ORIGINAL ARTICLE

Prognostic value of acid-base balance parameters assessed on admission in peripheral venous blood of patients with myocardial infarction treated with percutaneous coronary intervention

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KEY WORDS

anion gap, base excess, lactate, myocardial infarction, point-of-care test

ABSTRACT

INTRODUCTION Peripheral venous blood sample may be used to obtain acid-base balance parameters (PVA-BP) measured in rapid point-of-care test (POCT) analyzers on admission to an emergency department (ED). Thus, lactates, anion gap (AG), and base excess (BE) may be early prognostic markers in patients with myocardial infarction (MI).

OBJECTIVES We aimed to confirm the relationship between PVA-BP on admission and the outcome in patients with MI treated with percutaneous coronary intervention (PCI).

PATIENTS AND METHODS This was a retrospective, observational analysis of MI patients admitted primarily to an ED and secondly transferred to PCI department.

RESULTS A total of 336 patients (41.1% ST-elevated MI, 58.9% non–ST-elevated MI) were divided according to their lactate level, that is, G1 group with lactate below or equal to 2.0 mmol/l (n = 207) and G2 group with lactate above >2.0 mmol/l (n = 129). G2 patients had higher values of AG (mean, [SD], 9.6 [4.3] vs 6.8 [3.2] mEq/l; P <0.001) and lower BE (median [interquartile range], –0.7 [–3.9 to 0.8] vs 1.0 [–0.2 to 2.4] mEq/l; P <0.001). In-hospital nonsurvivors had higher values of lactates (4.0 [2.0–8.7] vs 1.7 [1.3–2.4] mmol/l; P <0.001), AG (10.5 [4.6] vs 7.7 [3.8] mEq/l; P <0.001), and lower BE (–4.8 [–10.6 to –1.8] vs 1.5 [–0.8 to 2.3] mEq/l; P <0.001) than the survivors. Lactates, AG, and BE correlated with Global Registry of Acute Coronary Events score (r = 0.361, P <0.001; r = 0.158, P = 0.004; r = –0.383, P <0.001, respectively). Only BE independently predicted both 30- and 365-day mortality in the whole group (hazard ratio [HR], 0.79; 95% CI, 0.65–0.95; P = 0.01 and HR, 0.89; 95% CI, 0.76–0.99; P = 0.04, respectively) as well as in-hospital mortality among patients without infarct-related out-of-hospital cardiac arrest (odds ratio, 0.74; 95% CI, 0.57–0.97; P = 0.03).

CONCLUSIONS In the patients admitted to the ED with MI treated with PCI the evaluation of PVA-BP in POCT analyzers may be a reliable tool for early risk stratification.

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INTRODUCTION The gold standard for treating patients with both ST-segment elevation (STEMI) and non–ST-segment elevation (NSTEMI) myocardial infarctions (MI) and hemodynamic instability is a rapid transport to a catheterization lab.[1,2] However, we must admit that in practice direct transportation to the catheterization lab fails in a significant number of patients with MI requiring urgent revascularization: the patients are first admitted to the emergency department

WHAT'S NEW?

In this retrospective study of patients with myocardial infarction, treated with percutaneous coronary intervention and primarily admitted to the emergency department, we confirmed that acid-base balance parameters in peripheral venous blood assessed in point-of-care test analyzers may serve as reliable tools for early risk stratification. Elevated lactate level is associated with worse clinical condition, and it can be suspected that lactic acidosis is a consequence of a more severe derangement (ie, in patients with out-of-hospital cardiac arrest). In contrast, base excess may be considered a marker of significant importance even in a subtle degree acidosis, because it predicts mortality in short- and long-term observation in the whole study group, as well as in-hospital mortality in patients without out-of-hospital cardiac arrest.

(ED) and later transferred for angiography, regardless of the type of MI. This may be due to numerous reasons, that is, nonspecific or late electrocardiogram (ECG) changes, ambiguous prehospital diagnosis, need for excluding another significant comorbidity, the patient presenting to the ED on their own.[3] Additionally, a diagnosis of an acute total occlusion of the coronary artery on the basis of ECG is quite obvious for an experienced cardiologist but the diagnostic accuracy of non-cardiologists (including emergency department physicians) interpreting ECG may be lower.[4] Having in mind that the specificity and sensitivity of ECG in the diagnosis of the acute total occlusion of the coronary artery is not satisfying, we believe that there is a need for additional risk stratifying tools in patients with MI admitted to the ED.[5]

The standard of care at the ED is to obtain peripheral venous blood samples on admission, regardless of initial triaging, in order to assess basic laboratory parameters. The availability of rapid point-of-care test (POCT) analyzers encourages the search for an additional marker that could indicate high-risk patients, who should be immediately transferred to the catheterization lab to improve their prognosis. An additional advantage is the on-site nature of POCT assay making the results more streamlined.

