Predictors and mid-term outcomes of nosocomial infection in ST-elevation myocardial infarction patients treated by primary angioplasty

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ABSTRACT

Background: Nosocomial infections (NI) are associated with high morbidity and mortality. Existing data on the impact of NI on patients with ST-elevation myocardial infarction (STEMI) is scarce.

Aim: Our aim was to determine the incidence, predictors, and prognosis of NI in a contemporary series of STEMI patients.

Methods: 1131 consecutive STEMI patients treated by primary percutaneous coronary intervention from January 2008 to December 2017 were analyzed. Binary logistic regression and Cox proportional hazard models were used to identify predictors of NI and major adverse cardio-cerebrovascular events (MACCE) at 1-year follow-up, respectively.

Results: Of all patients, 126 (11.1%) were diagnosed with NI (>48 hours from admission), mostly of respiratory (50.8%) and urinary (39.7%) tract origin. Insulin-treated diabetics were 3-fold more likely to develop NI. Other independent predictors were peripheral arterial disease, intra-aortic balloon pump insertion, age, lower systolic blood pressure, and higher peak creatine-kinase. Only pre-infarction angina was negatively related to NI. Age, peripheral arterial disease, femoral approach and larger infarct were related to MACCE at 1-year follow-up. NI in isolation was not independently related to MACCE (hazard ratio [HR], 1.24; 95% confidence interval [CI], 0.80–1.94; $P = 0.34$). However, we found a significant interaction between NI and smoking (HR, 2.33; 95% CI, 1.03–5.24; $P_{interc} = 0.04$).

Conclusion: Larger infarct size, hemodynamic instability, and co-morbidities were related to both NI and 1-year adverse events. Smokers who developed NI also had a higher 1-year risk of MACCE.

Key words: cross infection, myocardial infarction, outcomes, smoking ST-elevation myocardial infarction

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INTRODUCTION

The advent of reperfusion therapy, namely by percutaneous coronary intervention (PCI), has been critical for the decreased mortality of patients presenting with ST-elevation myocardial infarction (STEMI). However, in-hospital and mid-term adverse outcomes range from as low as 1% to more than 30% [1], emphasizing the need to identify and treat clinical features that may negatively impact patients' prognoses. In previous studies, nosocomial infections (NI) in STEMI patients have been related to higher mortality and longer hospital stay, along with higher health care costs [2, 3]. Mechanisms behind the adverse events of patients with STEMI patients complicated by NI may include the myocardial infarction-related inflammatory state, which might predispose to the development of sepsis, as well as the pro-thrombotic milieu induced by inflammation [4, 5].

Data about the incidence and impact of NI on prognosis following STEMI are scarce and vary according to definitions and the studied population. The reported incidence of infection in a recent octogenarian cohort undergoing primary PCI was nearly 30% [6], whereas another study found that 2.4% of STEMI patients included in a randomized trial developed a serious infection [2].

WHAT'S NEW?

Our study demonstrated that nosocomial infection is a relatively common complication of ST-elevation myocardial infarction, affecting more than 10% of the patients. Nosocomial infection was predicted by infarct size, hemodynamic instability, and co-morbidities. Pre-infarction angina was the only protective feature identified. Regarding nosocomial infection's impact at 1-year follow-up, we concluded that it does not constitute an independent predictor of major adverse cardio-cerebrovascular events (MACCE). However, smokers who complicate with nosocomial infection experience a higher 1-year MACCE incidence. Our study indicates that a continuous effort to treat STEMI patients early and to limit infarct size seems to be an effective way to prevent post-reperfusion nosocomial infections.

In this study, we aimed to address the prevalence and predictors of NI in a series of STEMI patients treated by PCI in a tertiary care center and to ascertain its impact on the incidence of major adverse cardio-cerebrovascular events (MACCE) at 1-year follow-up.

METHODS

Studied population and definitions

We conducted a retrospective study including consecutive adults (≥18 years old) with a diagnosis of STEMI treated with primary PCI, in a tertiary care center between January 1, 2008 and December 31, 2017. Considering our focus on NI, patients with a diagnosis of overt infection at the time of admission or <48 hours from admission were excluded, to assure that all infections included developed in the hospital setting and were not present at the time of admission [7].

