Kardiologia Polska 2017; 75, 2: 108–116; DOI: 10.5603/KP.a2016.0130

ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

# Total coronary occlusion of infarct-related arteries in patients with non-ST-elevation myocardial infarction undergoing percutaneous coronary revascularisation

Jarosław Karwowski[1], Lech Poloński[2], Marek Gierlotka[2], Andrzej Ciszewski[3], Michał Hawranek[2], Maciej Bęćkowski[1], Mariusz Gąsior[2], Ilona Kowalik[1], Hanna Szwed[1]

[1]2nd Department of Coronary Artery Disease, Institute of Cardiology, Warsaw, Poland
[2]3rd Department of Cardiology, Silesian Centre for Heart Diseases, Zabrze, Poland
[3]Invasive Cardiology Ward, Institute of Cardiology, Warsaw, Poland

## Abstract

**Background:** The prevalence and impact of total coronary occlusion of an infarct-related artery (IRA) on outcomes in patients with non-ST-elevation myocardial infarction (NSTEMI) remain unclear.

**Aim:** We evaluated the clinical significance of total coronary occlusion in NSTEMI patients.

**Methods:** A total of 2767 patients with NSTEMI enrolled in the Polish Registry of Acute Coronary Syndromes, who underwent percutaneous coronary interventions, were analysed. The patients were divided into two groups according to preprocedural culprit vessel thrombolysis in myocardial infarction (TIMI) flows (TIMI flow 0 — total coronary occlusion [TO]: 728, 26.3% of the patients, and TIMI flow 1–3 — non-total occlusion [non-TO]: 2039, 73.7% of the patients).

**Results:** Patients with total occlusion were younger, were more often current smokers, and had lower incidence of hypertension and diabetes mellitus. The left circumflex artery (LCx) was the major IRA in the TO group (48.1%), whereas the left anterior descending artery (LAD) was more commonly the IRA in the non-TO group (38.8%). Multivariate analysis revealed that LCx as the culprit lesion (OR ± 95 CI 1.54 [1.26–1.89], $p < 0.0001$) was an independent predictor of TIMI flow 0 in IRA. In-hospital and one-month mortality occurred more frequently in the TO group (4.0% vs. 1.7%, $p = 0.0005$ and 5.5% vs. 3.5%, $p = 0.0175$, respectively), no differences in the 12-, 24-, or 36-month mortalities were observed between these groups.

**Conclusions:** Only LCx as a culprit lesion was an independent predictor of total occlusion in IRAs. The NSTEMI patients with TO had higher in-hospital and one-month mortalities, but their long-term outcomes were similar to those of non-TO patients.

**Key words:** acute total coronary occlusion, long-term mortality, non-ST-segment elevation myocardial infarction, percutaneous coronary interventions

Kardiol Pol 2017; 75, 2: 108–116

## INTRODUCTION

The standard 12-lead electrocardiogram (ECG) plays a crucial role in the diagnosis and clinical decision-making process regarding the timing of percutaneous coronary intervention (PCI) in acute myocardial infarction (AMI) [1, 2]. Patients with AMI are generally classified as having ST-elevation myocardial infarction (STEMI) or non-ST-elevation myocardial infarction (NSTEMI) based on the appearance of the ECG. STEMI is caused by acute occlusion of the culprit artery associated with transmural ischaemia, whereas NSTEMI is usually caused by a transient or non-complete coronary occlusion that causes non-transmural subendocardial ischaemia [3]. However, it is well known that the sensitivity of ST-segment elevation in the identification of total occlusion (TO) is

**Address for correspondence:**
Jarosław Karwowski, MD, 2nd Department of Coronary Artery Disease, Institute of Cardiology, ul. Spartańska 1, 02–637 Warszawa, Poland, tel: +48 502913169, fax: +48 22 844 95 10, e-mail: karwowski.jarek@gmail.com

**Received:** 04.03.2016 **Accepted:** 13.07.2016 **Available as AoP:** 22.08.2016

suboptimal, particularly for occlusions in the posterolateral circulation.

Therefore, we can distinguish a difficult-to-diagnose group of patients with NSTEMI with occluded infarct-related arteries (IRAs), who can be termed "STEMI equivalent" [4]. It is likely that early invasive strategies with PCIs and full restoration of blood flow in the IRA would improve the outcomes of these patients compared to the outcomes obtained with conservative management or late PCI.

Based on these facts and the limited data that are available for the NSTEMI population, this study was conducted to identify the predictors of acute occlusion in IRA and to evaluate impact of total occlusion on the outcomes of NSTEMI patients.

## METHODS

### *Design of the registry*

We used data from the Polish Registry of Acute Coronary Syndromes (PL-ACS). The methods and results of an investigation of the first 100,193 patients have previously been described [5]. The PL-ACS registry is an ongoing, nationwide, multicentre, prospective, observational study of hospitalised patients across the entire spectrum of ACSs in Poland. All Polish regions collect data for the PL-ACS. A detailed protocol with the inclusion and exclusion criteria, methods, logistics, and definitions of all fields exists in the registry dataset. The protocol was revised in May of 2004 to comply with the Cardiology Audit and Registration Data Standards (CARDS) [6]. Patients with suspected ACS were screened for eligibility to enter the registry but were not enrolled until ACS was confirmed. If, within the acute phase of myocardial infarction, a patient was transferred to another hospital, both hospitals were required to complete the case report form. These hospitalisations were linked together during data processing and were subsequently analysed as one case of ACS. The data were collected by the skilled physicians who were in charge of each particular patient. Initial internal checks for missing or conflicting data and values outside of the expected range were implemented within the software. Once a month, the collected data were sent to the National Health Fund, where they were cross-checked with standard hospital reports. After data verification, the National Health Fund transferred the data to the central database in the Silesian Centre for Heart Diseases in Zabrze, Poland, where further checks were applied. All-cause mortality data with the accompanying exact dates of death were obtained from the official mortality records of the National Health Fund.