Lactates and other acid-base balance parameters (including anion gap [AG] and base excess [BE]) are considered good indicators of metabolic condition in critically ill patients.[6,7]

It is believed that the gold standard in order to analyze acid-base status is to obtain the arterial blood sample. However, there are some studies suggesting that peripheral venous blood sample may also be reliable.[8-12]

A number of studies[13-17] investigated the relationship between lactate, BE or AG and prognosis in patients with MI treated with percutaneous coronary intervention (PCI). However, this was observed with arterial concentrations of the aforementioned parameters or post-PCI implantation, and there are fewer studies on the assessment of these parameters in POCT from a blood sample obtained from a peripheral vein.[18]

We aimed to confirm the relationship between PVA-BP on admission and the outcome in patients with MI consecutively treated with PCI.

We hypothesized that an easy to obtain PVA-BP measurement in POCT may be the earliest laboratory marker of prognosis in patients with MI and could be viewed as an additional, effective tool to select MI patients who require revascularization.

PATIENTS AND METHODS We retrospectively analyzed medical records of consecutive patients who were initially admitted to the ED of the University Hospital, Kraków, Poland between January 1, 2016, and December 31, 2018 with MI, and then qualified for PCI treatment and standard medical therapy according to the European Society of Cardiology (ESC) guidelines.[1,2] The patients with MI who were not admitted primarily to the ED were excluded from this analysis. MI is a heterogeneous disease with various clinical presentations (including both relatively stable or unstable life-threatening conditions such as pulmonary edema or cardiogenic shock) and different electrocardiographic manifestations (STEMI or NSTEMI), which is why we decided to include both STEMI and NSTEMI patients without respect to MI subtypes. The diagnosis of MI was based on the ESC guidelines criteria.[1,2]

In our hospital, a peripheral venous blood sample is obtained directly after admission to the ED from all patients, regardless of their diagnosis and clinical condition. In this study, the blood sample was analyzed with an ABL90 90FLEX POCT analyzer (Radiometer, Copenhagen, Denmark) to measure biochemical parameters including lactate levels, BE, AG, serum glucose, troponin I, and creatinine. The estimated glomerular filtration rate (eGFR) was calculated from the Modification of Diet in Renal Disease formula.[19] Heart rate, arterial blood pressure, Killip class, and Global Registry of Acute Coronary Events (GRACE) risk score was assessed in all patients based on their clinical condition.[20] Thrombolysis in Myocardial Infarction (TIMI) coronary flow grade scores was evaluated before and after PCI.[21] A total occlusion of an infarct-related artery (IRA) was defined as TIMI = 0. After analysis of serial creatinine measurements over the course of hospitalization, acute renal injury was defined as a drop in eGFR by at least 30% in comparison with the admission values. Transthoracic 2-dimensional echocardiography was performed in the patients on admission to the cardiology department to measure their left ventricular ejection fraction (LVEF). On the basis of the data obtained from the Universal Electronic System for Registration of the Population in Poland, the occurrence of death from any cause assessed at 30 and 365 days since admission was evaluated for all the study participants. Our study was an observational retrospective analysis of anonymized electronic medical

records of patients treated in our hospital. It was conducted according to the guidelines of the Declaration of Helsinki and approved by the Bioethics Committee of the Jagiellonian University (1072.6120.373.2020).

Data availability The data associated with this paper are not publicly disclosed but are available from the corresponding author upon a reasonable request.

Statistical analysis Categorical variables are presented as numbers and percentages. Continuous variables are expressed as means and SD or medians and interquartile range (IQR). Normality was assessed by the Shapiro–Wilk test. Equality of variances was assessed using the Levene test. We divided the study population into 2 groups according to their lactate levels, that is, G1 group with lactate below or equal to 2.0 mmol/l, and G2 group with lactate above 2.0 mmol/l. Differences between the groups were compared using the Student or Welch *t* test depending on the equality of variances for normally distributed variables. The Mann–Whitney test was used for non-normally distributed continuous variables. Ordinal variables were compared using the Cochran–Armitage test for trend. Categorical variables were compared by the Pearson χ^2 test or by the Monte Carlo simulation for the Fisher test, if 20% of cells had an expected count of less than 5. The Spearman rank correlation coefficient was calculated to measure the monotonic trend between 2 variables. To analyze event-free survival in the selected risk groups, the Kaplan–Meier curves were drawn. The log-rank statistic was used to test the differences in the outcomes between the groups. Additionally, the multivariable Cox proportional hazard analyses were performed to identify independent predictors of mortality. The variables that were associated with the occurrence of 30- and 365-day mortality with a significance level of $P < 0.2$ in the bivariable models, as well as other variables judged to be of clinical importance, were selected for possible inclusion in the multivariable logistic regression model to predict the occurrence of the outcome.[22] The results were presented as hazard ratios (HRs) with 95% CIs. The proportional hazards model assumptions were checked using the Schoenfeld test and graphical diagnostics. Multivariable logistic regression was used for searching for possible covariates of the likelihood of in-hospital death. Then, odds ratios (ORs) and corresponding 95% CIs were calculated for covariates influencing in-hospital death. Statistical analyses were performed with JMP package, version 14.2.0 (SAS Institute Inc., Cary, North Carolina, United States) and R software, version 3.4.1 (R Core Team, Vienna, Austria).