All STEMI patients entered an anonymized prospective database which included demographic, clinical, and procedural characteristics. Data were obtained by medical chart review. According to the 4th Universal Definition of Myocardial Infarction, STEMI was defined as typical chest discomfort or other ischemic symptoms, associated with new ST-segment elevations in two contiguous leads or new bundle branch blocks with ischemic repolarization patterns. The ST-elevation cutpoints (measured at the J-point) were considered as follows: in leads V2–V3 ≥2 mm in men ≥40 years; ≥2.5 mm in men <40 years; ≥1.5 mm in women regardless of age, and ≥1 mm in all the other leads [8]. In addition, to be included in this study, all patients were required to have a culprit lesion identified and to have undergone PCI. Patients' treatment strategy followed per current guidelines [9].

NI was defined as an infection diagnosed 48 hours after hospital admission requiring antibiotics, which reflect infection's clinical impact with the need for specific treatment. Infection sites were grouped into the following main categories: "respiratory tract infection", "urinary tract infection", "catheter-related infection" and "other". Respiratory tract infection comprised both tracheobronchitis and pneumonia. Identification of a pathogen was not mandatory for diagnosis but was collected whenever possible. Urinary tract infection was defined in the presence of signs and symptoms and $>10^5$ CFU/ml on urine culture. A catheter-related infection required a positive tip culture and documentation of the same organism on peripheral blood. The "other" category included additional infection types in accordance with the Centers for Disease Control and Prevention/National Healthcare Safety Network (CDC/NHSN) criteria, that did not comprise enough patients to permit a separate category [7].

Clinical and demographic characteristics are detailed in Table 1. Pre-infarct angina (PIA) was diagnosed if a patient had arm, jaw, or chest pain in the preceding eight days before the diagnosis of STEMI. Total ischemic time and door-to-balloon time were the time elapsed from symptom onset (the time when chest pain became more intense and sustained) and presentation to the hospital or the passage of the coronary guidewire, respectively. Peripheral arterial disease (PAD) was considered if the patient had peripheral claudication and established aorto-iliac or peripheral disease.

Clinical follow-up was performed by record-linkage and ascertained by electronic records to check for the occurrence of a MACCE comprising death (any cause), a cerebrovascular accident (brain imaging was mandatory), new myocardial infarction in any vessel, or target lesion revascularization (TLR — new intervention on target lesion due to angina or ischemia), during the first year after the index STEMI. Patients having any of the aforementioned MACCE were censored. The study was approved by the hospital ethics committee (2019.128[108-DEFI/112-CE]), and the informed consent for the studied cohort was waived due to the retrospective nature of the analysis. The database was anonymized.

Statistical analysis

Categorical variables are expressed as absolute values and percentages, comparison was performed by Pearson chi-square or Fisher exact test, as appropriate. Continuous data are expressed as the median and interquartile range (IQR) and were compared using the Mann-Whitney U test. Normality of distribution was assessed from visual inspection of histograms and the Shapiro-Wilk test.

MACCE rates were plotted as Kaplan-Meier curves, and groups were compared using the log-rank test.

To identify the independent predictors of NI we ran a stepwise multivariable logistic regression that included variables with a P <0.1 in the univariable analysis. Cox proportional hazard models were used to identify predictors