### *Patients and definitions*

Between January and December 2008, a total of 4125 patients with NSTEMI were treated with PCI and registered in the PL-ACS. Patients with prior myocardial infarction, prior PCI, and prior coronary artery bypass grafting (CABG) were excluded from the analysis. Consequently, 2767 patients fulfilled the inclusion and exclusion criteria and were enrolled in this investigation. The patients were divided into two groups based on the presence of baseline thrombolysis in myocardial infarction (TIMI) flow grade 0 upon angiography as follows: baseline TIMI flow 0 (TO group, n = 728) and baseline TIMI flow ≥ 1 (non-TO group, n = 2039). TIMI Grade Flow is a scoring system from 0–3 referring to levels of coronary blood flow assessed during angiography: TIMI 0 flow (no perfusion) refers to the absence of any antegrade flow beyond a coronary occlusion; TIMI 1 flow (penetration without perfusion) is faint antegrade coronary flow beyond the occlusion, with incomplete filling of the distal coronary bed; TIMI 2 flow (partial reperfusion) is delayed or sluggish antegrade flow with complete filling of the distal territory; and TIMI 3 is normal flow that fills the distal coronary bed completely. NSTEMI was defined as (1) the absence of ST-segment elevation consistent with myocardial infarction ≥ 2 mm in the adjacent chest leads and ST-segment elevation ≥ 1 mm in two standard leads with new left bundle branch block and (2) positive cardiac necrosis markers. A coronary artery was considered an IRA (culprit) based on the following: angiographic features (definite or suspected thrombus, ruptured or ulcerated plaque, and the presence of TIMI of flow grade ≤ 2), ECG recordings, and echocardiographic findings. Culprit locations in the left anterior descending artery (LAD) were defined as anterior culprits. Culprit locations in either the right coronary artery (RCA) or the left circumflex artery (LCx) were defined as infero- or posterolateral culprits. In-hospital and long-term mortalities were defined based on death from all causes (cardiac and non-cardiac). The invasive strategy was defined as the performance of coronary angioplasty during the index hospitalisation. Decisions related to treatment modalities (i.e. the use of stents, intra-aortic balloon pump, glycoprotein IIb/IIIa inhibitors, and methods of angioplasty) were left to the discretion of the attending physicians.

### *Statistical analysis*

The continuous data are expressed as the arithmetic means ± the standard deviation for normally distributed variables or as the medians and $25^{th}$–$75^{th}$ percentile ranges for irregularly distributed variables. Normality was tested using the Kolmogorov-Smirnov test. The comparisons of groups were based on Student's two-sample t-tests or nonparametric Mann-Whitney tests as appropriate. The categorical data are presented as the absolute and relative frequencies. The differences in proportions between groups were analysed using $\chi^2$ tests. First, to determine the possible predictors of acute total coronary occlusion (baseline TIMI 0), the variables were investigated using univariate analyses. A multivariate logistic regression model was then developed using a directed stepwise approach. All variables with p values < 0.1 were entered into the model. The variables entered into the logistic

Table 1. Baseline characteristics and risk factors

| | TIMI 0 (n = 728) | TIMI 1–3 (n = 2039) | p |
|---|---|---|---|
| Age [years] | 62.6 ± 11.8 | 65.2 ± 11.1 | < 0.0001 |
| Female | 33.38% (243) | 35.65% (727) | 0.27 |
| Hypertension | 65.52% (477) | 73.57% (1500) | < 0.0001 |
| Diabetes mellitus | 20.33% (148) | 26.39% (538) | 0.001 |
| Hyperlipidaemia | 38.05% (277) | 41.98% (856) | 0.06 |
| Current smoking | 30.49% (222) | 25.55% (521) | 0.03 |
| Previous angina | 7.14% (52) | 8.29% (169) | 0.33 |
| Family history CAD | 10.03% (73) | 8.58% (175) | 0.24 |
| Previous stroke | 2.47% (18) | 2.35% (48) | 0.86 |
| Heart failure | 3.16% (23) | 3.24% (66) | 0.92 |
| PVD | 3.02% (22) | 4.12% (84) | 0.18 |
| Renal failure | 4.40% (32) | 6.13% (125) | 0.08 |
| Pulmonary disease | 1.37% (10) | 3.58% (73) | 0.003 |

Data are presented as percentage and number (in brackets) of patients or mean ± standard deviation; CAD — coronary artery disease; PVD — peripheral vascular disease; TIMI — thrombolysis in myocardial infarction

regression model were age, gender, hypertension, diabetes mellitus, current smoking, hypercholesterolaemia, renal failure, pulmonary disease, time to PCI (from onset), typical chest pain, culprit lesion LCx, and LAD. Correlated variables were not entered in the same multivariable model. The factors that met the significance criteria of < 0.05 were retained in the final model. All of the statistical hypotheses were two-sided with a 0.05 type I error rate. The statistical analyses were performed using the SAS statistical package, version 9.2 (SAS Institute Inc., Cary, NC, USA).