RESULTS A total of 336 patients (70.5% men) with MI were reviewed. Mean (SD) age was 66.6 (12.2) years. There were 138 (41.1%) STEMI and 198 (58.9%) NSTEMI patients. Arterial hypertension (78.9%) and hypercholesterolemia (68.5%) were the predominant coexisting diseases. Mean (SD) time from the onset of symptoms to PVA-BP determination was 409.9 (328.9) minutes. There were 45 patients (13.4%) with Killip class 3 or 4. Out-of-hospital cardiac arrest (OHCA) occurred in 19 individuals (5.7%). Median (IQR) GRACE score was 125.4 (101.4–157.2), mean (SD) LVEF was 45.3% (12.9). Total occlusion of the IRA was seen in 151 patients (44.9%). The left main coronary artery was IRA in 13 patients (3.9%) and 140 patients (41.7%) had a multivessel disease (MVD). In 19 patients (5.7%), TIMI 0 persisted after PCI. Mortality rates at 30 and 365 days were 9% (n = 30) and 15.7% (n = 52), respectively.

Whole study group There were 207 patients with lactate levels below or equal to 2.0 mmol/l (G1 group), and 129 patients with lactate levels above 2.0 mmol/l (G2 group). The patients with higher lactate levels were older and more frequently diabetic, nonsmokers, and in more severe general clinical state (higher heart rate, lower blood pressure including systolic, diastolic, and mean arterial blood pressure, higher Killip class, lower LVEF, higher GRACE score, and more frequent OHCA before admission). STEMI was observed more often in the G2 than the G1 group. Higher glucose level, lower eGFR, and signs of systemic acidosis (estimated by pH, AG, and BE levels) were noticed in the individuals with higher lactates. Troponin I level on admission was similar in both groups (**TABLE 1**).

The location of the culprit-related artery on angiography was similar in both groups. Total occlusion of IRA occurred more frequently in the G2 group. The patients with higher lactates (G2) had significantly higher mortality in all assessed time periods (**TABLE 2**).

The Kaplan–Meier curves in **FIGURE 1** display cumulative survival, stratified based on lactate levels on admission (G1 vs G2).

Sensitivity analysis In a sensitivity analysis, we excluded the patients with OHCA, and there were 207 patients in the G1 and 110 in the G2 group. The findings of the subgroup analysis were similar to the aforementioned in the whole study group, that is, the G2 patients were more frequently diabetic (43 [39.1%] vs 51 [24.6%]; $P = 0.006$), less often smoked (28 [25.5%] vs 81 [39.1%]; $P = 0.01$), and were in a more severe clinical condition (higher heart rate (median [IQR], 80.0 [70.0–90.0] vs 77.0 [70.0–85.3] bpm; $P = 0.03$), lower blood pressure including systolic (median [IQR], 142.0 [125.0–164.0] vs 150.0 [130.0–165.0] mm Hg; $P < 0.001$), diastolic (median [IQR], 80.0 [70.0–90.0] vs 82.0 [75.0–90.0] mm Hg; $P = 0.04$), and mean arterial blood pressure (median [IQR], 100.0 [89.0–112.7] vs 104.5 [96.3–114.5] mm Hg; $P = 0.048$), higher Killip

TABLE 1 Baseline clinical characteristics depending on the level of peripheral venous blood lactates (whole study group)

