Impact of chronic total occlusion on prognosis in cardiogenic shock due to unprotected left main coronary artery culprit lesion. Insights from the Polish Registry of Acute Coronary Syndromes

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ABSTRACT

Background: Notwithstanding readily available revascularization, significant advancements in mechanical circulatory support, and pharmacological progress, cardiogenic shock (CS) secondary to unprotected left main culprit lesion-related acute myocardial infarction (ULMCL-related AMI) is associated with very high mortality. In this population, chronic total occlusion (CTO) is relatively frequent.

Aims: This study sought to assess the association between the presence of CTO and 12-month mortality in patients with CS due to ULMCL-related AMI.

Results: The study included consecutive patients admitted for AMI-related CS with ULMCL who underwent percutaneous coronary intervention (PCI) and were enrolled in the prospective Polish Registry of Acute Coronary Syndromes (PL-ACS) between January 2017 and December 2021. The patients were stratified into two groups based on the presence of at least one CTO. The primary endpoint was all-cause death at 12 months. Of the 250 included patients, 60 (24%) patients had one or more CTOs of a major coronary artery (+CTO), and in 190 (76%) patients, the presence of CTO was not observed (–CTO). The 12-month mortality rates for the +CTO and –CTO patients were 85% and 69.8%, respectively (P log-rank = 0.03). After multivariable adjustment for differences in the baseline characteristics, the presence of CTO remained significantly associated with higher 12-month mortality (hazard ratio, 1.423; 95% CI, 1.027–1.973; P = 0.034).

Conclusions: Our analysis showed that in patients with CS due to ULMCL-related AMI treated with PCI, the presence of CTO is associated with worse 12-month prognosis.

Key words: acute myocardial infarction, cardiogenic shock, chronic total occlusion, prognosis

INTRODUCTION

Cardiogenic shock (CS) complicating acute myocardial infarction (AMI) is a critical clinical situation. Unfortunately, despite tremendous efforts and progress in its treatment, including pharmacotherapy advancements, rapid access to high-quality revascularization, and availability of mechanical circulatory support (MCS), CS remains the leading cause of death in AMI patients, with in-hospital mortality as high as 40%–50% [1–5]. Mortality is further increased with rates of up to 70% in cases of refractory CS [6].

Unprotected left main culprit lesion-related acute myocardial infarction (ULMCL-related AMI) is associated with a faster presentation

WHAT'S NEW?

The presence of chronic total occlusion (CTO) in patients with cardiogenic shock (CS) secondary to unprotected left main (LM) coronary artery culprit lesion-related acute myocardial infarction (AMI) treated with percutaneous coronary intervention (PCI) is independently related to higher 12-month mortality. The patients with AMI-related CS, just after diagnostic catheterization with a detected culprit lesion in the LM, should be stratified by the presence of CTO. Additional research is needed to understand the safety and efficacy of CS treatment in terms of the extent of revascularization and/or escalation therapy using mechanical circulatory support (MCS) based on the presence of CTO stratification.

of CS, more severe systemic organ failure, worse outcomes even in cases of successful revascularization [7], and very high mortality (up to 75%) [8].

It has been demonstrated that the presence of chronic total occlusion (CTO) of an artery other than the infarct-related one in patients hospitalized for AMI and AMI-related CS is strongly associated with higher rates of in-hospital and long-term mortality than in patients without CTO [9–13]. To the best of our knowledge, there is lack of data evaluating the presence of CTOs in patients with CS due to ULMCL-related AMI.

Thus we aimed to analyze the impact of CTO on long-term prognosis in this patient population using data from a large national multi-center registry.

METHODS

Design of the registry

We used data from the Polish Registry of Acute Coronary Syndromes (PL-ACS). The methodology and analysis have been previously described [14]. In brief, the PL-ACS registry is one of the largest in Europe. It is an ongoing, nationwide, multi-center, prospective, observational study of consecutively hospitalized Polish patients suffering the entire spectrum of acute coronary syndromes. The registry is a joint initiative of the Silesian Center for Heart Diseases and the Polish Ministry of Health. The National Health Fund, a nationwide public health insurance institution in Poland, provides logistical support.

Data on long-term all-cause mortality, including the exact date of death, were obtained from the National Health Fund by January 2022. Follow-up time was censored at one year or the end of follow-up time (whichever came first).

Study population and definitions

Between January 1, 2017, and December 31, 2021, a total of 4954 patients with AMI-related CS were enrolled in the PL-ACS. In this cohort, 321 consecutive patients had culprit lesions located in the LM and underwent LM percutaneous coronary intervention (PCI). Patients after coronary artery bypass grafting and/or missing medical history were excluded. Finally, 250 patients with ULMCL-related AMICS were analyzed and stratified into two groups based on the presence of at least one CTO lesion (+CTO group, n = 60, 24% vs. –CTO group, n = 190, 76%) (Figure 1).

The ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI) were defined according to the Fourth Universal Definition of Myocardial Infarction [15]. CS was defined as 1) systolic blood pressure <90 mm Hg (in the absence of hypovolemia and after proper fluid resuscitation) for at least 30 min or the need for pharmacological support to maintain systolic blood pressure above 90 mm Hg; and 2) signs and symptoms of end-organ hypoperfusion. The definition of ULMCL was at the discretion of the interventional cardiologist performing PCI, based on angiographic criteria such as the presence of thrombus, ulceration, degree of stenosis, distal flow, and anatomical characteristics of the rest of the coronary tree. CTO was defined as any 100% stenosis of the coronary artery on the index angiography, which the operators did not consider as the culprit lesion responsible for the index AMI based on clinical, angiographic, electrocardiographic, echocardiographic, or previous angiography findings. The primary outcome of interest was all-cause 12-month mortality. Secondary outcomes included the incidence of mechanical complications, stroke, major bleeding, resuscitated cardiac arrest, and death during the index hospitalization. The study was approved by the institutional review committee.

Statistical analysis

The continuous variables were presented as medians and interquartile ranges. The categorical variables were presented as percentages. Differences between categorical variables in the baseline characteristics, angiographic characteristics, and in-hospital outcomes were compared using Pearson's χ^2 test or Fisher's exact test, where appropriate. Quantitative variables were compared using the Wilcoxon rank sum test. The cumulative 1-year incidence of all-cause death in patients stratified by the presence of CTO was depicted with Kaplan-Meier curves. The log-rank test was used to compare mortality rates between groups. Additionally, landmark analysis was performed with a landmark set at 30 days (one month). Moreover, we have performed some sensitivity analyses, i.e., survival analysis in the subset of patients who underwent PCI for non-culprit lesion during the index hospitalization, the comparison of 12-month mortality between patients with CTO and those with subtotal stenoses (70%–99%) in the non-culprit vessels, in patients stratified by the location of CTO (right vs. left coronary artery) and in patients