## RESULTS

### *Baseline characteristics (medical histories and risk factors)*

A total of 2767 patients were included in the study. The number of patients with preprocedural TIMI 0 (TO group) was 728, and the number with preprocedural TIMI 1–3 was 2039. Table 1 illustrates the comparisons of the risk factors and baseline characteristics of the patients. The patients with TO were younger and more often were smokers, and they had significantly lower incidence of hypertension and diabetes mellitus in comparison to non-TO patients. Patients with pre-PCI TIMI flow grade 1–3 had a significantly higher frequency of pulmonary disease than did those with TIMI 0.

### *Clinical and electrocardiographic findings*

The patients with initial TIMI 0 had shorter delay to angioplasty, lower incidence of T-wave inversion on ECG, lower systolic blood pressure, higher incidence of pulmonary oedema upon admission, and lower ejection fraction compared to the non-TO group. The laboratory findings revealed that the patients with total occlusion had greater leucocytosis and creatine kinase MB isoenzyme (CK-MB) peaks and lower serum creatinine (Table 2).

### *Angiographic findings*

The angiograms revealed TO in 728 (26.3%) patients in the study population. The culprit in the TO group was most often located in the LCx (48.1%), and in the non-TO group it was most often seen in the LAD (38.8%). The patients with initial TIMI 0 had a significantly lower incidence of final TIMI 3 flows in the IRAs compared to the patients with initial TIMI 1–3 (Table 2).

### *Clinical outcomes*

The in-hospital and one-month mortality rates were significantly higher among the TIMI 0 patients (Table 3, Fig. 1). No differences in the death rate were observed at the 6-, 12-, or 36-month follow-ups. There were no statistically significant differences in the in-hospital complications.

### *Clinical factors related to acute total occlusion in the IRAs*

After adjustment for all clinical and angiographic characteristics, a multivariate logistic regression model demonstrated that only LCx as the culprit lesion was an independent predictor of total occlusion in infarct-related coronary artery. Older age, hypertension, pulmonary disease, and culprit lesion in the LAD were independent predictors of baseline TIMI 1–3 flows in the IRAs (Fig. 2).

Table 2. Clinical and angiographic findings on admission

| | TIMI 0 (n = 728) | TIMI 1–3 (n = 2039) | P |
|---|---|---|---|
| Admission-PCI time [min][a] | 95 [40–308] | 113 [50–360] | 0.007 |
| Symptom-PCI time [min][a] | 800 [356–1801] | 900 [375–3040] | 0.003 |
| Ischaemic changes on ECG: | | | |
| ST-segment depression | 44.8% (326) | 45.1% (919) | 0.89 |
| T-wave inversion | 21.0% (153) | 24.7% (504) | 0.04 |
| Sinus rhythm | 94.9% (691) | 94.6% (1929) | 0.75 |
| Atrial fibrillation | 4.3% (31) | 4.9% (99) | 0.51 |
| Heart rate [bmp] | 76.2 ± 11.8 | 76.9 ± 16.4 | 0.33 |
| Systolic BP [mm Hg] | 136.3 ± 24.2 | 140.4 ± 25.0 | 0.0002 |
| Diastolic BP [mm Hg] | 81.7 ± 14.2 | 81.7 ± 13.6 | 0.99 |
| LVEF [%] | 48.9 ± 9.6 | 49.9 ± 9.9 | 0.03 |
| Killip class 3 | 1.8% (13) | 1.5% (31) | 0.02 |
| Killip class 4 | 1.5% (11) | 1.2% (24) | 0.49 |
| GRACE score (points) | 131.0 ± 35.0 | 132.3 ± 33.1 | 0.35 |
| Serum creatinine [mg/dL][a] | 0.95 [0.80–1.13] | 1.0 [0.83–1.19] | 0.008 |
| CK-MB max. [mg/dL][a] | 71 [31.4–151.0] | 34 [17.1–68.5] | < 0.0001 |
| Admission glucose [mg/dL][a] | 114 [99–142] | 112 [96–141] | 0.22 |
| Admission HGB [g/dL] | 14.0 ± 1.6 | 13.9 ± 1.7 | 0.10 |
| Admission WBC [$10^3/\mu L$][a] | 9.7 [7.9–11.7] | 8.7 [7.1–10.8] | < 0.0001 |
| Culprit lesion: | | | |
| LAD | 21.7% (158) | 38.8% (791) | < 0.0001 |
| Cx | 48.1% (350) | 30.7% (627) | < 0.0001 |
| RCA | 29.5% (215) | 28.3% (576) | 0.51 |
| LM | 0.7% (5) | 2.2% (45) | 0.008 |
| Final TIMI 3 flow | 84.5% (615) | 98.1% (2001) | < 0.0001 |
| MVD | 47.7% (347) | 48.2% (982) | 0.82 |