Parameter	Lactates ≤2.0 mmol/l (n = 207)	Lactates >2.0 mmol/l (n = 129)	*P* value
Age, y	65.6 (11.5)	68.2 (13.1)	0.06
Female sex, n (%)	58 (38.0)	41 (31.8)	0.27
BMI	28 (4.6)	28.3 (4.7)	0.68
Smoking, n (%)	81 (39.1)	32 (25.4)	0.007
Hypertension, n (%)	165 (79.7)	100 (77.5)	0.37
Hypercholesterolemia, n (%)	145 (70.0)	85 (65.9)	0.25
Diabetes, n (%)	51 (24.6)	49 (38.0)	0.007
Atrial fibrillation, n (%)	28 (13.5)	23 (17.8)	0.18
History of myocardial infarction, n (%)	54 (26.1)	31 (24.0)	0.39
STEMI, n (%)	77 (37.2)	61 (47.3)	0.04
Killip class 3, n (%)	7 (3.4)	12 (9.3)	<0.001
Killip class 4, n (%)	3 (1.4)	23 (17.8)	<0.001
Cardiac arrest before admission, n (%)	0	19 (14.7)	<0.001
Heart rate, bpm	77.0 (70.0–85.5)	80.0 (70.0–90.0)	0.03
Systolic blood pressure, mm Hg	150.0 (130.0–165.0)	139.0 (118.0–160.0)	0.004
Diastolic blood pressure, mm Hg	82.0 (75.0–90.0)	80.0 (65.0–90.0)	0.002
Mean arterial pressure, mm Hg	104.5 (96.3–113.4)	98.2 (83.3–112.3)	0.001
Glucose level, mmol/l	7.0 (6.1–8.4)	9.8 (7.3–14.1)	<0.001
Troponin I, µg/l	0.18 (0.04–0.89)	0.14 (0.03–0.74)	0.4
eGFR, ml/min/1.73 m^2	97.1 (35.8)	83.9 (37.3)	0.001
LVEF, %	46.8 (12.6)	42.7 (13.1)	0.006
GRACE score	121.00 (96.0–140.8)	141.6 (113.8–186.7)	<0.001
Time from pain to lactate level measurement, min	455 (13.8)	328 (12)	<0.001
pH	7.39 (7.37–7.42)	7.38 (7.32–7.41)	<0.001
Lactates, mmol/l	1.4 (1.1–1.7)	2.9 (2.4–4.6)	<0.001
Anion gap, mEq/l	6.8 (3.2)	9.6 (4.3)	<0.001
Base excess, mEq/l	1.0 (–0.2 to 2.4)	–0.7 (–3.9 to 0.8)	<0.001

Data are presented as mean (SD) or median (interquartile range) unless indicated otherwise.

Abbreviations: BMI, body mass index; eGFR, estimated glomerular filtration rate; GRACE, Global Registry of Acute Coronary Events; LVEF, left ventricular ejection fraction; NSTEMI, non–ST-segment elevation myocardial infarction; STEMI, ST-segment elevation myocardial infarction

class (9 [8.2%] vs 7 [3.4%]; *P* <0.001 for Killip 3 and 12 [10.9%] vs 3 [1.4%]; *P* <0.001 for Killip 4), lower LVEF (mean [SD], 43.1 [13.3]% vs 46.8 [12.6]%; *P* = 0.02), and higher GRACE score (median [IQR], 133.6 [103.2–164.8] vs 121.0 [96.0–140.8]; *P* = 0.001). There was no difference in the frequency of STEMI between G1 and G2 group (77 [37.2%] vs 47 [42.7%]; *P* = 0.20), however, total occlusion of IRA occurred more frequently in the G2 group (57 [51.8%] vs 82 [39.6%]; *P* = 0.005). Higher glucose level (median [IQR], 8.5 [6.9–11.9] vs 7.0 [6.1–8.4] mmol/l; *P* <0.001), lower eGFR (mean [SD], 85.3 [35.9] vs 97.1 [35.8] ml/min/1.73m^2; *P* = 0.006) and signs of systemic acidosis (estimated by pH (median [IQR], 7.38 [7.34–7.41] vs 7.39 [7.37–7.42], *P* = 0.004), AG (mean [SD], 9.0 [3.6] vs 6.8 [3.2] mEq/l; *P* <0.001), and BE (median [IQR], –0.3 [–2.0 to 1.4] vs 1.1 [–0.2 to 2.4] mEq/l; *P* <0.001) were noticed in the G2 group. Troponin I level on admission was similar in G1 and G2 group (median [IQR], 0.14 [0.03–0.95] vs 0.19 [0.05–0.93] µg/l; *P* = 0.50). In comparison with in-hospital survivors, the patients who died had significantly higher lactate levels (median [IQR], 4.0 [2.0–8.7] vs 1.7 [1.3–2.4] mmol/l; *P* = 0.01), AG (mean [SD], 9.2 [3.2] vs 7.5 [3.5] mEq/l; *P* = 0.04) and lower BE (median [IQR], –4.8 [–10.6 to –1.8] vs 1.5 [–0.8 to 2.3]; *P* <0.001) (FIGURE 2).

Relationship between Global Registry of Acute Coronary Events score and acid-base balance parameters In the whole study group, significant correlations between GRACE score and lactates, AG, and BE were observed. In a subgroup analysis after exclusion of OHCA patients, a significant correlation between GRACE score and lactates and BE was observed. Interestingly, we found no correlation between troponin I level on admission and GRACE score for both whole study group and subgroup analysis (Supplementary material, *Figure S1*).

