Prognostic accuracy of mean arterial pressure and serum lactate level in patients with acute myocardial infarction

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ABSTRACT

Background: Mean arterial pressure (MAP) can be used to evaluate macro-circulatory perfusion while serum lactate concentration is a marker of tissue perfusion. It is important to note that the primary objective of initial medical interventions is to restore microcirculatory perfusion rather than focusing solely on macro-hemodynamics.

Aims: We aimed to investigate the prognostic value of the combination of MAP and serum lactate levels measured on admission to the hospital in relation to patients' 30-day survival rate in patients with acute myocardial infarction (MI).

Methods: Data from 532 consecutive patients with acute MI treated with percutaneous coronary intervention were analyzed. The study endpoint was 30-day all-cause mortality.

Results: We found that both MAP and lactate levels were relevant predictors of the 30-day mortality in multivariable Cox regression analysis (HR, 0.83; 95% CI, 0.71–0.97; $P = 0.02$ and HR, 1.16; 95% CI, 1.06–1.16; $P = 0.01$, respectively). There was a significant increase in the prognostic performance in relation to 30-day mortality for the combination of both MAP and lactate levels in comparison to MAP alone ($P = 0.03$ for comparison between areas under the curve). Conversely, the combination of MAP and lactates did not add a significant prognostic value in comparison to lactates alone ($P = 0.53$ for comparison between areas under the curve).

Conclusions: In patients with acute MI, serum lactate level seems to have a higher prognostic value in comparison to MAP. Our data suggest that on initial assessment of patients with acute myocardial infarction, we should move toward a tissue perfusion-based approach instead of focusing on a blood pressure-oriented strategy alone.

Key words: hemodynamics, lactates, mean arterial pressure, myocardial infarction, tissue perfusion

INTRODUCTION

Myocardial infarction (MI) is a heterogeneous disease with a variety of clinical presentations. Distinguishing severity on admission to the hospital is essential for implementing advanced treatment to reduce the risk of mortality in this population. Blood pressure (BP) assessment is one of the most commonly used hemodynamic variables in the examination of the circulatory system. In cardiac physiology, it is widely accepted that hemodynamics refers to the cardiovascular function in relation to blood flow through the circulation. Thus, we may consider the mean arterial pressure (MAP) as a reflection of the flow, while pressure and resistance can be used to evaluate cardiac output and may indicate the condition of the circulatory system at the level of macro-hemodynamics [1]. When admitting patients with acute MI, physicians consider BP as one of the most important parameters to be normalized to ensure proper organ perfusion. However,

WHAT'S NEW?

Despite the pathophysiological rationale that mean arterial pressure (MAP) and lactate levels could serve as indicators for both macro- and microperfusion, the simultaneous prognostic value of MAP combined with serum lactate level on admission to the hospital has not been studied extensively in patients with acute myocardial infarction. Thus, we conducted a study to assess if simultaneous assessment of lactates and MAP may improve the prognostic accuracy of either of these markers alone in identifying patients at increased risk of 30-day mortality. In this study, we confirmed that the prognostic value of lactates seems to be higher in comparison to MAP. Our data suggest that on initial assessment of patients with acute myocardial infarction, we should aim to obtain such blood pressure levels that enable achieving relevant tissue perfusion at the microcirculatory level instead of focusing on a blood-pressure-oriented strategy alone.

the impact of BP on clinical outcomes in patients with acute cardiac conditions remains poorly understood. Therefore, it is essential to monitor not only BP parameters but also tissue perfusion indicators [2]. However, current guidelines and protocols are still mostly focused on macrocirculatory parameters, such as BP, instead of promoting more attention to the tissue perfusion-based approach [3]. In our routine practice, we assume that a lower MAP should correlate with a worse clinical outcome when assessing patients in critical condition, yet previous investigations were inconclusive regarding the ideal MAP level in critically ill patients including those with acute cardiac conditions [4–6].

Moreover, a growing number of studies indicate that traditional hemodynamic parameters may not be reliable, and strategies based on macro-hemodynamics optimization in individual patients may not be appropriate [7, 8]. Additionally, there is evidence to suggest that despite normalization of hemodynamics in some groups of patients, tissue hypoperfusion is still ongoing [9].

If generalizing and accepting the premise that MAP can be a surrogate of perfusion reflecting macro-circulation, it is easy also to approve that lactate level can be an adequate biomarker for assessment of tissue perfusion reflecting microvascular circulation. Moreover, there have been several publications supporting the prognostic importance of lactate levels in patients with acute MI [10–13]. It has been widely accepted that lactate levels may be considered an important hemodynamic endpoint at the cellular level in different clinical scenarios [14].