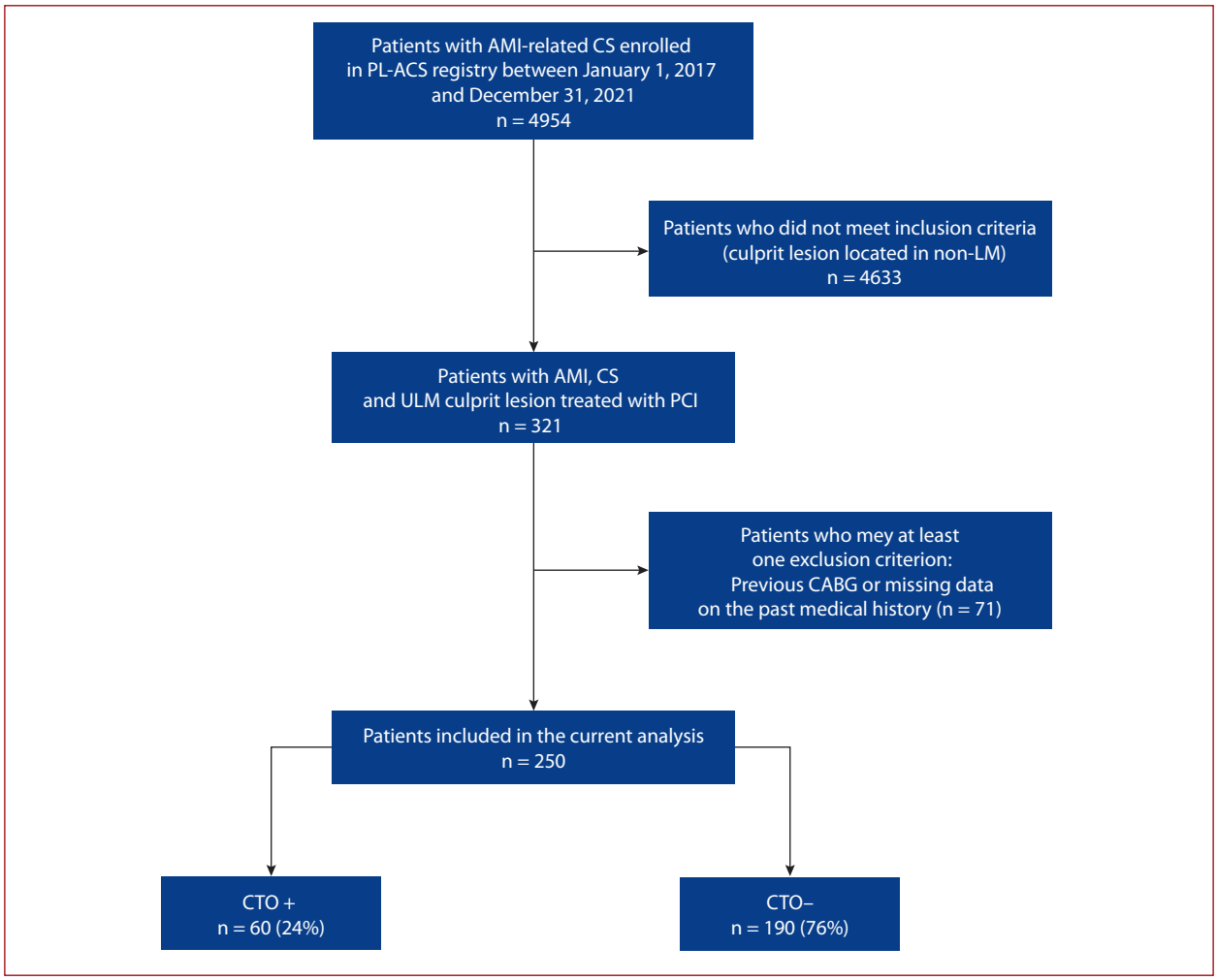

Figure 1. Study flowchart

Abbreviations: AMI, acute myocardial infarction; CABG, coronary artery bypass grafting; CS, cardiogenic shock; CTO, chronic total occlusion; LM, left main; PCI, percutaneous coronary intervention; PL-ACS, Polish Registry of Acute Coronary Syndromes; ULM, unprotected left main

stratified by the myocardial infarction presentation (STEMI vs. NSTEMI). The interaction between the presence of CTO and myocardial infarction classification was assessed using the likelihood ratio test. The unadjusted and adjusted Cox proportional-hazards models were created to analyze the relationship between CTO and 12-month mortality. The proportional hazards assumption was tested using the Schoenfeld residuals. The multivariable analysis was performed using the data set with missing values imputed by the random forest algorithm (using the missForest package). Clinically relevant baseline clinical characteristics variables with P <0.05 in the univariable models (chronic kidney disease, peripheral vascular disease, age, obesity, previous stroke) were included in the multivariable model. The level of statistical significance was set at P <0.05 (two-tailed). All statistical analyses were performed using R version 4.2.2 (R Core Team [2022]. R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria) and RStudio (RStudio Team [2020]. RStudio: Integrated Development for R. RStudio, PBC, Boston, MA, US).

RESULTS

In the whole population of patients with CS complicated by AMI, the rate of ULMCL was 6.5%. The baseline clinical characteristics of the study groups are presented in Table 1. The patients with CTO had a higher prevalence of diabetes and STEMI vs. NSTEMI than the patients without CTO. The angiographic and periprocedural characteristics are listed in Table 2. CTO patients had a higher frequency of multivessel coronary disease, higher prevalence of totally occluded ULMCL, lower rates of Thrombolysis In Myocardial Infarction (TIMI) 3 after PCI, and more often underwent PCI of non-CTO lesions during index hospitalization. The PCI of CTO during index hospitalization of 15 (25%) patients was reported, with a success rate defined as TIMI flow 3 in 5 (33%) patients. The in-hospital secondary outcomes are presented in Table 3. A total of 68% of the +CTO patients and 47% of the –CTO patients (P = 0.004) died during the index hospitalization. The follow-up for death was available for 249 of 250 patients, and the median follow-up time was 6 (1–306) days. At 12 months, a significant difference in the all-cause mortality rate was recorded: n = 51 (85%)

Table 1. Baseline clinical characteristics

Variable	Group			
	Overall n = 250[a]	CTO+ n = 60[a]	CTO– n = 190[a]	*P*-value[b]
Sex, male	184 (74%)	49 (82%)	135 (71%)	0.10
Age, years	70 (63–80)	69 (65–83)	71 (62–80)	0.45
Smoking				0.67
Current smoker	58 (32%)	12 (29%)	46 (32%)	
Former smoker	70 (38%)	18 (44%)	52 (36%)	
Never smoked	56 (30%)	11 (27%)	45 (31%)	
Hypertension	137 (61%)	36 (68%)	101 (59%)	0.26
Hyperlipidemia	65 (33%)	14 (31%)	51 (33%)	0.80
Diabetes	73 (33%)	24 (46%)	49 (28%)	0.02
Obesity	46 (20%)	9 (18%)	37 (20%)	0.70
Previous myocardial infarction	57 (24%)	15 (27%)	42 (23%)	0.52
Previous PCI	49 (20%)	11 (18%)	38 (20%)	0.72
Peripheral vascular disease	36 (15%)	8 (14%)	28 (15%)	0.80
Atrial fibrillation	31 (13%)	4 (6.8%)	27 (15%)	0.11
Chronic heart failure	35 (15%)	10 (17%)	25 (14%)	0.52
Previous stroke	21 (8.5%)	7 (12%)	14 (7.4%)	0.28
Chronic kidney disease	30 (12%)	10 (17%)	20 (11%)	0.22
Ejection fraction (%)	30 (20–40)	28 (20–35)	30 (20–40)	0.35
CA before admission	78 (31%)	18 (31%)	60 (32%)	0.87
Pain-to-admission time, hours	672 (240–2160)	720 (300–1740)	636 (240–2160)	0.52
ACS type				**<0.001**
STEMI	114 (46%)	16 (27%)	98 (52%)	
NSTEMI	136 (54%)	44 (73%)	92 (48%)	
SBP, mm Hg	90 (80–110)	90 (80–118)	90 (80–110)	0.75
DBP, mm Hg	60 (50–70)	60 (50–70)	60 (50–70)	0.58
HR, 1/min	90 (75–100)	94 (80–100)	90 (74–104)	0.39

