Prognostic implications of mean platelet volume on short- and long-term outcomes among patients with non-ST-segment elevation myocardial infarction treated with percutaneous coronary intervention: A single-center large observational study

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Abstract

Background: Mean platelet volume (MPV) is a simple and reliable indicator of platelet size that correlates with platelet activation and their ability to aggregate. We studied the predictive value of MPV in patients with non-ST-segment elevation myocardial infarction (NSTEMI) treated with percutaneous coronary intervention (PCI).

Methods: We analyzed the consecutive records of 1001 patients who were hospitalized due to NSTEMI at our center. The primary end point was a composite end point that included the rates of all-cause death, non-fatal myocardial infarction, and acute coronary syndrome (ACS) driven revascularization at 12 months. The enrolled patients were stratified according to the quartile of the MPV level at admission.

Results: Along with the increasing quartile of MPV, the 12-month composite end point increased significantly (p = 0.010), and this association remained significant after the risk-adjusted analyses (per 1 fL higher MPV; adjusted hazard ratio [HR] 1.13; 95% confidence interval [CI] 1.02–1.27; p = 0.026). In the multivariate analysis, the MPV was also an independent factor of all-cause mortality (per 1 fL increase; adjusted HR 1.34; 95% CI 1.12–1.61; p = 0.0014) and death or non-fatal myocardial infarction (per 1 fL increase; adjusted HR 1.16; 95% CI 1.03–1.31; p = 0.017).

Conclusion: In patients with NSTEMI treated with PCI, a high MPV value was associated with a significantly increased incidence of long-term adverse events, particularly for all-cause mortality.

Keywords

Long-term prognosis, mean platelet volume, non-ST-segment elevation myocardial infarction, percutaneous coronary intervention

Introduction

Platelets represent an important link between inflammation and thrombosis [1]. Thrombotic complications of atherosclerosis are strictly connected with platelet function, including their activation and the ability to aggregate [2, 3]. Mean platelet volume (MPV) reflects the average size of the platelets (which range from 7.5 fL to 10.5 fL), and larger platelets are usually relatively young and have greater thrombogenic potential [4–6].

The increased MPV value is associated, among others, with hypertension [7], diabetes mellitus [8, 9], renal failure [10], and atrial fibrillation [11]. Higher MPV values are also observed in the elderly [12], obese [13], and smokers [5, 14, 15]; however, in other studies, the relationship between MPV and risk factors for cardiovascular disease is questionable [16–18]. Among the recent studies performed in patients with stable coronary artery disease (CAD) and ST-elevation myocardial infarction (STEMI), the impact of MPV on long-term prognosis was demonstrated [19–22]. The prognostic significance of MPV in non-ST-segment elevation myocardial infarction (NSTEMI) was only previously explored in two small-scale studies [23, 24] and one large-scale cohort of patients treated both invasively and non-invasively [25]. Therefore, in this study, we evaluate the prognostic role of MPV on the admission of patients with NSTEMI treated with percutaneous coronary intervention (PCI) in relation to long-term outcomes.

Methods

Study design

We analyzed consecutive records of 1001 unselected patients hospitalized for NSTEMI at our center from January 2006 to December 2012. The enrolled patients were stratified by the quartile of the MPV level at admission: I quartile—MPV < 10.2 fL; II quartile—MPV 10.2–11.0 fL; III quartile—MPV 11.0–11.7 fL; and IV quartile—MPV > 11.7 fL. All of the included patients underwent coronary angiography with PCI using standard devices. All interventional and therapeutic
strategies, including the choice of the stent type, periprocedural anti-thrombin, and antiplatelet therapy, were made at the discretion of the operating physician or heart team. Before and after the intervention, pharmacological treatment was administered according to the European Society of Cardiology guidelines [26].

A blood sample was collected by venipuncture, and the blood was drawn into a test tube containing an anticoagulant (EDTA). The samples were tested within 30 min of collection to minimize variations due to sample aging. The blood counts were determined using automated Sysmex SF 3000 (2006–2009) and Sysmex XS1000i (2010–2012) hematology analyzers (Sysmex Corporation, Kobe, Japan). According to the manufacturer standards, the MPV cut-off values for these two analyzers are identical, which was confirmed in our database (MPV value 2006–2009: median: 11.0 [10.1–11.8]; MPV value 2010–2012: median: 11.0 [10.3–11.7]). Our institutional laboratory has all of the necessary certificates. Two authorized external companies performed the device checks—one of the companies conducted these checks during every quarter of the year, and another one on random days once a month. Moreover, laboratory employees operated the machines on a daily basis, and the manufacturer performed service once every 6 months.