Despite the pathophysiological rationale that MAP and lactate levels could serve as indicators for perfusion both at macro- and microcirculatory levels, only a handful of published articles evaluated the simultaneous prognostic value of MAP on admission combined with markers of tissue perfusion at microcirculatory level in critically ill patients [15]. Moreover, there are no similar articles about acute MI patients. Our goal was to determine which of the indicators of the perfusion (MAP reflecting macrovascular circulation or serum lactate levels as a surrogate of microvascular perfusion) has a superior prognostic significance on admission in patients with MI. Thus, we decided to study if combining lactates with MAP may improve the prognostic accuracy of either MAP alone or lactates alone in identifying patients at increased risk of 30-day mortality. We hypothesized that lactate levels might have a greater prognostic value in this particular group of patients. We obtained a confirmation of our hypothesis, which should result in increasing physicians' awareness that instead of aiming for an ideal BP, we should aim for a BP that maintains adequate cellular perfusion at the microcirculatory level.

METHODS

Data collection and study endpoint

We retrospectively analyzed the medical records of unselected, consecutive patients who were admitted to the University Hospital, Kraków, Poland from January 1, 2019 to March 31, 2023. These patients had acute MI and were treated with percutaneous coronary intervention; they received standard medical therapy according to the European Society of Cardiology guidelines [13]. Each patient included in the study had serum lactate levels measured on admission to the hospital with a point-of-care analyzer. For BP measurements, systolic (SBP) and diastolic blood pressure (DBP) measurements were obtained by a physician during initial assessment using calibrated sphygmomanometers just after presentation to the emergency room. In our study, MAP was calculated as 1/3 (SBP) + 2/3 (DBP). Other clinical data including demographics, medical history, in-patient clinical course, laboratory results, treatment, and in-hospital outcomes were obtained from the electronic medical records used by the University Hospital in Krakow. The estimated glomerular filtration rate was calculated using the Modification of Diet in Renal Disease formula. The heart rate, Killip class, and Global Registry of Acute Coronary Events (GRACE 2.0) risk scores were assessed in all patients on admission. Death from any cause within 30 days of hospital admission was considered the study endpoint. We used the National Electronic Population Registration System in Poland to collect data concerning the incidence of death. The flowchart of the study is presented in Figure 1. The study was conducted in accordance with the guidelines of the Declaration of Helsinki and was approved by the Bioethics Committee of the Jagiellonian University, decision number 118.6120.118.2023.