Data are presented as percentage and number (in brackets) of patients or mean ± standard deviation; [a]Median, with 25th, 75th percentiles range in brackets; BP — blood pressure; CK-MB — creatine kinase MB isoenzyme; Cx — circumflex coronary artery; ECG — electrocardiogram; HGB — haemoglobin; LAD — left anterior descending artery; LM — left main coronary artery; LVEF — left ventricular ejection fraction; MVD — multivessel coronary disease; PCI — percutaneous coronary intervention; RCA — right coronary artery; TIMI — thrombolysis in myocardial infarction; WBC — white blood cells

### *Comparison of the occluded anterior vs. infero- or posterolateral culprit lesions*

The number of patients with an occluded anterior culprit lesion was 163 (22.45%), and the number with an infero- or posterolateral lesion was 563 (77.55%). The group with infarct artery-supplied anterior territories had a lower ejection fraction upon admission, a significantly higher incidence of T-wave inversion, and a lower incidence of ST-segment depression on ECG.

There were no differences in the short- or long-term mortalities between the groups (Table 4).

## DISCUSSION

Our study demonstrated the incidence, predictors, and implications of acute total coronary occlusion in patients with NSTEMI. The main findings of the present study were as follows: 1) more than one-fourth of the NSTEMI patients had totally occluded infarct-related coronary arteries; 2) interestingly, the in-hospital and one-month mortalities were significantly higher among the TIMI 0 patients than among the TIMI 1–3 patients, but after 30 days, these differences were no longer observed; and 3) multivariate analysis revealed that only the LCx as the culprit lesion was an independent predictor of total occlusion in the culprit artery.

Table 3. Clinical outcomes

| | TIMI 0 (n = 728) | TIMI 1–3 (n = 2039) | p |
|---|---|---|---|
| In-hospital: | | | |
| Death | 3.98% (29) | 1.72% (35) | 0.0005 |
| Myocardial infarction | 0 (0) | 0.44% (9) | 0.12 |
| Stroke | 0.27% (2) | 0.15% (3) | 0.61 |
| TVR | 0.69% (5) | 0.49% (10) | 0.56 |
| Non-CABG bleeding | 2.61% (19) | 3.04% (62) | 0.55 |
| 1-month death | 5.49% (40) | 3.48% (71) | 0.017 |
| 6-month death | 7.8% (57) | 6.8% (138) | 0.34 |
| 12-month death | 9.3% (68) | 8.8% (180) | 0.68 |
| 24-month death | 12.2% (89) | 11.67% (238) | 0.69 |
| 36-month death | 14.3% (104) | 14.47% (259) | 0.90 |

Data are presented as percentage and number (in brackets) of patients; CABG — coronary artery bypass grafting; TIMI — thrombolysis in myocardial infarction; TVR — target vessel revascularisation

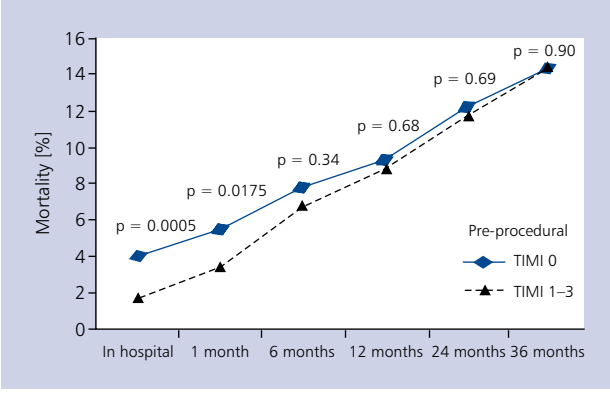


Figure 1. Mortality curves according to preprocedural TIMI flow in the infarct-related artery; TIMI — thrombolysis in myocardial infarction

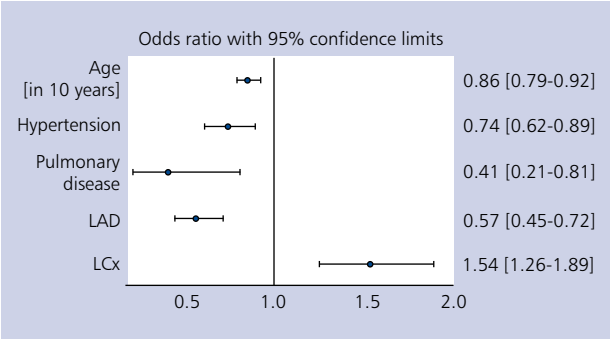


Figure 2. Independent predictors of total occlusion (TIMI 0) in the infarct-related artery; TIMI — thrombolysis in myocardial infarction; LAD — left anterior descending artery; LCx — left circumflex artery