Table 1. Baseline characteristics of STEMI patients

	All patients (n = 1131)	Infection (n = 126)	No infection (n = 1005)	*P*-value
Age, years, median (IQR)	62.0 (53.0–72.0)	70.0 (62.0–80.3)	61.0 (52.5–71.0)	<0.001
Men, n (%)	839 (74.2)	78 (75.7)	761 (61.9)	0.001
Pre-infarction angina, n (%)	356 (31.6)	24 (19.4)	332 (33.2)	0.002
BMI, kg/m^2, median (IQR)	26.0 (23.9–28.4)	26.0 (24.0–28.0)	26.0 (23.9–28.5)	0.74
Medical history				
Hypertension, n (%)	626 (55.6)	81 (64.8)	545 (54.4)	0.03
Dyslipidemia, n (%)	600 (53.3)	25 (53.2)	533 (53.6)	0.94
Peripheral arterial disease, n (%)	100 (8.9)	25 (11.0)	75(7.5)	<0.001
Smoker, n (%)	564 (50.0)	52 (41.6)	512 (51.1)	0.045
History of CABG, n (%)	15 (1.3)	2 (1.6)	13 (1.3)	0.68[a]
History of MI, n (%)	87 (7.8)	11 (8.9)	76 (7.6)	0.62
Diabetes mellitus				<0.001
No, n (%)	847 (76.0)	79 (63.7)	768 (77.5)	
Yes, without insulin, n (%)	221 (19.8)	29 (23.4)	192 (19.4)	
Yes, with insulin, n (%)	47 (4.2)	16 (12.9)	31 (3.1)	
Total ischemic time, hours, median (IQR)	4.0 (2.5–7.8)	4.0 (2.5–9)	4.0 (2.5–7.71)	0.76
Door-to-balloon time, hours, median (IQR)	1.3 (0.8–2.0)	1.5 (1.0–2.5)	1.3 (0.8–2.0)	0.11
Creatinine clearance, ml/min, median (IQR)	84.0 (60.1–110.0)	60.0 (42.7–85.0)	87.0 (64.0–111.4)	<0.001
Hemoglobin at admission, g/dl, median (IQR)	14.2 (12.9–15.2)	13.5 (12.0–14.9)	14.30 (13.0–15.3)	<0.001
Systolic pressure, mm Hg, median (IQR)	120 (103–136)	101(90–128)	120 (105–137)	<0.001
Staged PCI, n (%)	205 (18.3)	14 (11.4)	191 (19.2)	0.04
Killip class				<0.001
1, n (%)	845 (75.3)	53 (43.4)	792 (79.2)	
2, n (%)	122 (10.9)	13 (10.7)	109 (10.9)	
3, n (%)	38 (3.4)	9 (7.4)	29 (2.9)	
4, n (%)	117 (10.4)	47 (38.5)	70 (7.0)	
LAD, n (%)	477 (42.2)	50 (39.7)	427 (42.6)	0.54
TIMI score, median (IQR)	3 (2–5)	6 (4–8)	3 (2–5)	<0.001
Peak CK, U/l, median (IQR)	1667 (904–3017)	1972 (1010– 3812)	1649 (893–2914)	0.06
Radial approach, n (%)	772 (68.6)	71 (57.3)	701 (70.0)	0.004
Glycoprotein IIb/IIIa inhibitors, n (%)	236 (21.1)	26 (26.1)	210 (21.1)	0.98
IABP insertion, n (%)	31 (2.8)	9 (7.1)	22 (2.2)	0.005[a]
Length of hospital stay, days, median (IQR)	6 (5–8)	12 (7–20)	6 (5–7)	<0.001

[a]Fisher's exact test.

Abbreviations: BMI, body mass index; CABG, coronary artery bypass grafting; CK, creatine-kinase; IABP, intra-aortic balloon pump; IQR interquartile range; LAD, left anterior descending artery; MI, myocardial infarction

of MACCE during the follow-up, variables with a P <0.1 on univariable analyses were included in multivariable equations. The presence of possible interactions between NI and all the other variables was tested. Statistical analysis was conducted using Statistical Package for Social Sciences (SPSS version 25.0) and a two-tailed P <0.05 was considered significant for all tests.

RESULTS

From January 2008 to December 2017, of the 1150 STEMI consecutive patients screened, 12 were excluded for presenting an infection at the time of admission and 7 for developing an infection <48 hours after admission. From the 1131 patients included in the study, 126 (11.1%) developed a NI, mostly of respiratory (50.8%) and urinary (39.7%) tract origin (Figure 1). The median time until the diagnosis of NI was 3 days (IQR, 2–6).

