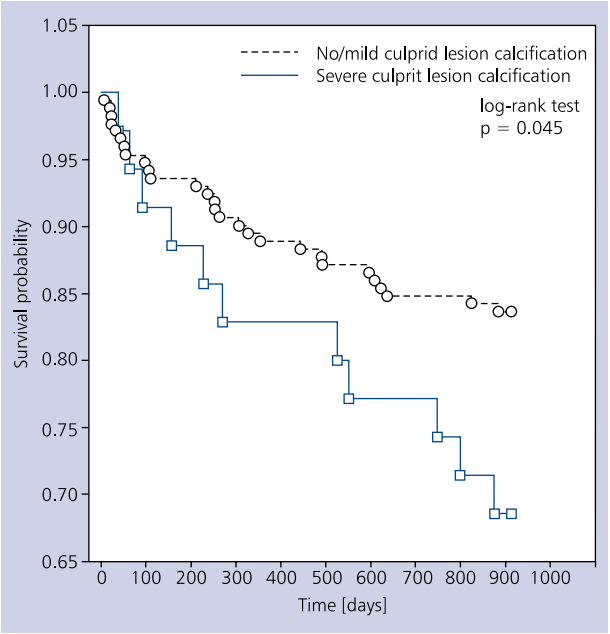

Figure 3. Kaplan-Meier survival curves

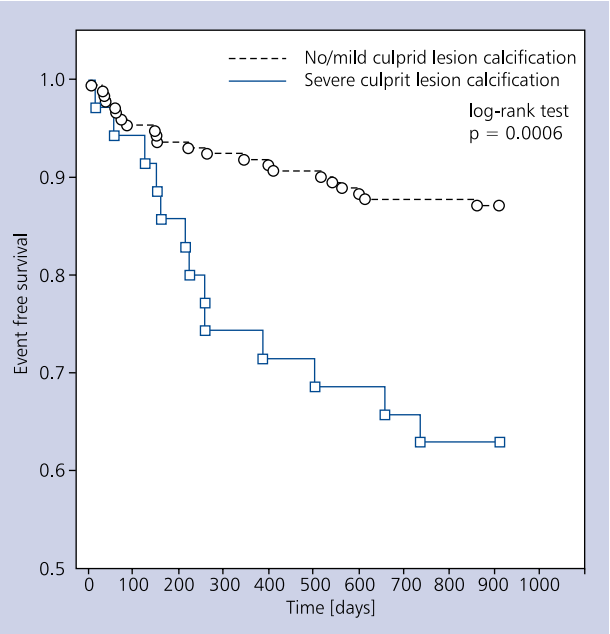

Figure 4. Kaplan-Meier event-free (acute coronary syndrome-free) survival curves

severe CAC undergoing PCI due to MI in long-term follow-up. The number of adverse events in our population is markedly higher than in other reports with all-cause mortality, reaching 31% after 30 months in patients with severe TLC. In our opinion, high rates of major adverse cardiac events (MACE) may be attributed to: singling out only patients with severe TLC in angiography, who are most prone to adverse events and, unlike in clinical trials, including all consecutive patients. High rates of MACE cannot be easily contributed to frequent BMS implantation in our study because univariate and multivariate Cox regression analyses showed no decrease in any of the analysed endpoints after DES implantation.

The prevalence of CAC, visualised in computed tomography, in the general population reaches over 90% in men and 67% in women above 70 years of age [10]. The reported frequency of severe CAC in patients undergoing PCI due to stable coronary artery disease (CAD) and ACS differs between publications and ranges from 5.9% to 20% [4, 9]. In our study 17% of patients had severe CAC in target lesions, which is comparable with reports regarding patients undergoing PCI due to stable CAD, and rather high for patients with ACS. Some reports showed differences in the prevalence of CAC depending on race and nationality [11]. Earlier publications showed that predicting factors of moderate/severe CAC in