It is well known that early reopening of a completely occluded coronary artery reduces myocardial damage, prevents heart failure, and improves clinical outcome [7, 8]. Acute total occlusion in the IRA causes transmural ischaemia that generates a mean spatial ST vector directed toward the area of the predominant epicardial injury, which results in ST-segment elevation. In contrast, subendocardial ischaemia caused by a transient coronary occlusion or microembolisation with components of a non-occlusive thrombus might result in "electrical silence" or ST-segment depression [3, 9]. Current clinical guidelines take into account this phenomenon and recommend urgent revascularisation only in patients with STEMI [1]. However, the sensitivity of ECG for the identification of complete occlusion in the posterolateral circulation is suboptimal, particularly in cases in which the circumflex artery represents the culprit lesion [10]. In the present study, which was based on a large unselected NSTEMI population, TO was observed in 26.3% of the patients. Our data showed that the NSTEMI group with TO that was treated with percutaneous revascularisation exhibited higher in-hospital and one-month mortality rates than did the non-TO group, and no differences in mortality rates were observed at the follow-ups after more than 30 days. It is likely that the greater short-term mortality among the subjects with total occlusion can be explained by the higher CK-MB peak-assessed infarct size, the effect of which was not weakened by the shorter delay to angioplasty among the patients with TO, and the lack of significant differences in the GRACE risk scores between the groups. The results from previous studies are inconsistent, and the researchers evaluated only short-term outcomes. A study by Bahrmann et al. [11] showed that 29% of patients with NSTEMI presented with acute TO. This population treated

Table 4. Clinical characteristics and outcomes in depending on infarct-related artery localisation

| | Anterior 22.45% (n = 163) | Infero- and posterolateral 77.55% (n = 565) | p |
|---|---|---|---|
| Admission-to-PCI time [min][a] | 112 [40–330] | 91 [40–305] | 0.55 |
| Symptom-to-PCI time [min][a] | 709 [340–1798] | 812.5 [365–1804] | 0.68 |
| Ischaemic changes on ECG: | | | |
| ST-segment depression | 35.6% (58) | 47.4% (268) | 0.007 |
| T-wave inversion | 29.4% (48) | 18.5% (105) | 0.003 |
| LVEF [%] | 47.0 ± 10.7 | 49.5 ± 9.2 | 0.02 |
| Killip class 3 | 1.2% (2) | 1.9% (11) | 0.74 |
| Killip class 4 | 1.8% (3) | 1.4% (8) | 0.72 |
| GRACE score (points) | 131.1 ± 34.6 | 130.9 ± 35.2 | 0.96 |
| Angiographic findings: | | | |
| Final TIMI 3 flow | 81.0% (132) | 85.5% (483) | 0.16 |
| MVD | 45.4% (74) | 48.3% (273) | 0.51 |
| Clinical outcomes: | | | |
| In-hospital death | 3.7% (6) | 4.1% (23) | 0.82 |
| 1-month death | 6.13% (10) | 5.3% (30) | 0.68 |
| 12-month death | 9.8% (16) | 9.2% (52) | 0.81 |
| 24-month death | 14.7% (24) | 11.5% (65) | 0.27 |
| 36-month death | 16.6% (27) | 13.6% (77) | 0.34 |

Data are presented as percentage and number (in brackets) of patients. [a]Median, with 25th, 75th percentiles range in brackets; ECG — electrocardiogram; LVEF — left ventricular ejection fraction; MVD — multivessel coronary disease; PCI — percutaneous coronary intervention; TIMI — thrombolysis in myocardial infarction

with an early invasive strategy had a higher six-month rate of non-fatal reinfarction with no difference in mortality rate in comparison to NSTEMI with non-TO. Wang et al. [12] showed that the culprit artery was occluded in 27% of NSTEMI patients and that these patients had larger infarct size and higher six-month mortality.

In this study, we highlighted the differences in the baseline characteristics and clinical findings between the TO and non-TO groups. Patients with TO were younger, were more often current smokers, and had lower incidence of cardiovascular risk factors, such as hypertension and diabetes mellitus. These results were in accordance with those that have been reported in registries comparing STEMI and NSTEMI populations [13–15]. Our findings may suggest that NSTEMI patients with TO are often misdiagnosed and should be considered as undetected by 12-lead ECG STEMI equivalents. However, it is difficult to compare STEMI and NSTEMI patients who present with TO because of the heterogeneities of these groups. The entire NSTEMI population consists of various groups of patients. The main group are patients with non-completely occluded IRAs and those who only have subendocardial ischaemia [3]. Another group is composed of patients with gradually increasing TO and well-developed collaterals that prevent transmural ischaemia and ST-segment elevation on ECG [16]. There are also NSTEMI patients with acute TO of the infero- and posterolateral circulations due to transmural ischaemia that are not detected by ECG. It is likely that some NSTEMI patients with totally occluded culprit arteries supplying the posterolateral and inferior myocardia share the same pathophysiologies with STEMI patients. This group may be misclassified due to the absence of appropriate precordial ECG leads that could help identify TO in this territory. The question remains whether this misdiagnosis affects the outcomes. In the present analysis, among NSTEMI patients with total occlusion, 77.55% had the culprit localised in the infero- and posterolateral circulation and only 22.45% in the anterior circulation. There were no differences in the short- or long-term mortality rates between these groups.

The median time from symptoms to angioplasty in the TO group was 13.3 h and in the non-TO group was 15 h. Whether the outcome may have been improved with earlier intervention is difficult to evaluate. Current clinical guidelines in NSTEMI do not recommend routine early invasive strategy; the clinical trials did not show any benefit [1]. But there is a lack of data in patients with NSTEMI and TO. Probably, in the case of complete interruption of blood supply, rapid restoration of flow could result in smaller infarct size and better prognosis.