The demographic, clinical, and angiographic data collected during the current hospitalization were retrieved from the recorded Silesian Center for Heart Disease Electronic Database. The angiographic parameters were recorded on the basis of a visual assessment by two experienced interventional cardiologists. The follow-up data with accompanying exact dates of death, myocardial infarction (MI), and acute coronary syndrome (ACS)-driven revascularization were obtained from the official National Health Fund records. The follow-up status at 12 months was available for 100% of all of the included patients.

Definitions

NSTEMI was defined as the absence of the ST-segment elevation on the electrocardiogram consistent with an infarction of ≥2 mm in at least two contiguous precordial leads or at least two adjacent limb leads, or new or presumably new left bundle branch block and the presence of positive cardiac necrosis markers [26]. The presence of multivessel coronary artery disease (MV CAD) was defined as a significant stenosis of two or more major epicardial coronary arteries involving the territory of the right, left anterior descending (LAD), or left circumflex coronary vessels. A stenosis ≥50% of the diameter in the left main or proximal segment of LAD and a stenosis ≥70% of the diameter in other segments were considered hemodynamically significant. A coronary artery was considered to be an infarct-related artery (IRA) if one of the following criteria was present: definite or suspected thrombus, a ruptured or ulcerated plaque, the presence of thrombolysis in the myocardial infarction (TIMI) flow grade ≤2, and stenosis ≥70% of the diameter. At the angiographic analysis, only vessels >2 mm diameter were regarded as a possible target for revascularization. Periprocedural MI was defined as >20% elevation of cardiac troponin level in patients with increased and stable or falling troponins, occurring within 48 h of the procedure, according to the Third Universal Definition of MI [27]. The epicardial coronary artery flow was assessed using TIMI flow grades. Angiographic success was defined as the achievement of a minimum stenosis diameter reduction to <20% in the presence of TIMI flow grade 3.

End points

The primary end point was the incidence of the composite end point defined as (1) all-cause death, (2) non-fatal MI, and (3) ACS-driven revascularization at the 12-month observation period. The non-fatal MI was defined as an ischemic event that met the European Society of Cardiology/American College of Cardiology criteria for MI and was clinically separate from the baseline ACS at the time of admission [28]. Periprocedural MI was also included in this point. ACS-driven revascularization was defined as an additional, unplanned PCI or coronary artery bypass grafting of a previously revascularized coronary artery and was performed as an urgent procedure due to ACS. The secondary outcome included the occurrence of the components of the composite end point.

Statistical analyses

The statistical analysis included the comparison of baseline angiographic and procedural characteristics, and the in-hospital and 12-month adverse events. Sample size of n ≥ 974 was sufficient to detect hazard ratios (HRs) of 1.25 or greater under the assumption of the 25% combined event point at the end of follow-up period. Moreover, we assumed that the standard deviation (SD) of MPV would be 0.9, and the adjusted R-squared would be 0.2 (calculations were performed using PASS 14 statistical software (Power Analysis and Sample Size Software, Kaysville, Utah)). The analyzed variables are expressed as numbers and percentages. The continuous variables were summarized using an arithmetic mean with SD for data following normal distribution or a median with an interquartile range (IQR) for data demonstrating a non-normal distribution. Categorical variables were presented using frequency tables for both absolute numbers and percentages. The significance of the quartile of the MPV value on admission was evaluated using the Jonckheere–Terpstra test for continuous variables and the Cochran–Armitage test for categorical parameters. The 12-month composite end point, all-cause mortality, non-fatal MI, and ACS-driven revascularization were analyzed using the Kaplan–Meier method for all patients. The effects of the evaluated parameters on the 12-month incidence of adverse events were assessed using the multivariate Cox proportional hazard regression models with the results expressed as HRs and 95% confidence intervals (CIs). Candidate variables were entered into the model including parameters with significant influence on the composite end point and its components in univariate analysis: age (per year), prior MI, diabetes mellitus, peripheral artery disease, ST deviations in ECG on admission, left bundle branch block on admission, serum creatinine on admission (per 10 µmol/L), hemoglobin on admission (per 1 mmol/L), left ventricular ejection fraction (per 1%), three-vessel CAD, chronic total occlusion of a non-culprit vessel, PCI of the right coronary artery, success of PCI in the culprit vessel, cardiogenic shock, and pulmonary edema during hospitalization. A two-sided p-value ≤0.05 was considered significant. The STATISTICA 10 software (StarSoft Inc., Tulsa, OK, USA), MedCalc (MedCalc Software, Mariakerke, Belgium), and SPSS ver. 17.0.1 (SPSS, Inc., Chicago, IL, USA) were used for the calculations.