TABLE 2 Angiography results and patient outcome depending on the level of peripheral venous blood lactates (whole study group)

Parameter	Lactates ≤2.0 mmol/l (n = 207)	Lactates >2.0 mmol/l (n = 129)	*P* value
Infarct-related artery			
Acute total occlusion	82 (39.6)	69 (53.5)	0.009
LAD	69 (33.3)	45 (34.9)	0.43
LMCA	7 (3.4)	6 (4.7)	0.38
Cx	54 (26.1)	35 (27.1)	0.47
RCA	66 (31.9)	43 (33.3)	0.44
Multivessel disease	83 (40.1)	57 (44.2)	0.27
TIMI 0 after PCI	9 (4.3)	10 (7.8)	0.09
GP IIb/IIIa inhibitor	46 (22.2)	30 (23.3)	0.46
Worsening of kidney function	36 (17.6)	32 (25.0)	0.07
Time from admission to death from any cause			
30 days	8 (3.9)	22 (17.1)	<0.001
365 days	16 (7.7)	36 (27.9)	<0.001

Data are presented as number (percentage) of patients.

Abbreviations: Cx, left circumflex artery; GP IIb/IIIa, glycoprotein IIb/IIIa; LAD, left anterior descending artery; LMCA, left main coronary artery; PCI, percutaneous coronary intervention; RCA, right coronary artery; TIMI, thrombolysis in myocardial infarction

Assessment of 30- and 365-day mortality and in-hospital mortality In the multivariable Cox regression analysis of PVA-BP, only BE was an independent prognostic factor for both 30- and 365-day mortality in the whole study group. In the logistic regression analysis, lower values of BE were also associated with higher in-hospital mortality of the patients without OHCA (TABLES 3 and 4).

DISCUSSION

The main finding of this study is that in MI patients submitted initially to the ED and later treated with PCI, the evaluation of PVA-BP obtained from peripheral venous blood and assessed in POCT may be a useful tool for early risk stratification. Higher lactate level is associated with worse clinical condition. BE, AG, and lactates correlated with the GRACE score and BE was proved to be an independent predictor of both in-hospital, and post-discharge mortality.

The prognostic value of lactate as an indirect indicator of tissue hypoxia has been recognized for many years,[23,24] for example, in patients with multi-organ failure or sepsis,[24-26] but lactate metabolism is still far from being completely understood.[7] The clinical role of lactate in acute cardiac conditions has been also assessed and its high level is typically seen in MI patients with cardiogenic shock or advanced stages of other acute cardiac conditions.[27-29] Contrary to that, BE or AG may probably indicate a more subtle degree of acidosis.[13,14,30] So far, it has been confirmed that BE may be even superior to lactate levels in the prediction of mortality after a cardiac surgery.[31] There are also some studies confirming the usefulness of BE and AG as risk stratification tools in patients with MI,[12,13,32] however, there is a limited number of studies assessing the value of PVA-BP in the ED using POCT analyzers in the patients with symptoms of MI. Schmiechen et al[33] were the first to examine the use of venous lactate level in the ED to evaluate patients with chest pain. They found that lactate levels of 2.2 mmol/l translated into 96% sensitivity and 55% specificity in the diagnosis of MI. Later, Gatien et al[34] found that venous lactate is highly sensitive in the diagnosis of MI, particularly in patients with chest pain lasting for more than 2 hours.

We showed a strong relationship between high lactate values and the severity of clinical condition on admission, that is, lower blood pressure, higher Killip class, tachycardia, lower LVEF, and more frequently OHCA. The aforementioned findings confirm that regardless of the baseline clinical characteristics (no differences between groups in the history of previous MI, hypertension, and hypercholesterolemia were observed), lactate concentration is a marker of systemic hypoperfusion and hemodynamic instability. Other laboratory parameters of metabolic acidosis in the group of patients with higher lactate levels confirm this thesis. Similar observations were made by Lazzeri et al,[13] who reported coexistence of high lactate values, lower pH, BE, and higher AG values. In our study, even after exclusion of the OHCA patients, there were significant differences in the levels of lactates, AG, and BE between the hospital survivors and nonsurvivors.

The association between higher lactate values and both higher frequency of diabetes and higher serum glucose values on admission requires further explanation. The relationship between high lactate levels and hyperglycemia is complex and not fully understood but this phenomenon is not limited to the diabetic group. Acute hyperglycemia is a well-known marker of poor prognosis in patients with MI. It is also not limited to the diabetic group,[35-38] and it cannot be explained by poorly controlled or previously unrecognized diabetes. Increased level of lactate and glycemia are secondary to catecholamine action in stress conditions, with subsequent inflammatory response, a downregulation of insulin, and increased insulin resistance leading to myocardial damage. The mechanism described above may be even more significant in nondiabetic patients.[36,37] Moreover, it is known that lactate is the end product of glucose metabolism during hypoxia but it is possible that acute hyperglycemia in critically ill patients could be more directly linked to lactate metabolism.[39] As found in septic patients, accumulated lactate can be oxidized to pyruvate, transformed into glucose by gluconeogenesis or into glycogen via the Cori cycle.[40] Due to the aforementioned relationships, we decided to include glucose level on admission into our regression models. However, in our study, stress hyperglycemia