[a]Median (interquartile ranges) or frequency (%); [b]Pearson's χ^2 test, Wilcoxon rank sum test, Fisher's exact test

Abbreviations: ACS, acute coronary syndrome; CA, cardiac arrest; DBP, diastolic blood pressure; HR, heart rate; NSTEMI, non-ST-segment elevation myocardial infarction; SBP, systolic blood pressure; STEMI, ST-segment elevation myocardial infarction; other — see Figure 1

Table 2. Angiographic and periprocedural characteristics

Variable	Group			
	Overall n = 250[a]	CTO+ n = 60[a]	CTO– n = 190[a]	*P*-value[b]
Door-to-catheter time, minutes	28 (10–90)	30 (12–73)	26 (10–90)	0.52
Multivessel disease				<0.001
1VD or isolated LM	135 (54%)	3 (5.0%)	132 (69%)	
2VD	57 (23%)	22 (37%)	35 (18%)	
3VD	58 (23%)	35 (58%)	23 (12%)	
LAD CTO	23 (9.2%)	23 (38%)	0 (0%)	**<0.001**
Cx CTO	21 (8.4%)	21 (35%)	0 (0%)	**<0.001**
RCA CTO	35 (14%)	35 (58%)	0 (0%)	**<0.001**
TIMI flow in LM before PCI				**<0.001**
0	78 (31%)	38 (63%)	40 (21%)	
1	26 (10%)	3 (5.0%)	23 (12%)	
2	32 (13%)	4 (6.7%)	28 (15%)	
3	114 (46%)	15 (25%)	99 (52%)	
Vascular access				**0.003**
Radial	99 (40%)	16 (27%)	83 (44%)	
Femoral	145 (58%)	39 (66%)	106 (56%)	
Other	5 (2.0%)	4 (6.8%)	1 (0.5%)	
LM PCI	250 (100%)	60 (100%)	190 (100%)	NA
Non-culprit vessel PCI	143 (57%)	42 (70%)	101 (53%)	**0.022**
TIMI flow in LM after PCI				**0.001**
0–2	53 (24%)	15 (47%)	38 (20%)	
3	168 (76%)	17 (53%)	151 (80%)	
Glycoprotein IIb/IIIa inhibitor	98 (39%)	21 (36%)	77 (41%)	0.49
IABP	28 (11%)	9 (15%)	19 (10%)	0.28
Advanced MCS	1 (0.4%)	0 (0%)	1 (0.5%)	1.0
CTO PCI during index admission	15 (6.0%)	15 (25%)	0 (0%)	**<0.001**
CABG during index admission	4 (1.6%)	0 (0%)	4 (2.1%)	0.57

[a]Median (interquartile ranges) or frequency (%); [b]Wilcoxon rank sum test, Pearson's χ^2 test, Fisher's exact test

Abbreviations: Cx, circumflex artery; IABP, intra-aortic balloon pump; LAD, left anterior descending artery; MCS, mechanical circulatory support; NA, not applicable; RCA, right coronary artery; TIMI, Thrombolysis In Myocardial Infarction; VD, vessel disease; other — see Figure 1

Table 3. In-hospital outcomes

Variable	Group			
	Overall n = 250[a]	CTO+ n = 60[a]	CTO– n = 190[a]	*P*-value[b]
Mechanical complications	6 (2.4%)	0 (0%)	6 (3.2%)	0.34
Stroke	5 (2.0%)	2 (3.4%)	3 (1.6%)	0.33
Major bleeding	8 (3.2%)	4 (6.8%)	4 (2.1%)	0.093
Resuscitated cardiac arrest	89 (36%)	28 (47%)	61 (32%)	0.032
Death	130 (52%)	41 (68%)	89 (47%)	0.004