Results

The median age of the 1001-patient study population was 64.7 ± 10.7 years with 164 (16.4%) patients 80 years or older. The clinical characteristics of each quartile group are shown in Table I. There were no differences between the mean age and the sex of the patients across all of the groups. With an increasing value of MPV, observed trends were toward a higher frequency of hypertension, body mass index, hemoglobin, serum creatinine on admission, and ST-segment deviation at the initial ECG. The 6-month risk measured by the GRACE risk score and the probability of major bleeding measured by the CRUSADE risk score were
Table I. The baseline clinical characteristics of the study groups across the quartile of the mean platelet volume at admission.

| Variable                      | Level of mean platelet volume (fL) | N = 1001 |
|-------------------------------|-----------------------------------|----------|
|                               | N = 251 | N = 251 | N = 250 | n = 249 | p-Value |
| Age, years ± SD               | 64.8 ± 10.5 | 64.6 ± 10.3 | 64.4 ± 9.9 | 65.0 ± 11.8 | 0.63 |
| Age ≥ 80 years, %             | 7.5      | 7.9      | 6.4      | 11.6     | 0.076  |
| Males, %                      | 68.1     | 68.9     | 63.6     | 65.4     | 0.31   |
| Arterial hypertension, %      | 60.9     | 61.7     | 69.0     | 69.2     | 0.017  |
| Prior MI, %                   | 33.4     | 32.2     | 34.5     | 38.4     | 0.20   |
| Prior PCI, %                  | 25.9     | 23.1     | 26.1     | 31.9     | 0.091  |
| Prior CABG, %                 | 7.1      | 12.7     | 6.8      | 12.0     | 0.30   |
| Atrial fibrillation, %        | 7.9      | 5.1      | 4.8      | 7.6      | 0.86   |
| Peripheral artery disease, %  | 10.7     | 9.1      | 12.4     | 10.1     | 0.87   |
| Prior stroke, %               | 2.3      | 4.3      | 6.8      | 4.4      | 0.14   |
| Diabetes mellitus, %          | 27.4     | 29.8     | 26.9     | 36.8     | 0.055  |
| Requiring insulin treatment, %| 12.7     | 12.3     | 12.8     | 14.5     | 0.53   |
| Hypercholesterolemia, %       | 29.4     | 33.0     | 30.5     | 31.5     | 0.77   |
| Obesity, %                    | 24.7     | 25.9     | 24.9     | 29.5     | 0.28   |
| COPD, %                       | 4.3      | 4.7      | 4.8      | 3.2      | 0.56   |
| History of cigarette smoking, %| 39.8    | 35.8     | 39.7     | 40.4     | 0.67   |
| Current cigarette smoker, %   | 23.1     | 19.9     | 19.6     | 21.8     | 0.73   |
| Familial history of MI, %     | 23.1     | 25.1     | 25.7     | 24.2     | 0.73   |
| Chest pain, %                 | 93.6     | 93.6     | 94.7     | 94.7     | 0.46   |
| Onset from 2 h                | 2.5      | 3.8      | 2.5      | 4.7      | 0.31   |
| Onset from 2 to 12 h          | 39.1     | 34.3     | 35.0     | 33.8     | 0.27   |
| Onset from 12 to 24 h         | 17.6     | 24.3     | 20.2     | 24.4     | 0.18   |
| Onset from 24 h to 7 days     | 30.7     | 27.6     | 26.2     | 26.9     | 0.32   |
| Onset from more than 7 days   | 10.1     | 10.0     | 16.0     | 10.3     | 0.48   |
| Killip III, %                 | 1.5      | 5.5      | 4.0      | 4.0      | 0.29   |
| Killip IV, %                  | 1.5      | 0.8      | 0.8      | 1.2      | 0.69   |
| Heart rate, bpm ± SD          | 78.0 ± 15.5 | 78.4 ± 14.8 | 77.9 ± 16.5 | 79.2 ± 19.1 | 0.98 |
| SBP, mmHg ± SD                | 148.7 ± 28.3 | 152.7 ± 29.7 | 151.1 ± 27.2 | 150.6 ± 29.5 | 0.38 |
| DBP, mmHg ± SD                | 87.5 ± 16.9 | 89.6 ± 17.8 | 89.3 ± 17.9 | 88.7 ± 17.4 | 0.44 |
| ST deviation, %               | 38.2     | 36.8     | 44.4     | 49.5     | 0.013  |
| LBBB, %                       | 3.5      | 6.8      | 4.4      | 6.1      | 0.41   |
| RBBB, %                       | 6.3      | 7.2      | 4.0      | 3.6      | 0.075  |
| BMI, kg/m² ± SD               | 27.9 ± 4.5 | 28.4 ± 4.4 | 28.8 ± 5.4 | 29.2 ± 4.8 | 0.0050 |
| WBC, 10³/µL (IQR)             | 9.1 (7.3–11.4) | 9.2 (7.2–10.9) | 9.0 (7.1–11.1) | 9.5 (7.5–11.6) | 0.61 |
| Hemoglobin, g/dL ± SD         | 8.3 ± 1.2  | 8.4 ± 1.1  | 8.5 ± 1.0  | 8.7 ± 1.0  | 0.0031 |
| Glucose, mmol/L (IQR)         | 6.2 (5.4–8.0) | 6.4 (5.4–8.4) | 6.6 (5.6–8.6) | 6.6 (5.5–8.8) | 0.16  |
| Serum creatinine, mmol/L (IQR)| 79.2 (65.3–97.3) | 78.7 (66.1–93.3) | 79.0 (66.1–97.2) | 83.2 (71.9–102.8) | 0.037 |
| GFR, ml/min/1.73 m² (IQR)     | 83.9 (61.7–101.1) | 85.0 (65.5–103.3) | 82.7 (66.9–102.9) | 78.6 (57.7–94.9) | 0.079 |
| GRACE risk score, points ± SD | 114.3 ± 26.6 | 111.7 ± 32.1 | 111.6 ± 28.0 | 114.2 ± 32.6 | 0.44  |
| CRUSADE risk score, points (IQR)| 24 (16–39) | 22 (16–33) | 25 (16–34) | 26 (18–38) | 0.44  |
| LVEF, % ±SD                   | 44.7 ± 10.0 | 44.4 ± 9.1 | 43.2 ± 9.8 | 43.1 ± 10.9 | 0.073 |