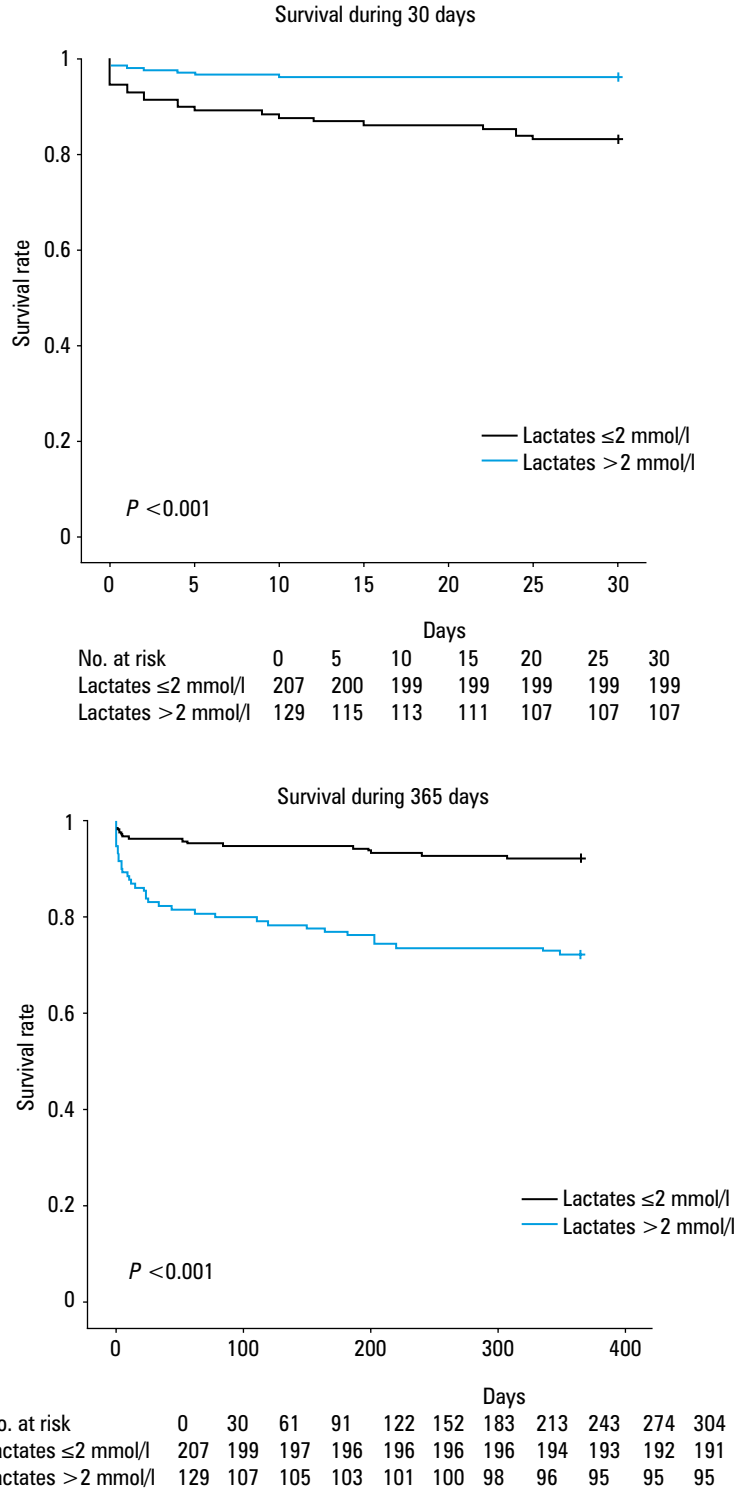

FIGURE 1 The Kaplan–Meier curve displaying cumulative survival in the whole study group, stratified by the lactate level on admission (≤2.0 mmol/l [n = 207] vs >2.0 mmol/l [n = 129])

seemed to be inferior in predicting mortality as compared with BE.