[a]Frequency (%); [b]Fisher's exact test, Pearson's χ^2 test

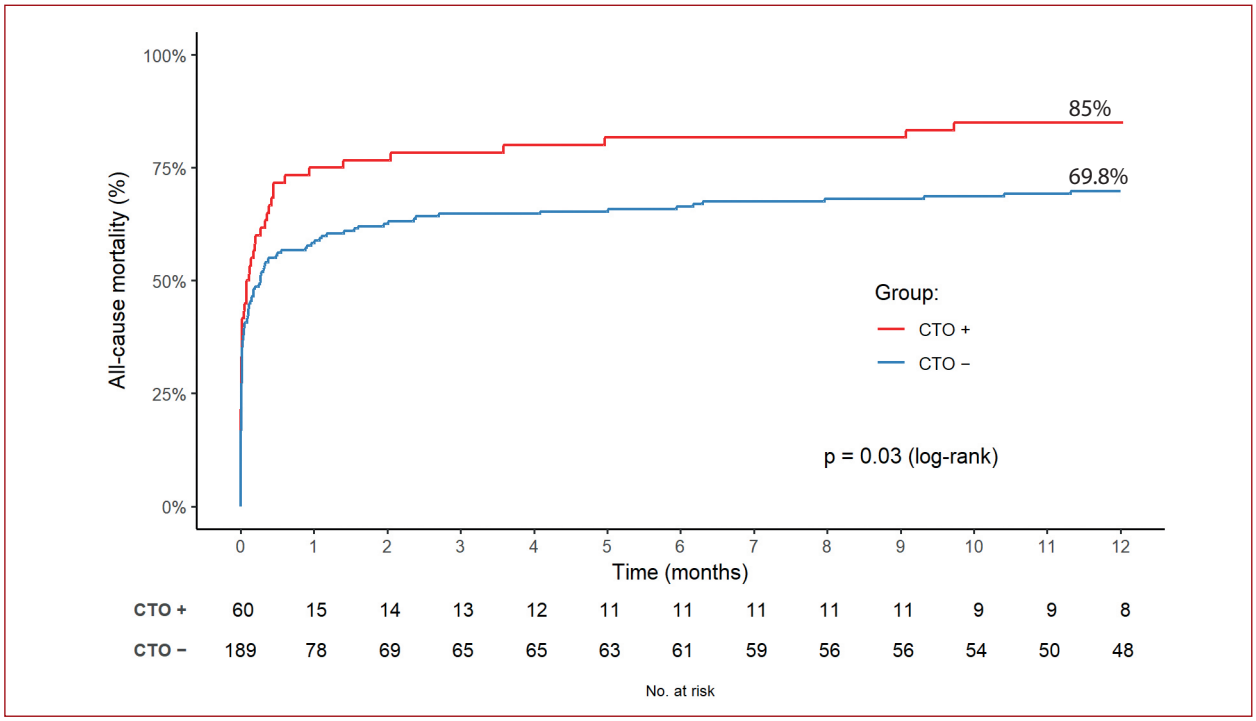

Figure 2. Kaplan–Meier curves presenting the incidence of all-cause 12-month mortality in groups stratified by the presence of chronic total occlusion (CTO) in non-culprit vessels

Abbreviation: see Table 1

of the +CTO and n = 132 (69.8%) of the –CTO (log-rank *P*-value of 0.03) (Figure 2). Most deaths occurred within the first 30 days following the index admission: n = 45 (75%) in the +CTO and n = 110 (58.3%) in –CTO groups (log-rank *P*-value of 0.04) (Figure 3). Similar observations were made in the subgroup of patients who underwent PCI in a non-CTO coronary artery during index hospitalization (12-month mortality of 83.3% in +CTO and 66.3% in the –CTO patients, the log-rank *P*-value of 0.04) (Supplementary material, *Figure S1*). Moreover, patients with CTO were at higher risk of all-cause death when compared to patients without CTO but with angiographically significant lesions (70%–99% stenosis) in the non-culprit vessel (85.0% vs. 69.9%, log-rank *P*-value of 0.02) (Supplementary material, *Figure S2*). Further analysis showed similar 12-month mortality in +CTO patients, irrespective of CTO location in the right or left coronary artery (log-rank *P*-value of 0.42) (Supplementary material, *Figure S3*). The effect of CTO on 12-month mortality was also comparable in STEMI and NSTEMI patients (*P*-value for the interaction of 0.62) (Supplementary material, *Figure S4*). The relationship between the baseline clinical characteristics and 12-month mortality in the univariable analysis is presented in Supplementary material, *Table S1*. In the multivariable analysis, the presence of CTO was independently associated with increased risk of 12-month mortality (hazard ratio 1.423; 95% CI, 1.027–1.973; *P* = 0.034) (Figure 4).