Patients who developed a NI were older, more often men, non-smokers, and had more comorbidities. They also had lower hemoglobin, lower creatinine clearance, and lower systolic blood pressure on admission, as well as a higher Killip class during the hospital stay and a higher TIMI score for STEMI on arrival (Table 1). NI was also related

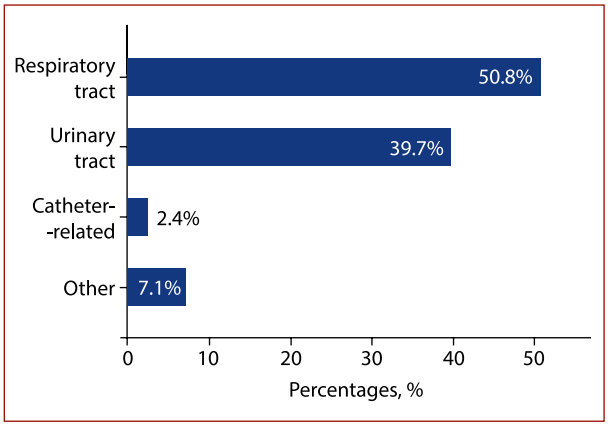

Figure 1. Nosocomial infection site

Table 2. Predictors of nosocomial infection during hospitalization

	Univariable		Multivariable	
	OR (95% CI)	*P*-value	OR (95% CI)	*P*-value
Age, years	1.05 (1.03–1.07)	<0.001	1.05 (1.02–1.07)	<0.001
Men vs women	0.52 (0.35–0.77)	0.001	0.68 (0.41–1.13)	0.14
Pre-infarction angina (yes vs no)	0.48 (0.30–0.77)	0.002	0.56 (0.33–0.95)	0.03
BMI, kg/m^2	0.98 (0.93–1.04)	0.51		
Medical history (yes vs no)				
Hypertension	1.54(1.05–2.27)	0.03	1.19 (0.73–1.94)	0.49
Dyslipidemia	1.01 (0.70–1.47)	0.94		
PAD	3.12 (1.90–2.14)	<0.001	2.74 (1.52–4.95)	0.001
Smoker	0.68 (0.47–0.99)	0.046	1.72 (0.99–3.00)	0.06
History of CABG	1.23 (0.28–5.52)	0.79		
History of MI	1.19 (0.61–2.31)	0.61		
Diabetes (vs no)				
Yes, without insulin	1.47 (0.93–2.31)	0.10	1.25 (0.73–2.15)	0.41
Yes, with insulin	5.02 (2.63–9.58)	<0.001	3.40 (1.53–7.56)	0.003
Total ischemic time, hours	1.01 (0.99–1.03)	0.38		
Door-to-balloon time, hours	1.04 (0.98–1.09)	0.25		
Creatinine clearance, ml/min	0.98 (0.97–0.99)	<0.001	0.99 (0.98–1.00)	0.08
Hemoglobin at admission, g/dl	0.82(0.74–0.90)	<0.001	0.99 (0.86–1.13)	0.83
Systolic pressure, mm Hg	0.99(0.98–0.99)	<0.001	0.99 (0.98–1.00)	0.002
Staged PCI (yes vs no)	0.54 (0.30–0.96)	0.04	0.54 (0.27–1.08)	0.08
LAD vs Non-LAD	0.89 (0.61–1.30)	0.54		
Peak CK, U/l ×10^3	1.10 (1.02–1.18)	0.01	1.12 (1.03–1.22)	0.01
Femoral vs Radial Approach	1.74 (1.19–2.54)	0.004	1.27 (0.81–2.01)	0.30
Glikoprotein IIb/IIIa inhibitors (yes vs no)	0.99 (0.62–1.57)	0.98		
IABP insertion (yes vs no)	3.42 (1.54–7.59)	0.003	3.09 (1.12–8.47)	0.03

Abbreviations: CI, confidence interval; OR, odds ratio; PAD, peripheral arterial disease; PCI, percutaneous coronary intervention. Other — see Table 1

to the use of an intra-aortic balloon pump (IABP), whereas utilization of a radial approach and staged PCI for multivessel coronary disease were more prevalent in patients without NI. Length of hospital stay was significantly longer in patients with NI (median 6 vs 12 days). There were no other significant differences between groups.