Table 4. Univariate and multivariate Cox regression models

	Cox proportional hazard models					
	Univariate models			Multivariate model		
	HR	95% CI	p	HR	95% CI	p
Predictors of recurrent ACS hospitalisation						
Age (every 5 years)	1.25	1.06–1.47	0.007	1.21	1.03–1.44	0.02
Severe TLC	3.29	1.65–6.53	0.0007	2.8	1.4–5.6	0.004
Previous MI	2.19	1.13–4.25	0.02	1.89	0.97–3.69	0.06
Hypertension	3.93	0.94–16.4	0.06	–	–	–
DES implantation	1.45	0.73–2.88	0.29	–	–	–
Full revascularisation	0.48	0.22–1.07	0.07	–	–	–
TnI maximal level	0.87	0.7–1.07	0.18	–	–	–
Procedural success	0.4	0.14–1.15	0.08	–	–	–
STEMI	0.52	0.25–1.19	0.09	–	–	–
Predictors of all-cause death						
Age (every 5 years)	1.35	1.15–1.59	0.0002	1.29	1.07–1.55	0.009
LVEF	0.96	0.93–0.98	0.002	0.95	0.92–0.98	0.003
Severe TLC	2.03	1.01–4.08	0.05	1.15	0.56–2.39	0.7
Full revascularisation	0.29	0.12–0.68	0.005	0.48	0.19–1.2	0.12
Procedural success	0.28	0.12–0.67	0.004	0.65	0.26–1.63	0.36
Current smoker	0.28	0.09–0.79	0.02	0.64	0.2–2.02	0.45
Diabetes	2.1	1.1–3.99	0.02	1.5	0.77–2.92	0.23
TnI maximal level	0.95	0.78–1.14	0.56	–	–	–
STEMI	0.51	0.25–1.04	0.07	–	–	–
Hypertension	2.04	0.73–5.76	0.18	–	–	–
Predictors of combined secondary endpoint						
Age (every 5 years)	1.28	1.13–1.43	< 0.0001	1.25	1.1–1.42	0.0004
LVEF	0.96	0.94–0.98	0.0006	0.96	0.94–0.99	0.002
Severe TLC	2.53	1.51–4.23	0.0004	1.7	1.0–2.94	0.05
STEMI	0.49	0.29–0.85	0.01	0.77	0.43–1.39	0.39
Full revascularisation	0.42	0.24–0.74	0.003	0.69	0.37–1.28	0.24
Procedural success	0.29	0.14–0.59	0.0006	0.67	0.32–1.43	0.3
Previous MI	1.54	0.96–2.48	0.07	–	–	–
Diabetes	1.47	0.88–2.45	0.14	–	–	–
Hypertension	2.18	0.99–4.77	0.06	–	–	–
Stroke	1.81	0.93–3.55	0.08	–	–	–
DES implantation	0.95	0.56–1.59	0.85	–	–	–

ACS — acute coronary syndrome; CI — confidence interval; DES — drug eluting stent; HR — hazard ratio; LVEF — left ventricular ejection fraction; MI — myocardial infarction; STEMI — ST segment elevation myocardial infarction; TLC — target lesion calcification; TnI — troponin I

patients with ACS are advanced age, male sex, history of hypertension, and diagnosis STEMI [4]. In our study, except for age, there were no major differences in patients' baseline clinical and laboratory characteristics. Moreover, we showed that the prevalence of severe TLC was lower in patients with STEMI, which seems more consistent with the pathophysiological concept of plaque rupture. Conflicting results show that new, large-scale studies are needed to determine predictive factors of severe CAC in ACS patients, which may be useful in early risk stratification of ACS patients.

Our study showed that the number of MACE's in patients with severe TLC increases with time. Duration of follow-up is of paramount importance in every clinical trial. Especially in interventional trials, a delayed effect of intervention may

be observed [12]; however, our study was not designed to investigate the causes of subsequent coronary interventions and lesion failure. The pathophysiological cause of unfavourable long-term outcome after PCI in highly calcified coronary lesions (HCCL) is still uncertain. What we do know is that coronary lesions are very diverse regarding plaque characteristics. CAC increases the risk of procedural failure and PCI complications mostly due to its physical properties [13]. Different plaque composition causes various lesion reactions to balloon dilatation [14]. Calcium accumulations significantly increase the force needed to fully expand balloons and stents, therefore raising the risk of malapposition, restenosis, and thrombosis [15, 16]. Calcifications are usually unevenly distributed in the lesion; therefore, lesion resistance is not homogenous. Tension applied by (especially semi-compliant) balloons to the vessel wall in CAC is distributed unevenly and increases the risk of dissection [17]. Obstructive severe calcifications in segments proximal to the target lesion impairs device delivery, therefore increasing the risk of procedure failure [18]. PCI in HCCL is also especially challenging to DES because calcium may damage the polymer or drug coating leading to inadequate drug delivery and device ineffectiveness [19]. All the factors mentioned above may be responsible for higher rates of recurrent ACS in patients with severe TLC. The results of our study, and possible delayed effect of severe TLC on adverse events, shows that a longer follow-up period should be considered in future studies concerning patients with HCCL.

Nowadays, various treatment options are available allowing plaque modification with high procedural success rate and favourable safety profile [13]. The two affirmed as most effective in facilitating PCI in HCCL are (RA and recently introduced orbital atherectomy (OA). RA showed favourable results in treating HCCL in recent registries; however, there are no randomised trials concerning the use of RA in patients with severe TLC and AMI [20–22]. OA, a newcomer to coronary interventions, was first introduced in coronary arteries in 2013 in the ORBIT I trial [23]. Recent studies showed promising results of OA in hospital as well as in long-term follow-up [24, 25]. On the other hand, manufacturers of both devices included thrombus-containing lesions as a contraindication to the procedure. This is in concordance with our experience, as only one patient included in the study underwent RA. Despite good results of patients with ACS treated with RA, this plaque modification technique is not applicable to all ACS patients, so we should strive for new solutions to improve the outcomes of all patients with ACS and severe TLC [20].