Our study showed that more than one-fourth of the NSTEMI population had occlusive culprit arteries. However, it is difficult to predict TO of the IRA in patients with NSTEMI. In multivariate analysis, only the LCx as the culprit lesion was identified as an independent predictor of TO in the culprit artery. In the present study the LCx constituted about half of cases with TO in the NSTEMI group. It has been shown that the presence of ST segment elevation detects acute occlusions of the LAD and RCA in 70% to 92% of cases, but only in 32% to 48% of cases for LCx-related myocardial infarction [17–19]. Greater attention of physicians should be placed in searching for ST-segment depression in leads V1–V3 as a marker LCx occlusion, especially in patients with ongoing symptoms [20].

Based on possible predictors and the limited sensitivity of surface ECG, it is difficult to identify acute total coronary occlusion in NSTEMI patients. One of the helpful methods might be the use of additional leads. Perron et al. [21] found that the addition of seven inverted leads to the standard 12 positive leads maximally increases the sensitivity of ECG for the detection of acute transmural ischaemia with a minimal loss of specificity. It has also been reported that a technique known as body surface potential mapping improves the detection of occlusions of the culprit arteries in patients without ST-segment elevation on initial 12-lead ECGs [22, 23]. Body surface potential mapping could increase STEMI detection by 27.5% over that obtained with standard ECG [24]. Moreover, transthoracic echocardiography might be helpful, particularly for patients with non-evident changes in ECG and isolated lateral or posterior ischaemia [25]. It has previously been described that hypokinesis or akinesis of the ventricular myocardium appears before the development of ST-segment changes on the ECG [26].

### *Limitations of the study*

Our study had several limitations. The first limitation is the retrospective nature of the analysis. Second, the culprit arteries were determined by cardiologists in the catheterisation laboratories (using electrocardiographic, angiographic, and echocardiographic findings), and the selection of the IRAs among the patients with multivessel disease might have differed between operators. Patients with previous myocardial infarctions, PCIs, and CABGs were excluded from the study because the culprit vessel in these subjects might have been incorrectly interpreted due to chronic TO. Furthermore, there was a lack of data about detailed angiographic features, such as the presence or absence of collateral circulation in the coronary angiograms.

## CONCLUSIONS

More than one-fourth of the NSTEMI patients included in this study had a totally occluded infarct-related coronary artery. These patients had higher in-hospital and one-month mortalities, but their long-term outcomes were similar to those of the non-TO group. It was difficult to distinguish the TO group of patients; only LCx as the culprit lesion was identified as an independent predictor of complete artery occlusion.

### *Acknowledgements*

We thank all of the physicians and nurses who participated in the PL-ACS and the members of the Expert Committee and the staff members of the National Health Fund of Poland for their logistic support. The Polish Registry of Acute Coronary Syndrome is supported by an unrestricted grant from the Polish Ministry of Health, Warsaw, Poland.

***Conflict of interest:*** *none declared*

### *References*

1. Hamm CW, Bassand JP, Agewall S, et al. ESC Committee for Practice Guidelines. ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation: The Task Force for the management of acute coronary syndromes (ACS) in patients presenting without persistent ST-segment elevation of the European Society of Cardiology (ESC). Eur Heart J. 2011; 32(23): 2999–3054, doi: 10.1093/eurheartj/ehr236, indexed in Pubmed: 21873419.
2. Steg PhG, James SK, Atar D, et al. Task Force on the management of ST-segment elevation acute myocardial infarction of the European Society of Cardiology (ESC). ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. Eur Heart J. 2012; 33(20): 2569–2619, doi: 10.1093/eurheartj/ehs215, indexed in Pubmed: 22922416.
3. Davies MJ. Pathophysiology of acute coronary syndromes. Indian Heart J. 2000; 52(4): 473–479, doi: 10.1201/b16507-16, indexed in Pubmed:11084798.
4. Wagner G, Lim T, Gettes L, et al. Consideration of pitfalls in and omissions from the current ECG standards for diagnosis of myocardial ischemia/infarction in patients who have acute coronary syndromes. Cardiol Clin. 2006; 24(3): 331–42, vii, doi: 10.1016/j.ccl.2006.04.012, indexed in Pubmed: 16939827.
5. Poloński L, Gasior M, Gierlotka M, et al. Polish Registry of Acute Coronary Syndromes (PL-ACS). Characteristics, treatments and outcomes of patients with acute coronary syndromes in Poland. Kardiol Pol. 2007; 65(8): 861–873, indexed in Pubmed: 17853315.
6. Flynn MR, Barrett C, Cosío FG, et al. The Cardiology Audit and Registration Data Standards (CARDS), European data standards for clinical cardiology practice. Eur Heart J. 2005; 26(3): 308–313, doi: 10.1093/eurheartj/ehi079, indexed in Pubmed: 15618029.
7. Baigent C, Collins R, Appleby P, et al. ISIS-2: 10 year survival among patients with suspected acute myocardial infarction in randomised comparison of intravenous streptokinase, oral aspirin, both, or neither. The ISIS-2 (Second International Study of Infarct Survival) Collaborative Group. BMJ. 1998; 316(7141): 1337–1343, doi: 10.1136/bmj.316.7141.1337, indexed in Pubmed: 9563981.
8. Franzosi MG, Santoro E, De Vita C, et al. Ten-year follow-up of the first megatrial testing thrombolytic therapy in patients with acute myocardial infarction: results of the Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto-1 study. The GISSI Investigators. Circulation. 1998; 98(24): 2659–2665, doi: 10.1161/01.cir.98.24.2659, indexed in Pubmed: 9851950.
9. Hurst JW. Thoughts about the abnormalities in the electrocardiogram of patients with acute myocardial infarction with emphasis on a more accurate method of interpreting ST-segment displacement: part I. Clin Cardiol. 2007; 30(8): 381–390, doi: 10.1002/clc.20088, indexed in Pubmed: 17680618.