Predictors of NI

Fourteen variables were eligible for multivariable analysis, as shown in Table 2. Diabetic patients on insulin therapy were approximately 3 times more likely to develop in-hospital infection (odds ratio [OR], 3.40; 95% confidence interval [CI], 1.53–7.56; $P = 0.003$), however, this association was not significant for non-insulin treated diabetes (OR, 1.25; 95% CI, 0.73–2.15; $P = 0.41$). Other predictors of NI were PAD (OR, 2.74; 95% CI, 1.52–4.95; $P = 0.001$) and the need for an IABP (OR, 3.09; 95% CI, 1.12–8.47; $P = 0.03$). NI was also statistically more prevalent in older patients (OR, 1.05 per year of age; 95% CI, 1.02–1.07; $P < 0.001$), those with lower systolic blood pressure on admission (OR, 0.99 per mm Hg rise; 95% CI, 0.98–1.00, $P = 0.002$) and those who had a higher peak creatine-kinase (CK) activity (OR, 1.12 per unit rise; 95% CI, 1.03–1.22; $P = 0.01$). On the contrary, PIA was negatively related to NI (OR, 0.56; 95% CI, 0.33–0.95; $P = 0.03$).

Impact of NI on outcomes

As observed on the Kaplan-Meier survival curve (Figure 2), in a 1-year follow-up, the occurrence of MACCE was more than twice as common in the NI group: 47 (37.3%) vs 193 (14.6%), log-rank $P < 0.001$, driven by a larger difference in the first month after STEMI. The statistically significant difference between patients with and without NI is consistent for all composite events of MACCE, except target lesion revascularization (Supplementary material).

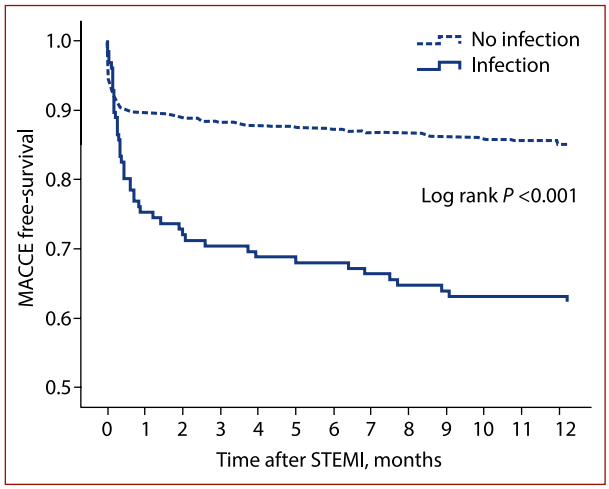

Figure 2. Kaplan-Meier survival curve showing the probability of a STEMI patient to remain free of a MACCE event according to nosocomial infection

Table 3 shows the proportional hazard Cox analysis for predictors of MACCE at 1-year follow-up. The strongest MACCE predictor was PAD (hazard ratio [HR], 3.16; 95%

Table 3. Predictors of MACCE at 1-year follow-up

	Univariable		Multivariable (without interaction)	
	HR (95% CI)	*P*-value	HR (95% CI)	*P*-value
Age, years	1.04 (1.03–1.05)	<0.001	1.02 (1.00–1.04)	0.04
Men vs women	0.67 (0.50–0.91)	0.009	1.15 (0.76–1.73)	0.52
Pre-infarction angina (yes vs no)	0.62 (0.44–0.86)	0.005	0.83 (0.56–1.23)	0.35
BMI, kg/m^2	0.98 (0.94–1.02)	0.34		
Medical history (yes vs no)				
Hypertension	1.82 (1.34–2.48)	<0.001	1.13 (0.76–1.68)	0.54
Dyslipidemia	0.82 (0.62–1.09)	0.17		
PAD	4.46 (3.23–6.15)	<0.001	3.16 (2.05–4.87)	<0.001
Smoker	0.55 (0.41–0.74)	<0.001	1.01 (0.65–1.56)	0.98
History of CABG	1.66 (0.62–4.46)	0.32		
History of MI	1.72 (1.11–2.66)	0.02	1.20 (0.69–2.07)	0.52
Diabetes (vs no)				
Yes, without insulin	1.36 (0.96–1.92)	0.09	1.16 (0.76–1.77)	0.49
Yes, with insulin	3.21 (1.98–5.20)	<0.001	1.23 (0.63–2.40)	0.54
Total ischemic time, hours	1.02 (1.01–1.03)	0.008	1.01 (0.99–1.04)	0.81
Door-to-balloon time, hours	1.04 (1.00–1.08)	0.07	1.03 (0.97–1.07)	0.32
Creatinine clearance, ml/min	0.98 (0.97–0.98)	<0.001	1.00 (0.99–1.00)	0.27
Hemoglobin at admission, g/dl	0.76 (0.71–0.82)	<0.001	0.85 (0.77–0.94)	0.002
Systolic pressure, mm Hg	0.98 (0.97–0.99)	<0.001	0.99 (0.98–1.00)	0.003
Staged PCI (yes vs no)	0.57 (0.40–0.90)	0.02	0.65 (0.38–1.10)	0.11
LAD vs Non-LAD	0.81 (0.60–1.08)	0.15		
Peak CK, U/l ×10^3	1.08 (1.02–1.15)	0.02	1.11 (1.04–1.19)	0.002
Femoral vs radial approach	3.18 (2.39–4.24)	<0.001	1.85 (1.30–2.64)	0.001
Glikoprotein IIb/IIIa inhibitors (yes vs no)	0.85 (0.59–1.23)	0.40		
IABP insertion (yes vs no)	4.04 (2.38–6.86)	<0.001	1.38 (0.62–3.09)	0.44
Nosocomial infection	2.73 (1.96–3.79)	<0.001	1.24 (0.80–1.94)	0.34