In our cohort, there was a prevalence of nonsmokers in patients with higher lactate concentrations—likely explained by the smoking paradox, a widely-known phenomenon where lower mortality rates are observed in smokers vs nonsmokers during MI. An explanation is that deleterious effects of smoking are manifested by the occurrence of MI earlier than in nonsmokers with similar age-adjusted risk factors.[41,42]

We did not find any differences in the frequency of a specific culprit artery depending on lactate level, however, the patients with acute total occlusion of the IRA had higher concentrations of lactates on admission than those without total occlusion of the IRA. This observation has significant clinical implications, because it is known that total occlusion results in a worse prognosis.[43,44] It is also known that both STEMI and NSTEMI can occur with or without complete occlusion of the IRA. Especially in NSTEMI,

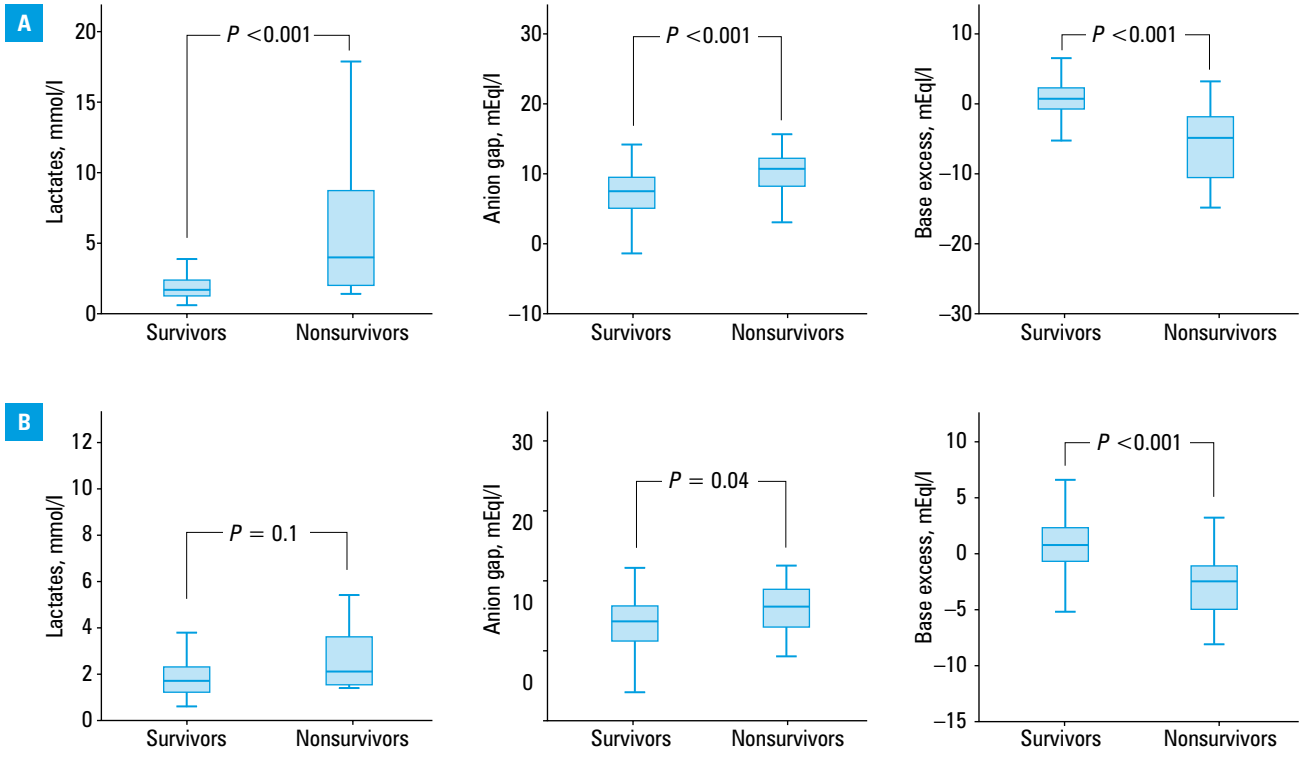

FIGURE 2 Lactate, anion gap, and base excess in the comparison between in-hospital survivors and nonsurvivors. Boxes represent the median and interquartile range. **A** – whole study group; **B** – subgroup of patients without out-of-hospital cardiac arrest

TABLE 3 Multivariable Cox regression analysis for 30- and 365-day mortality (whole study group)

Variable	Hazard ratio	95% CI	*P* value
Multivariable Cox regression analysis: 30-day mortality			
STEMI	6.28	1.70–23.13	0.006
LVEF (per 1%)	0.94	0.90–0.98	0.003
Female sex	1.47	0.53–4.07	0.46
Age (per 1 year)	1.03	0.98–1.07	0.25
Glucose (per 1 mmol/l)	0.96	0.86–1.07	0.96
Base excess (per 1 mEq/l)	0.79	0.65–0.95	0.01
Anion gap (per 1 mEq/l)	0.97	0.83–1.13	0.69
Lactates (per 1 mmol/l)	0.97	0.78–1.30	0.971
Multivariable Cox regression analysis: 365-day mortality			
STEMI	3.17	1.57–6.14	0.001
LVEF (per 1%)	0.96	0.94–0.99	0.004
Female sex	1.23	0.60–2.53	0.59
Age (per 1 year)	1.06	1.03–1.09	<0.001
Glucose (per 1 mmol/l)	1.01	0.93–1.09	0.87
Base excess (per 1 mEq/l)	0.89	0.76–0.99	0.04
Anion gap (per 1 mEq/l)	0.98	0.88–1.09	0.71
Lactates (per 1 mmol/l)	1.03	0.84–1.27	0.78

Abbreviations: see TABLE 1

the sensitivity and specificity of the ECG to determine if MI with total occlusion of the IRA is present is unsatisfactory,[4,45] leading to delays in revascularization. Any additional tool suggesting a MI with total occlusion is of crucial clinical importance.