10. Krishnaswamy A, Lincoff AM, Menon V. Magnitude and consequences of missing the acute infarct-related circumflex artery. Am Heart J. 2009; 158(5): 706–712, doi: 10.1016/j.ahj.2009.08.024, indexed in Pubmed: 19853686.
11. Bahrmann P, Rach J, Desch S, et al. Incidence and distribution of occluded culprit arteries and impact of coronary collaterals on outcome in patients with non-ST-segment elevation myocardial infarction and early invasive treatment strategy. Clin Res Cardiol. 2011; 100(5): 457–467, doi:10.1007/s00392-010-0269-9, indexed in Pubmed: 21165625.
12. Wang TY, Zhang M, Fu Y, et al. Incidence, distribution, and prognostic impact of occluded culprit arteries among patients with non-ST-elevation acute coronary syndromes undergoing diagnostic angiography. Am Heart J. 2009; 157(4): 716–723, doi: 10.1016/j.ahj.2009.01.004, indexed in Pubmed:19332201.
13. Abbott JD, Ahmed HN, Vlachos HA, et al. Comparison of outcome in patients with ST-elevation versus non-ST-elevation acute myocardial infarction treated with percutaneous coronary intervention (from the National Heart, Lung, and Blood Institute Dynamic Registry). Am J Cardiol. 2007; 100(2): 190–195, doi: 10.1016/j.amjcard.2007.02.083, indexed in Pubmed: 17631068.
14. Polonski L, Gasior M, Gierlotka M, et al. PL-ACS Registry Pilot Group. A comparison of ST elevation versus non-ST elevation myocardial infarction outcomes in a large registry database: are non-ST myocardial infarctions associated with worse long-term prognoses? Int J Cardiol. 2011; 152(1): 70–77, doi: 10.1016/j.ijcard.2010.07.008, indexed in Pubmed: 20684999.
15. Goldberg RJ, Currie K, White K, et al. Six-month outcomes in a multinational registry of patients hospitalized with an acute coronary syndrome (the Global Registry of Acute Coronary Events [GRACE]). Am J Cardiol. 2004; 93(3): 288–293, doi: 10.1016/j.amjcard.2003.10.006, indexed in Pubmed:14759376.
16. Antman EM, Braunwald E. Acute myocardial infarction. In: Braunwald E, Zipes DP, Libby P, eds. Heart disease: A Textbook of Cardiovascular Medicine. 6th Ed. Philadelphia, PA: WB Saunders Co Ltd; 2001: 1114–1251.
17. Huey BL, Beller GA, Kaiser DL, et al. A comprehensive analysis of myocardial infarction due to left circumflex artery occlusion: comparison with infarction due to right coronary artery and left anterior descending artery occlusion. J Am Coll Cardiol. 1988; 12(5): 1156–1166, doi: 10.1016/0735-1097(88)92594-6, indexed in Pubmed: 3170958.
18. Schmitt C, Lehmann G, Schmieder S, et al. Diagnosis of acute myocardial infarction in angiographically Documented Occluded Infarct Vessel. Chest, 2001; 120: 1540–1546.
19. Berry C, Zalewski A, Kovach R, et al. Surface electrocardiogram in the detection of transmural myocardial ischemia during coronary artery occlusion. Am J Cardiol. 1989; 63(1): 21–26, doi: 10.1016/0002-9149(89)91069-2, indexed in Pubmed: 2521194.
20. Thygesen K, Alpert JS, Jaffe AS, et al. Writing Group on the Joint ESC/ACCF/AHA/WHF Task Force for the Universal Definition of Myocardial Infarction, ESC Committee for Practice Guidelines (CPG). Third universal definition of myocardial infarction. Eur Heart J. 2012; 33(20): 2551–2567, doi:10.1093/eurheartj/ehs184, indexed in Pubmed: 22922414.
21. Perron A, Lim T, Pahlm-Webb U, et al. Maximal increase in sensitivity with minimal loss of specificity for diagnosis of acute coronary occlusion achieved by sequentially adding leads from the 24-lead electrocardiogram to the orderly sequenced 12-lead electrocardiogram. J Electrocardiol. 2007; 40(6): 463–469, doi: 10.1016/j.jelectrocard.2007.07.002, indexed in Pubmed: 17993301.
22. Daly Mj, Finlay Dd, Guldenring D, et al. Detection of acute coronary occlusion in patients with acute coronary syndromes presenting with isolated ST-segment depression. Eur Heart J Acute Cardiovasc Care. 2012; 1(2): 128–135, doi: 10.1177/2048872612448977, indexed in Pubmed: 24062900.
23. Owens C, McClelland A, Walsh S, et al. Comparison of value of leads from body surface maps to 12-lead electrocardiogram for diagnosis of acute myocardial infarction. Am J Cardiol. 2008; 102(3): 257–265, doi: 10.1016/j.amjcard.2008.03.046, indexed in Pubmed: 18638583.
24. Hoekstra JW, O'Neill BJ, Pride YB, et al. Acute detection of ST-elevation myocardial infarction missed on standard 12-Lead ECG with a novel 80-lead real-time digital body surface map: primary results from the multicenter OCCULT MI trial. Ann Emerg Med. 2009; 54(6): 779–788.e1, doi: 10.1016/j.annemergmed.2009.06.525, indexed in Pubmed: 19766352.
25. Corya BC. Applications of echocardiography in acute myocardial infarction. Cardiovasc Clin. 1975; 7(2): 113–127, doi: 10.4324/9780203414699_chapter_14, indexed in Pubmed: 1104166.
26. Oh JK, Seward JB, Tajik AJ. The Echo Manual. New Delhi, India: Wolters Kluwer Health; 2006.