Abbreviations: see Table 1 and 2

CI, 2.05–4.87; P <0.001). Age (HR, 1.02; 95% CI, 1.00–1.04; P = 0.04), lower hemoglobin concentration (HR, 0.85; 95% CI, 0.77–0.94; P = 0.002), lower systolic blood pressure on admission (HR, 0.99; 95% CI, 0.98–1.00; P = 0.003), a higher peak CK activity (HR, 1.11; 95% CI, 1.04–1.19; P = 0.002), and the utilization of a femoral approach (HR, 1.85; 95% CI, 1.30–2.64; P = 0.001) were also found to be independent predictors of MACCE. NI was not found to be an independent predictor of MACCE (HR, 1.24; 95% CI, 0.80–1.94; P = 0.34). An interaction between NI and smoking was identified (HR, 2.33; 95% CI, 1.03–5.24; P_{interc} = 0.04). No more interactions were found between NI and other plausible variables. Furthermore, interaction with smoking was not significant when the infection site was considered (P_{interc} = 0.29). As seen in Figure 3, dividing patients into four groups according to the presence of NI and smoking habits, a significant difference between the incidence of MACCE at 1-year follow-up was observed (P <0.001), with smokers who have a NI being the most affected group (42.3%).

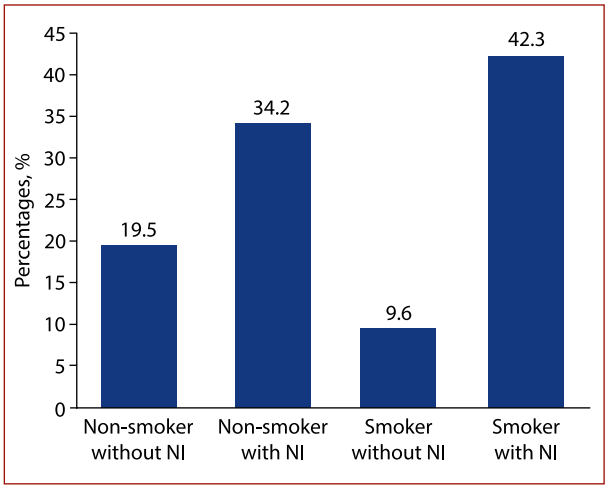

Figure 3. Incidence of MACCE at 1-year follow up according to the presence of nosocomial infection (NI) or smoking habits

DISCUSSION

Our study reveals that 11.1% of STEMI patients had a NI during the hospital stay, a prevalence lower than reported by some studies in mixed populations undergoing PCI (from 16% to nearly 30%) [6, 10, 11], but higher than others (from 2.4 to 5%) [2, 3, 12]. This may reflect the inhomogeneous definitions of hospital-acquired infection and various indications for PCI (from stable disease to STEMI).