DISCUSSION

Cardiogenic shock complicated by AMI is one of the most severe and challenging acute clinical settings, requiring the greatest medical attention. The prevalence of CS ranges between 4%–10% [2, 3, 16]. Despite the current advances in multilevel treatment approaches, CS continues to entail an unacceptable early and long-term mortality risk [17], which has not changed over the last decade [18].

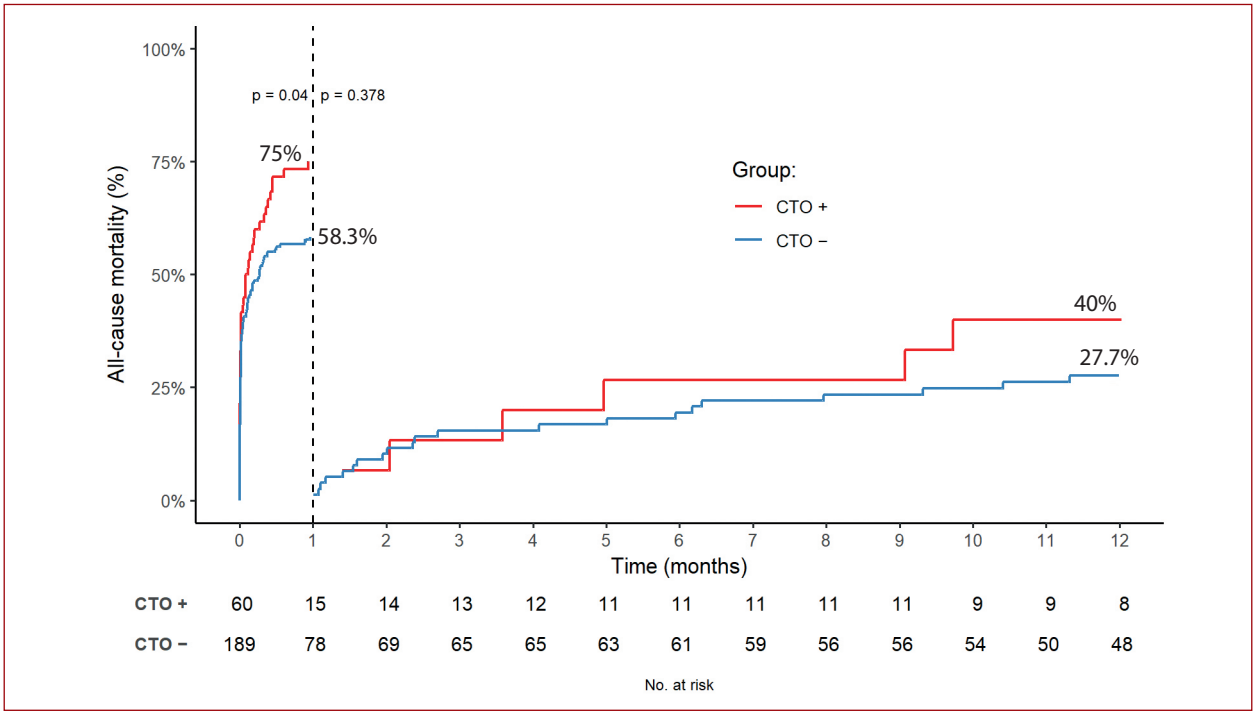

Figure 3. The results of landmark analysis for 12-month all-cause mortality in patients stratified by the presence of chronic total occlusion (CTO)