**Cite this article as:** Karwowski J, Poloński L, Gierlotka M, et al. Total coronary occlusion of infarct-related arteries in patients with non-ST-elevation myocardial infarction undergoing percutaneous coronary revascularisation. Kardiol Pol. 2017; 75(2): 108–116, doi: 10.5603/KP.a2016.0130.

# Całkowita okluzja tętnicy odpowiedzialnej za zawał wśród pacjentów z zawałem serca bez uniesienia odcinka ST leczonych przezskórną angioplastyką wieńcową

Jarosław Karwowski[1], Lech Poloński[2], Marek Gierlotka[2], Andrzej Ciszewski[3], Michał Hawranek[2], Maciej Bęćkowski[1], Mariusz Gąsior[2], Ilona Kowalik[1], Hanna Szwed[1]

[1]II Klinika Choroby Wieńcowej, Instytut Kardiologii, Warszawa
[2]III Klinika Kardiologii, Śląskie Centrum Chorób Serca, Zabrze
[3]Oddział Kardiologii Inwazyjnej, Instytut Kardiologii, Warszawa

## Streszczenie

**Wstęp:** Występowanie i wpływ całkowitej okluzji tętnicy odpowiedzialnej za zawał (IRA) na rokowanie chorych z zawałem serca bez uniesienia odcinka ST (NSTEMI) pozostają niejasne.

**Cel:** Celem pracy była ocena znaczenia klinicznego ostrej całkowitej okluzji tętnicy wieńcowej wśród pacjentów z NSTEMI.

**Metody:** Przeanalizowano dane o 2767 pacjentach z NSTEMI włączonych do Ogólnopolskiego Rejestru Ostrych Zespołów Wieńcowych. Chorzy zostali podzieleni na dwie grupy wg przedproceduralnego przepływu w IRA, określonego w skali TIMI flow (TIMI flow 0 — całkowita okluzja [TO]: 728; 26,3% pacjentów i TIMI flow 1–3 — niecałkowita okluzja [non-TO]: 2039; 73,7% pacjentów).

**Wyniki:** Pacjenci z przedproceduralnym TIMI flow 0 byli młodsi, rzadziej występowało u nich nadciśnienie tętnicze, cukrzyca i choroby płuc, częściej byli aktualnymi palaczami. Gałąź okalająca lewej tętnicy wieńcowej (LCx) była główną IRA w grupie TO (48,1%), podczas gdy gałąź przednia zstępująca lewej tętnicy wieńcowej (LAD) była najczęściej występującą IRA w grupie non-TO (38,8%). Analiza wieloczynnikowa wykazała, że zwężenie w LCx było niezależnym predyktorem przepływu TIMI 0 w IRA (OR ± 95 CI: 1,53 [1,21–1,93], $p < 0,0001$). Śmiertelność wewnątrzszpitalna i 30-dniowa były wyższe w grupie TO (4,0% vs. 1,7%; $p = 0,0005$ i 5,5% vs. 3,5%; $p = 0,0175$, odpowiednio), nie zaobserwowano różnic w zakresie śmiertelności rocznej, 2- i 3-letniej pomiędzy grupami.

**Wnioski:** Tętnica okalająca jako IRA jest jedynym niezależnym predyktorem całkowitej okluzji wśród chorych z NSTEMI. Pacjenci z NSTEMI z całkowitą okluzją charakteryzują się wyższą śmiertelnością wewnątrzszpitalną i 30-dniową, jednak ich rokowanie długoterminowe jest porównywalne do pacjentów bez całkowitej okluzji.

**Słowa kluczowe:** całkowita okluzja tętnicy odpowiedzialnej za zawał, przezskórna angioplastyka wieńcowa, zawał serca bez uniesienia odcinka ST, śmiertelność długoterminowa

Kardiol Pol 2017; 75, 2: 108–116

**Adres do korespondencji:**
lek. Jarosław Karwowski, II Klinika Choroby Wieńcowej, Instytut Kardiologii, ul. Spartańska 1, 02–637 Warszawa, tel: +48 502913169, faks: +48 22 844 95 10, e-mail: karwowski.jarek@gmail.com
**Praca wpłynęła:** 04.03.2016 r. **Zaakceptowana do druku:** 13.07.2016 r. **Data publikacji AoP:** 22.08.2016 r.