Abbreviations: BMI = body mass index; DBP = diastolic blood pressure; CABG = coronary-artery bypass grafting; COPD = chronic obstructive pulmonary disease; GFR = glomerular filtration rate; IQR = interquartile range; LBBB = left bundle branch block; LVEF = left ventricular ejection fraction; MI = myocardial infarction; PCI = percutaneous coronary intervention; RBBB = right bundle branch block; SBP = systolic blood pressure; SD = standard deviation; WBC = white blood cells.

Similar in all of the quartiles of the MPV level. Angiographic findings across the analyzed groups are described in Table II. Patients with a higher level of MPV had a higher frequency of chronic total occlusion and a lower frequency of planned PCI at discharge. Table III shows the cardiac event rates over a hospital stay index and a follow-up period of 12 months in the study population across the quartiles of the MPV level.

There was evidence during the hospitalization index that patients with an increased MPV value had a higher frequency of target vessel revascularization (p = 0.027), but there was no significant difference in the incidence of death (p = 0.38) and recurrent non-fatal MI (p = 0.91). The 12-month Kaplan–Meier curves for the composite end point and its components are presented in Figure 1. In general, the association between the baseline MPV value and the composite end point was significant at the 12-month follow-up in both univariate (per 1 fL increase; HR 1.13; 95% CI 1.01–1.26; p = 0.025) and multivariate analyses (per 1 fL increase in MPV; adjusted HR 1.13; 95% CI 1.02–1.27; p = 0.026; Table IV). Furthermore, the 12-month all-cause mortality was significantly higher along with an increasing level of MPV (6.7% vs. 5.5% vs. 10.0% vs. 12.8% in each subsequent quartile of MPV; p = 0.0047). The association remained significant after the risk-adjusted analyses (adjusted HR 1.34; 95% CI 1.12–1.61; p = 0.0014). In the multivariate analysis, the level of MPV was also an independent predictor of death or non-fatal MI (adjusted HR 1.16; 95% CI 1.03–1.31; p = 0.017). We did not observe the influence of the MPV level on the other outcomes, in particular the incidence of non-fatal MI (HR 1.07; 95% CI 0.91–1.25; p = 0.42) and ACS-driven revascularization (HR 1.06; 95% CI 0.90–1.23; p = 0.49).
Table II. Angiographic and procedural characteristics of the study groups across the quartile of the mean platelet volume at admission.