Abbreviation: see Table 1

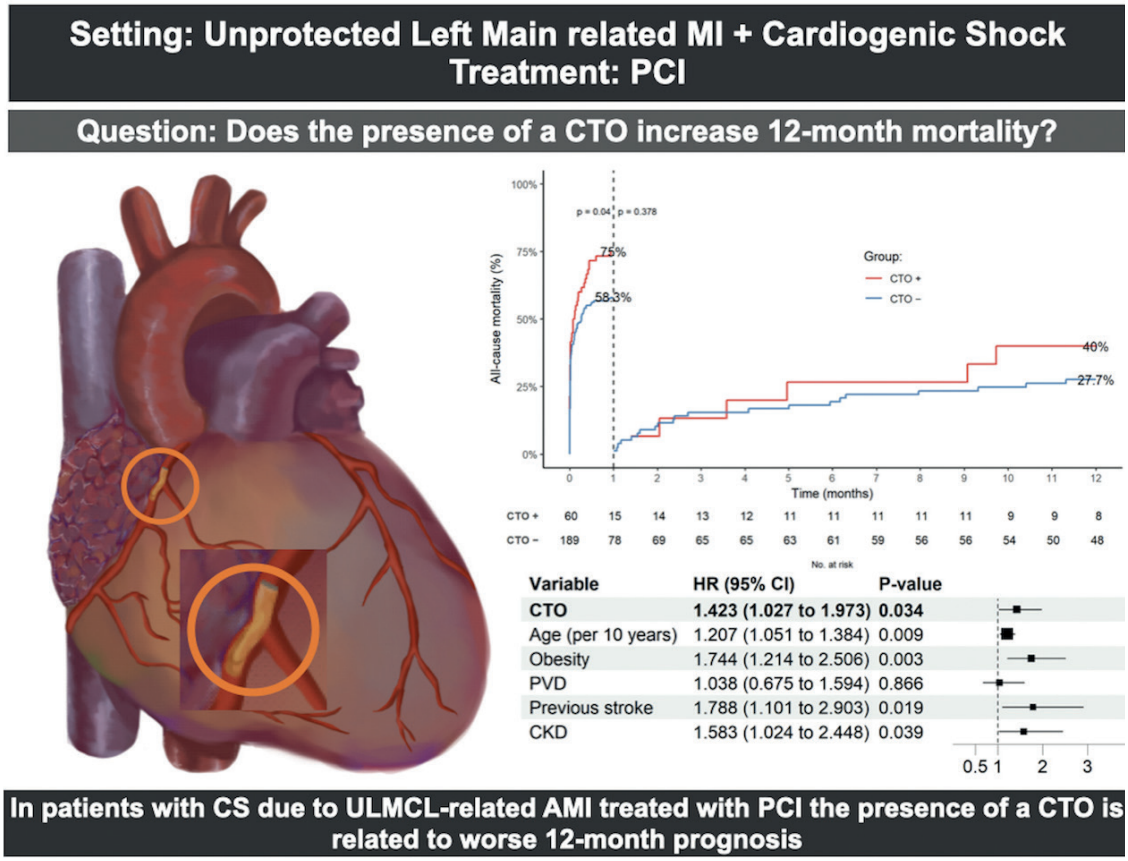

Figure 4. Multivariable analysis of the entire study population outcomes. Forest plot presenting hazard ratios (HR) and 95% confidence intervals (CI) for the variables included in the multivariable Cox regression model for 1-year all-cause mortality

Abbreviations: CKD, chronic kidney disease; CTO, chronic total occlusion; MI, myocardial infarction; PCI, percutaneous coronary intervention; PVD, peripheral artery disease

Presently, coronary revascularization in the acute phase of CS related to AMI has been shown as the only factor modifying prognosis positively [19]. Therefore, prompt coronary angiography to detect a culprit lesion-related AMI complicated with CS is mandatory. Patients with CS complicated by AMI and a culprit lesion located in the left coronary main artery form a particular subgroup in this setting, with higher risk of mortality even after adjusting for confounding clinical and procedural characteristics [8, 20]. Additionally, in CS patients, CTO of an artery other than the culprit vessel is relatively common [12, 13]. We have, therefore, hypothesized that the presence of CTO may be a marker of a worse prognosis, which may be useful for risk stratification in patients with AMI complicated by CS related to ULMCL.

The current practice guidelines for managing heart failure published by the European Society of Cardiology and the guidelines for myocardial revascularization created by both the European Society of Cardiology and the European Association for Cardio-Thoracic Surgery do not recommend any strategy of treatment in the subpopulation of patients with CS due to ULMCL-related AMI and concomitant CTO in an artery other than the culprit lesion [21, 22]. To the best of our knowledge, there are no published data concerning the impact of CTO on the short- and long-term prognosis in this group of patients.

Therefore, our study aimed to evaluate the role of CTO in predicting long-term mortality in patients with CS secondary to ULMCL-related AMI. The main findings from this investigation can be summarized as follows: first, CTO was relatively frequent in this cohort of patients (24%); second, the presence of CTO in patients with CS due to ULMCL-related AMI was associated with increased risk of long-term mortality, also after adjustment for potential confounders in the multivariable analysis; third, most deaths occurred within the first 30 days following the index admission, and the relationship between CTO and worse outcomes was particularly noticeable within this period.

The presence of CTO in a vessel other than the culprit one in patients with CS is relatively high, with a recorded prevalence of 25%–30% [13, 23], consistent with the rate reported in this study. The reason why concurrent CTO is associated with worse prognosis in patients with CS secondary to ULMCL-related AMI is unknown and may be partially explained by the higher risk profile of CTO patients (higher prevalence of diabetes, higher percentage of multivessel coronary disease, lower rate of PCI success as assessed by TIMI flow). However, after adjustment for differences in baseline characteristics by multivariable Cox regression analysis, CTO remains an independent predictor of 12-month mortality.

Similarly, in the published sub-analysis of the IABP-SHOCK II (Intraaortic Balloon Pump in Cardiogenic Shock) trial of the prognostic impact of a CTO in a non-infarct related artery (non-IRA) in STEMI, Saad et al. [24] demonstrated that CTO in a non-IRA was an independent predictor of one-year mortality. Interestingly, CTO in a non-IRA was a predictor of ventricular arrhythmias requiring defibrillation at 30-day follow-up, which is in line with our findings of a higher rate of in-hospital resuscitated cardiac arrest in the CTO group as compared to non-CTO.