Limitations of the study

Data was collected in 2012. This allowed us to collect long-term follow-up but, on the other hand, resulted in a large percentage of implanted BMS. Frequent BMS implantation in AMI was not uncommon at that time; however, it is unseen in more recent publications. Moreover, multivariate Cox regression models showed no impact of BMS implantation on prognosis.

This was a single-centre study conducted in a high-volume catheterisation laboratory, so our results may differ from those obtained in smaller centres.

The methodology of follow-up (receiving data from the Polish National Health Fund) enabled us to achieve complete data on all patients. Unfortunately, we were able to obtain only the information about the reason of hospitalisation and not the performed treatment. Therefore, we lack the information on the frequency of target vessel/lesion failure etc. Moreover, no information on the cause of death was available, so we can report only all-cause mortality.

CONCLUSIONS

Despite numerous reports, CAC in patients with ACS still seems to be an underestimated problem among interventional cardiologists. Severe TLC is not uncommon in patients with ACS. Our study is, to our knowledge, the first showing the incidence of severe CAC in a real-life population and its negative impact on PCI success rates and long-term clinical outcomes. The number of adverse events increases with time, so longer follow-up should be considered in future studies regarding this topic. The underlying pathophysiological mechanism causing poorer prognosis and higher rates of recurrent ACS is yet to be determined in further studies. Due to unfavourable outcome of patients with ACS and severe TLC, new therapeutic options for this populations should be considered.

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Conflict of interest: *none declared*

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Pacjenci z nasilonymi zwapnieniami w naczyniu odpowiedzialnym za niedokrwienie, poddawani zabiegom angioplastyki wieńcowej z powodu zawału serca cechują się złym rokowaniem w obserwacji odległej

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Streszczenie

Wstęp: Aktualne dane dotyczące wyników leczenia przezskórnego pacjentów z nasilonymi zwapnieniami w tętnicach wieńcowych pochodzą z badań klinicznych o krótkim okresie obserwacji. Nadal brakuje aktualnych informacji na temat odległych wyników zabiegów angioplastyki u chorych z zawałem serca i nasilonymi zwapnieniami w naczyniu odpowiedzialnym za niedokrwienie.

Cel: Celem pracy była ocena częstości występowania i wpływu na odległe wyniki leczenia przezskórnego nasilonych zwapnień w naczyniu odpowiedzialnym za niedokrwienie u pacjentów hospitalizowanych z powodu zawału serca.

Metody: Oceniono dane kliniczne i zabiegowe 206 kolejnych pacjentów poddanych zabiegom angioplastyki wieńcowej z powodu zawału serca. Pierwszorzędowymi punktami końcowymi były zgon z dowolnej przyczyny i kolejna hospitalizacja z powodu ostrego zespołu wieńcowego.

Wyniki: Nasilone zwapnienia w naczyniu odpowiedzialnym za niedokrwienie były obecne u 17% chorych. Pacjenci ci byli starsi (71 *vs*. 65 lat; p = 0,02) i częściej hospitalizowani z rozpoznaniem zawału serca bez uniesienia odcinka ST. Skuteczność zabiegów angioplastyki wieńcowej (80% *vs*. 97%; p < 0,0001) oraz odsetek pełnej rewaskularyzacji podczas pierwszego zabiegu (14% *vs*. 41%; p = 0,003) były również niższe w tej grupie chorych. Podczas 30-miesięcznej obserwacji osoby z nasilonymi zwapnieniami w naczyniu odpowiedzialnym za niedokrwienie częściej doznawali kolejnego ostrego zespołu wieńcowego (37% *vs*. 13%; p = 0,0005) oraz charakteryzowali się wyższą śmiertelnością całkowitą (31% *vs*. 16%; p = 0,04). Wieloczynnikowy model regresji Coxa wskazał nasilone zwapnienia w naczyniu odpowiedzialnym za niedokrwienie jako niezależny predyktor kolejnej hospitalizacji z powodu ostrego zespołu wieńcowego (HR 2,8; 95% CI 1,4–5,6; p = 0,004).

Wnioski: Nasilone zwapnienia w naczyniu odpowiedzialnym za niedokrwienie nie są rzadkim zjawiskiem u chorych hospitalizowanych z powodu zawału serca. Ich obecność jest czynnikiem predykcyjnym wystąpienia kolejnego ostrego zespołu wieńcowego w tej populacji pacjentów.

Słowa kluczowe: zawał serca, przezskórne interwencje wieńcowe, zwapnienia, rewaskularyzacja

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