| Variable | Level of mean platelet volume (fL) | N = 1001 |
|----------|-----------------------------------|---------|
|          | <10.2 | 10.2–11.0 | 11.0–11.7 | >11.7 | p-Value |
| PCI IRA  |       |          |          |       |
| PCI ad hoc, % | 95.2 | 96.8 | 96.8 | 97.1 | 0.093 |
| LM, %    | 1.5 | 1.9 | 4.0 | 4.4 | 0.22 |
| LAD, %   | 31.0 | 33.0 | 34.4 | 36.1 | 0.22 |
| Cx, %    | 35.0 | 33.0 | 29.6 | 30.5 | 0.20 |
| RCA, %   | 29.4 | 28.6 | 28.8 | 22.8 | 0.12 |
| Bypass, %| 2.7 | 3.1 | 3.2 | 6.0 | 0.073 |
| Restenotic lesion, % | 9.1 | 7.9 | 6.0 | 8.8 | 0.69 |
| Bifurcation of LM, % | 2.3 | 3.9 | 4.4 | 4.4 | 0.23 |
| Bifurcation other than LM, % | 13.9 | 15.9 | 17.6 | 14.0 | 0.84 |
| Baseline TIMI flow grade 0–1, % | 29.0 | 23.1 | 28.4 | 27.7 | 0.92 |
| Stent implantation, % | 90.0 | 91.2 | 90.4 | 90.3 | 0.98 |
| Balloon predilatation, % | 64.5 | 65.7 | 62.4 | 63.8 | 0.69 |
| Balloon postdilatation, % | 11.9 | 11.5 | 12.0 | 12.8 | 0.73 |
| DES, %   | 20.3 | 25.5 | 18.0 | 23.6 | 0.82 |
| Procedural Glycoprotein IIb/IIIa inhibitor, % | 8.7 | 8.3 | 6.8 | 10.8 | 0.56 |
| Dissection, % | 7.9 | 7.1 | 8.0 | 6.0 | 0.49 |
| No/slow reflow, % | 1.2 | 0.8 | 3.2 | 2.8 | 0.066 |
| Final TIMI flow grade 3 after PCI, % | 95.2 | 93.2 | 91.6 | 91.1 | 0.059 |
| Procedural success of PCI IRA, % | 93.6 | 91.6 | 90.0 | 90.3 | 0.14 |
| PCI of additional artery during hospitalization, % | 18.3 | 16.7 | 18.0 | 18.0 | 0.96 |
| Angiographic success of all lesions, % | 92.0 | 91.2 | 88.0 | 89.9 | 0.25 |
| Planned PCI after discharge, % | 14.7 | 12.7 | 13.2 | 8.4 | 0.046 |
| Planned CABG after discharge, % | 2.3 | 4.3 | 4.0 | 5.2 | 0.14 |

Abbreviations: CAD = coronary artery disease; Cx = circumflex artery; DES = drug eluting stent; GP = glycoprotein; IRA = infarct-related artery; LAD = left anterior descending artery; LM = left main; LVEF = left ventricular ejection fraction; PCI = percutaneous coronary intervention; RCA = right coronary artery; TIMI = thrombolysis in myocardial infarction.

Discussion

The main findings of our analysis conducted on a “real world” group of 1001 consecutive patients with NSTE-MI undergoing PCI proved that a single measurement of MPV on admission was an independent predictor of all-cause mortality in the 12-month observation. The elevation of the MPV value by 1 fL increases the risk of the composite endpoint in the 12-month follow-up by 13%. Furthermore, with an increasing value of MPV, a 34% higher risk of all-cause death and a 16% higher risk of death or non-fatal re-infarction were observed. Basing on post hoc analysis, the study achieves 99% of power at alpha = 0.05 to detect adjusted HR equal to 1.34.