It was observed that the patients with higher concentrations of lactates had a significantly shorter time from the symptom onset to the ED admission. This can be explained as patients with higher baseline lactate levels likely felt more pain and sought help more rapidly than the ones with lower lactate levels.[43,44]

Patients after OHCA are at a very high risk of death and the lactates in this population are an already known prognostic factor.[46] In order to avoid this cofounder, we performed sensitivity analysis and studied the role of PVA-BP in the subgroup of patients without OHCA. We confirmed that even after exclusion of the patients with OHCA, worse clinical condition was observed in the individuals with higher lactate levels. Moreover, BE, AG and lactate values differed significantly among the hospital survivors and nonsurvivors. Despite the fact that in the whole study group the patients with higher lactate levels (G2) had higher 30- and 365-day mortality in the Kaplan–Meier curves, we found that only BE was an independent predictor of mortality in both short- and long-term follow-up according to our Cox regression model analyses. Moreover, BE was also a significant risk factor for in-hospital mortality in the logistic regression model even after exclusion of the patients with OHCA. It can be suspected that lactic acidosis is a consequence of a more severe derangement (ie, patients with OHCA). Contrary to that, BE may be considered a significant marker even in acidosis of a subtle degree.

So far, a number of different scales of risk stratification in MI patients, with very good predictive

TABLE 4 In-hospital mortality logistic regression model (subgroup of patients without out-of-hospital cardiac arrest)

Variable	Odds ratio	95% CI	*P* value
STEMI	8.04	1.53–42.40	0.014
LVEF (per 1%)	0.92	0.88–0.97	0.002
Female sex	0.66	0.15–2.85	0.10
Age (per 1 year)	1.06	0.99–1.13	0.09
Glucose (per 1 mmol/l)	0.86	0.72–1.07	0.20
Base excess (per 1 mEq/l)	0.74	0.57–0.97	0.03
Anion gap (per 1 mEq/l)	1.16	0.91–1.49	0.24
Lactates (per 1 mmol/l)	0.78	0.40–1.53	0.47

Abbreviations: see **TABLE 1**

values for prognosis, have been validated. However, largely due to the need to consider many clinical, electrocardiographic, and laboratory parameters, they are often time-consuming and difficult to use routinely. One commonly used scale is the GRACE risk score, which helps to determine the urgency of invasive diagnostics in patients with NSTEMI.[2] Unfortunately, only cardiologists typically employ this scale. In our study, lactates, AG, and BE on admission in MI patients showed a significant correlation with the GRACE score calculated in the whole study group, and lactates and BE in the subgroup analysis. Troponins, on the other hand, were not found to correlate with the GRACE score; an important observation because physicians may be more prudent to qualify a patient for revascularization in ambiguous cases based on elevated lactate levels rather than to delay the decision while waiting for serial troponin levels. It is worth mentioning that when grouped based on lactate levels and consequently, more severe general conditions, troponin levels did not differ significantly between the lactate groups.

Our study has several limitations. Firstly, it was a single-center observational study. Furthermore, due to technical limitations, only patients admitted to the ED and not admitted directly to the catheterization lab were assessed. We must acknowledge that our results should be considered with caution because, at least theoretically, the levels of acid-base balance parameters may differ depending on both the blood sampling location and tourniquet time duration. However, there is evidence from numerous studies on the congruency between venous and arterial blood sample measurements that is satisfactory for acid-base balance parameters at least as a stratification tool.[5-10] Additionally, we conducted our analysis before the SARS-CoV-2 infection era, thus we did not assess the potential impact of COVID-19 on PVA-BP values in patients with MI.

Conclusions PVA-BP assessed in POCT analyzers may be a reliable tool for early risk stratification in patients admitted to the ED with MI treated with PCI. Elevated lactate levels are associated with worse clinical condition, however, BE seems to be a superior prognostic factor because it predicts mortality in short- and long-term observation in the whole study group as well as in-hospital mortality in the subgroup of patients without OHCA.

SUPPLEMENTARY MATERIAL

Supplementary material is available at www.mp.pl/paim.

ARTICLE INFORMATION

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