These last interactions may shed some light on the discussion of the potential mechanism underlying increased mortality in patients with CTO after AMI complicated by CS due to ULMCL. Nombela-Franco et al. [25], in the VACTO (Ventricular Arrhythmias and Chronic Total Coronary Occlusion) Primary Study, showed the prognostic importance of CTO in the incidence of appropriate implantable cardioverter-defibrillators (ICD) interventions for ventricular arrhythmia and its impact on poor survival in a cohort of patients receiving ICD treatment for primary prevention of sudden cardiac death from ischemic cardiomyopathy. Consistently, Di Marco et al. [26] showed that the presence of CTO was associated with higher scar burden and was an independent predictor of ventricular tachycardia recurrence after successful ventricular tachycardia ablation.

Current practice guidelines recommend that prophylactic implantations of the ICD for the primary prevention of sudden cardiac death in patients with MI and depressed left ventricular contractility should be delayed for at least 40 days [21]. According to our findings, emphasizing that most patients died within the first 30 days following the index admission, we can postulate that in patients with CS-complicated AMI secondary to ULMCL, the presence of concomitant CTO may provide an additional vital parameter for risk stratification and may be a matter of other investigations in this group of patients.

Finally, some studies showed that in stable patients undergoing unprotected left main PCI, CTO of the right coronary artery (RCA) may be associated with increased risk of periprocedural complications and mortality [27, 28]. This may be because during unprotected left main PCI, a large region of myocardium is jeopardized, and in the absence of a patent RCA, hemodynamic deterioration is more likely [28]. However, not all studies found a negative effect of the lack of RCA flow on outcomes, which may be partially explained by differences between studies in left main PCI complexity, clinical characteristics of included patients, or clinical context [28, 29]. Indeed, our study showed that in the setting of CS, the prognosis of patients with ULMCL and any CTO is poor, irrespective of CTO location.

Our study underlines the prognostic value of concurrent CTO in the very high-risk population of patients with CS due to ULMCL-related AMI. However, owing to the observational nature of our study, the causal relationship between CTO and worse outcomes cannot be confirmed. Moreover, even if, intuitively, CTO revascularization might seem beneficial in patients with CS, there are no data supporting such an approach. Despite significant technical progress, CTO PCI remains a complex procedure with success strongly related to the operator's skills and a relatively higher rate of periprocedural complications, which seems

to be of special importance in the setting of CS [30]. Moreover, PCI of ULM in the absence of RCA support, differed neither in the prevalence of periprocedural complications nor in long-term survival, as compared to PCI with RCA support [31]. Notably, the landmark CULPRIT-SHOCK (Culprit Lesion Only PCI Versus Multivessel PCI in Cardiogenic Shock) trial showed no benefit of immediate complete revascularization compared with culprit-lesion-only PCI in STEMI patients with CS [23]. Importantly, in this trial, no modifying effect of CTO on this finding was observed [13].

Acute myocardial infarction is the most essential reason for CS development, resulting in a subsequent sudden and significant decrease in myocardial contractility. This leads to a vicious circle of reduced cardiac output (CO), hypotension, coronary perfusion worsening, and further ischemic deterioration of myocardial function with inadequate critical organ perfusion [31]. Thus, several MCS devices have been developed aiming to break the circle and change the unfavorable prognosis in CS complicated by AMI. Furthermore, apart from augmentation of CO with the subsequent improvement of systemic perfusion, MCS can also reduce the burden of myocardial ischemia [32]. However, it is challenging to determine the appropriate time to escalate therapy to an MCS device or determine which MCS device should be used. Again, we speculate that the presence of a CTO might help improve the selection of patients with CS secondary to ULMCL-AMI who may benefit from MCS, but further studies regarding this issue are needed.

Limitations

There are several limitations to our analysis that should be acknowledged. Due to the observational character of the study, the causal relationship between the presence of CTO and higher mortality cannot be confirmed. Despite data adjustment in the multivariable analysis, the results could still be biased by potentially important parameters that were not available in the registry. Additionally, owing to the limited sample size, this analysis was underpowered to evaluate the association between successful CTO recanalization and outcomes in the present analysis. Finally, as it is a single-country study, it may not apply to other populations.

CONCLUSIONS

In a large registry, we found that in patients with CS secondary to unprotected left main coronary artery culprit lesion-related AMI treated with PCI, the presence of CTO is associated with a higher 12-month mortality.

Supplementary material

Supplementary material is available at https://journals.viamedica.pl/kardiologia_polska.

Article information

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