Recently, MPV emerged as a cardiovascular risk factor in the general population and in patients with CAD and ACS. For instance, in an Austrian population of 200 000 people, an MPV of greater than 11.01 fL was an independent risk factor for cardiovascular mortality. In long-term observation, all-cause mortality was 1.5 times higher and CAD-related mortality was 1.8 times higher in individuals in the highest quintile of the MPV value [3]. In a multivariate analysis performed by Ozkan et al., MPV was documented to be an independent MI risk factor in men under the age of 45 and in women under the age of 55 [19]. Moreover, some other publications underline the prognostic significance of MPV in CAD patients [20–22]. Among others, Eisen et al. demonstrated that in a population of over 7500 CAD patients undergoing PCI, MPV was higher in individuals with ACS than patients with stable CAD [20].

In addition, Khandehar et al. reported that in patients with stable CAD, a higher MPV value was observed in comparison with healthy individuals [2], whereas in ACS, the MPV value was higher in cases of MI than in unstable CAD [5]. For instance, Khode et al. showed that MPV is significantly higher in patients with MI than in ones with stable CAD and lowest in the control group [29]. Additionally, Pal et al. demonstrated a greater MPV value in ACS patients compared with non-ACS patients [30].

To assess CAD severity and complexity, Murat et al. used Gensini and SYNTAX scores [31]. The patients were divided into two groups according to the MPV value—one with a low MPV (8.3 ± 0.9 fl) and another with a high MPV (10.8 ± 0.4 fl). Both the Gensini and SYNTAX scores were higher in patients with high MPV values compared with those in the low MPV group. In a multivariate analysis, MPV was found to be an independent factor correlated with the degree of coronary artery atherosclerosis. Moreover, Liu et al. found that a lower level of MPV was an independent factor associated with LAD-located...
Table III. Pharmacotherapy at discharge and in-hospital and long-term outcomes of the study groups across the quartile of the mean platelet volume at admission.

| Level of mean platelet volume (fL) | N = 1001 |
|----------------------------------|----------|
| <10.2                            | 251      |
| 10.2–11.0                        | 251      |
| 11.0–11.7                        | 250      |
| >11.7                            | 249      |

| Variable                        | N = 251 | N = 251 | N = 250 | n = 249 | p-Value |
|---------------------------------|---------|---------|---------|---------|---------|
| **Pharmacotherapy at discharge**|         |         |         |         |         |
| Aspirin, %                      | 98.7    | 98.7    | 99.5    | 97.9    | 0.58    |
| PY12 inhibitors, %              | 95.5    | 90.2    | 92.6    | 93.7    | 0.68    |
| Beta-blocker, %                 | 91.8    | 90.2    | 90.5    | 92.9    | 0.19    |
| Angiotensin-converting enzyme inhibitor, % | 83.7    | 90.2    | 85.6    | 86.7    | 0.64    |
| Statin, %                       | 89.4    | 94.2    | 91.8    | 92.9    | 0.29    |
| Nitrate, %                      | 25.2    | 22.0    | 27.4    | 27.3    | 0.33    |
| Calcium antagonist, %           | 15.8    | 17.1    | 17.2    | 18.6    | 0.43    |
| Diuretic, %                     | 33.7    | 33.8    | 31.9    | 42.7    | 0.069   |
| **In-hospital outcomes**        |         |         |         |         |         |
| Death, %                        | 1.5     | 2.3     | 2.4     | 2.8     | 0.38    |
| Non-fatal MI, %                 | 1.2     | 1.9     | 1.6     | 1.2     | 0.91    |
| Periprocedural MI, %            | 1.2     | 1.2     | 0.8     | 1.2     | 0.96    |
| TVR, %                          | 1.5     | 1.5     | 3.2     | 4.4     | 0.027   |
| Major bleeding, %               | 2.3     | 2.3     | 3.2     | 2.4     | 0.84    |
| Hospital stay, days (IQR)       | 5 (4–7) | 5 (4–7) | 5 (4–7) | 6 (4–8) | 0.44    |
| **12-month outcomes**           |         |         |         |         |         |
| Composite end point, %          | 20.7    | 18.7    | 24.0    | 25.3    | 0.010   |
| Death, %                        | 6.7     | 5.5     | 10.0    | 12.8    | 0.0047  |
| Non-fatal MI, %                 | 10.7    | 11.1    | 10.8    | 10.0    | 0.77    |
| Death/non-fatal MI, %           | 16.3    | 15.1    | 19.6    | 21.2    | 0.076   |
| ACS-driven revascularization, % | 13.1    | 7.5     | 11.6    | 11.6    | 0.95    |

Abbreviations: ACS = acute coronary syndrome; IQR = interquartile range; MI = myocardial infarction; TVR = target vessel revascularization.