Consistent with most studies [6, 10, 12], pulmonary and urinary tract infections were the most frequent NI site. Even though primary PCI carries vascular invasiveness, the incidence of bloodstream infections was low. As expected, we observed a prolonged hospital stay in infected patients compared to the non-infected group.

Age, diabetes on insulin therapy, PAD, insertion of an IABP, low blood pressure, and high peak CK were identified as factors that favor infection. This comes as no surprise, since it may reflect patients with larger infarcts, requiring more invasiveness, as well as those who are more prone to infections (the elderly, the diabetics, and those with established PAD). Insulin therapy most likely works as a marker of diabetes progression, signaling patients with aggravated immune, vascular, and neurological dysfunction, rather than representing a direct result of prior antidiabetic therapy on NI risk [13–15].

The insertion of an IABP can understandably lead to an increase in bacteremia, wound, respiratory, and urinary tract infections as these patients frequently require a prolonged stay in intensive care units. IABP's complications have a considerable discrepancy of prevalence reported in the literature (0.9% to 7%) [16–19]. Also, IABP's correlation to NI likely reflects STEMI's severity (hemodynamic instability or cardiogenic shock), rather than an effect of the IABP itself. According to previous reports, a femoral rather than radial approach is associated, not only with a higher rate of bleeding and vascular complications, but morbidity and mortality as well [20–22]. However, the correlation with NI reported in our study may likely be related to the operator's preference in patients who arrive unstable to the catheterization laboratory, and so the reasoning behind the cause-and-effect relationship to predict infection may be the same as for the IABP.

PIA was a protective characteristic. It is likely related to the smaller infarct size caused by preconditioning which limits the reperfusion-injury phenomenon [23, 24], rather than having a direct influence on the development of a NI.

At 1-year follow-up, MACCE was independently associated with age, PAD, low hemoglobin concentration and low systolic pressure on admission, a higher peak CK activity, and femoral approach. Despite the unadjusted statistically significant difference in MACCE's incidence between patients with and without infection, it was not an independent predictor of these events on multivariate Cox model analysis. This is probably explained by the overlap of risk factors for infection and MACCE, namely age, PAD, and larger infarctions. This signals that features that favor infection are similar to those favoring adverse events, undermining a cause-and-effect relationship. Nevertheless, NI could function as a marker of frailty, helping physicians identify STEMI patients who are more prone to deteriorate clinically and might benefit from close surveillance.

Our analysis also showed a significant interaction between infection and smoking, seemingly not related to the infection site (namely, respiratory or urinary). Since smoking contributes both to the development of infection and cardiovascular disease in the long term [25, 26], and mortality from infection was also reported to be higher in smokers [27], this signals a tendency for a synergic effect between infection and smoking on MACCE. However, it is also reasonable to speculate that a nosocomial infection is more a sign of an underlying lung dysfunction then aggravated by heart insufficiency translated in mid-term events, than a causal risk factor *per se*.

Notwithstanding our findings, some series had shown that infection was significantly associated with a 30 or 90-day mortality. These cohorts only addressed "serious" infections and only captured short-term follow-up [2, 3]. On the other hand, another series reported that only pneumonia, and not infection in other sites, was associated with an increased risk of adverse events for an elderly population who underwent PCI irrespective of the indication [6]. Hence, the association between smoking habits and NI could be related to respiratory tract infections and might not be the same for urinary tract infections. We believe this is a hypothesis that should be addressed in future studies.

Limitations

The 48 hour cutpoint used for the definition of nosocomial infection is debatable, however, it is widely accepted and utilized in the literature. A major limitation of our study is the relationship between common risk factors to predict adverse events and the risk of a NI. The relative impact of NI in follow-up is, therefore, difficult to filter, despite the confounding variables incorporated in the multivariate equation and interaction analysis. Another limitation is the well-known limitation of a retrospective analysis, with its inherent bias to assume a cause-and-effect relationship between NI and outcomes. Lastly, being a single-center cohort study, the results may not be representative of all patients with STEMI undergoing PCI.

CONCLUSION

Our study determined that NI is a relatively common complication of STEMI (11.1%), with most risk factors that predict NI also being related to mid-term adverse events. NI does not constitute an independent predictor of MACCE, however, its occurrence during the first year was more than two times higher in smokers who complicate with a NI.

Supplementary material

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

Article information

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