Figure 1. Kaplan–Meier survival curves for 12-month rates of composite end point (A), death of any cause (B), non-fatal myocardial infarction, (C) and ACS-driven revascularization (D) according to the quartile of the mean platelet volume at admission.
Table IV. Unadjusted and adjusted hazard ratios for occurrence of the 12-month composite end point in the study groups depending on the level of mean platelet volume at admission (per 1 fL increase).

| Factor (MPV on admission per 1 fL increase) | Unadjusted HR | 95% CI   | p-value | Adjusteda HR | 95% CI   | p-Value |
|---------------------------------------------|---------------|----------|---------|--------------|----------|---------|
| 12-month outcomes                           |               |          |         |              |          |         |
| Composite end point                         | 1.13          | 1.01–1.26| 0.025   | 1.13         | 1.02–1.27| 0.026   |
| Death                                       | 1.26          | 1.06–1.50| 0.0089  | 1.34         | 1.12–1.61| 0.0014  |
| Non-fatal MI                                 | 1.07          | 0.91–1.25| 0.42    | –            | –        | –       |
| Death/non-fatal MI                           | 1.15          | 1.03–1.30| 0.021   | 1.16         | 1.03–1.31| 0.017   |
| ACS-driven revascularization                 | 1.06          | 0.90–1.23| 0.49    | –            | –        | –       |

aAdjusted HR for the following parameters: three-vessel coronary artery disease, age (per 1 year more), body mass index (per 1 kg/m² more), cardiogenic shock during hospitalization, chronic total occlusion of non-culprit vessel, diabetes mellitus, hemoglobin on admission (per 1 mmol/L), left ventricular ejection fraction (per 1%), percutaneous coronary intervention of right coronary artery, peripheral arteries disease, prior myocardial infarction, pulmonary edema during hospitalization, serum creatinine at admission (per 10 µmol/L), ST deviations in ECG on admission, success of percutaneous coronary intervention in the culprit vessel, left bundle branch block at admission.

Abbreviations: ACS = acute coronary syndrome; CI = confidence interval; MI = myocardial infarction.

Considered the results presented, and the fact that MPV is a routinely assessed parameter of a complete blood count, it is worth emphasizing the prognostic impact of MPV in combination with the GRACE scale [24]. Wan et al. analyzed the influence of the MPV value in combination with the GRACE score in 290 patients with ACSs [37]. During a median period of 52 months, the multivariate analysis has shown that the GRACE score with the addition of MPV had a better prognostic value than the GRACE score alone. Similar results and conclusions were obtained by Niu et al. in a study performed on 509 patients with ACS [38]. Moreover, the addition of the MPV level to the GRACE score permitted the reclassification of the measured risk in 16% of patients. Our research, conducted on a large group of patients with NSTEMI treated with PCI, confirmed that the increase in MPV value was connected with a higher risk of long-term adverse events and may be potentially utilized as an important element of long-term risk scores to assess outcomes in patients with NSTEMI.

Strengths and limitations

The current study does have some limitations that bear mentioning. This study is a single-center observational study derived from a real-life practice with inherent weakness related to retrospective analysis. The severity and location of coronary lesions were based on a visual assessment by the operator and without quantitative and functional assessment. The results of our multivariate analysis may be biased due to the potential impact of important factors that are not available in our database. With data on mortality obtained from the National Health Fund, only the all-cause mortality was available. We had no insight into the cause of death (i.e., cancer or cardiovascular death). In our analysis, advanced statistical models (C statistics, Net Reclassification Index, Integrated Discrimination Improvement) were not applied. These models should be considered in future studies.

Finally, the data used in this analysis of patients treated in a highly specialized cardiology center may not reflect the general population of NSTEMI patients. Nonetheless, potential disadvantages are diminished by the fact that the patients’ data were input electronically by the attending physician upon the patient’s admission to our center. Strengths of this study include a large patient cohort, detailed data on clinical and laboratory parameters, and a long-term follow-up.

Conclusion

In patients with NSTEMI treated with PCI, a high MPV value at admission was associated with a significant increase of long-term adverse event incidence, particularly in all-cause mortality.
Declaration of interest
The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the article.

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