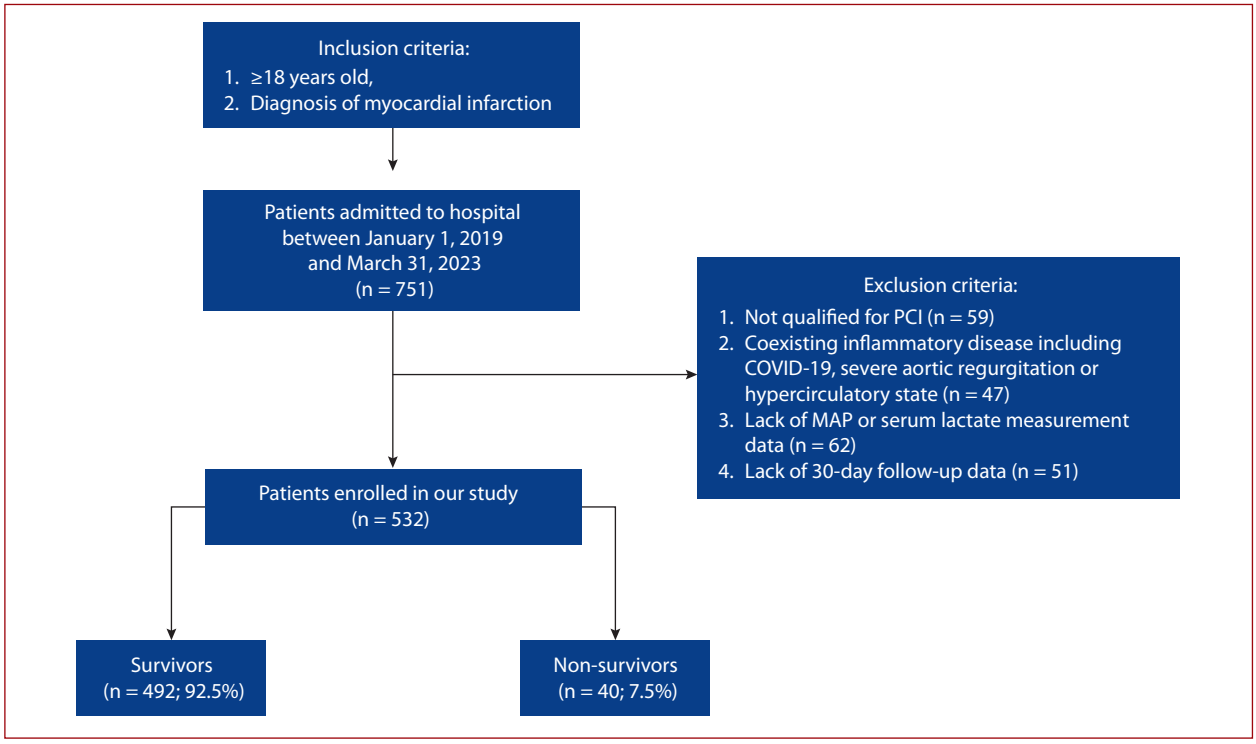

Figure 1. Flow chart of the study group

Abbreviations: MI, myocardial infarction; MAP, mean arterial pressure; PCI, percutaneous coronary intervention

Statistical analysis

Categorical variables were presented as numbers and percentages. Continuous variables were expressed as means and standard deviations (SD) or medians and interquartile ranges. Normality was assessed by the Shapiro–Wilk test. Equality of variances was assessed using Levene's test. Differences between groups were compared using Student's or Welch's t-tests depending on the equality of variances for normally distributed variables. The Mann–Whitney U-test was used for non-normally distributed continuous variables. Categorical variables were compared by Pearson's χ^2 test or by the Monte Carlo simulation for Fisher's test if 20% of cells had an expected count of less than 5. The area under the receiver operating characteristic curve (AUC) was calculated to assess the diagnostic capability of MAP, lactates, or a combination of both MAP and lactates on admission in predicting 30-day mortality. Receiver operating characteristic (ROC) curve analysis was performed, and in the next step, a comparison of AUCs was done with DeLong et al. [16] method to find if combining lactates with MAP may improve the prognostic accuracy of MAP alone or lactates alone in identifying patients at increased risk of 30-day mortality. Cox proportional hazard analyses were performed to assess the predictive value of studied parameters in terms of 30-day mortality. The prognostic relevance of clinically significant risk factors (i.e., age, sex, ST-segment elevation myocardial infarction [STEMI], arterial hypertension, diabetes mellitus, smoking, out-of-hospital cardiac arrest, history of MI, left ventricular ejection fraction <30%, heart rate on admission, MAP on admission, lactates level on admission, glomerular filtration rate on admission) was also tested in univariable and multivariable Cox-regression analyses. Parameters with *P*-value <0.05 in univariate analysis were included in multivariable analysis. The results were presented as hazard ratios (HR) with a 95% confidence interval. The proportional hazards model assumptions were checked using the Schoenfeld test and graphical diagnostics. The multicollinearity effect among predictors was excluded by calculating Variance Inflation Factors for each of the predictors included in the multivariable Cox regression model. The concordance index was assessed as Kendall's coefficient of concordance (Kendall's W = 0.920; *P* <0.001). Statistical analyses were performed with JMP®, Version 14.2.0 (SAS Institute INC., Cary, NC, US) and SPSS® Version 22 (IBM Corp.®).

RESULTS

Clinical characteristics of study subjects

Our study group comprised 532 patients with acute MI (70% males). The mean (SD) age was 67 (12.5) years. There were 232 (44%) STEMI and 300 (56%) non-ST-segment elevation myocardial infarction patients. Arterial hypertension (70%) and diabetes mellitus (27%) were the predominant co-existing diseases. There were 58 patients (11%) with a Killip class of 3 or 4. Out-of-hospital cardiac arrest occurred in 24 cases (4.5%). The 30-day all-cause mortality rate was 7.5% (n = 40). On admission, the mean (SD) MAP was 101.3 (19.5) mmol/l, and the median (interquartile range) serum lactate level was 1.5 (1.1–2.1) mmol/l.

Table. 1 Baseline clinical characteristics and patient's outcome according to 30-day survival status

Parameter	Survivors (n = 492; 92.5%)	Non-survivors (n = 40; 7.5%)	*P*-value
Age, years, mean (SD)	66.1 (12.2)	78.2 (11.7)	<0.001
Sex, male, n (%)	346 (70.3)	26 (65.0)	0.29
STEMI, n (%)	208 (42.3)	24 (60.0)	0.02
Diabetes mellitus, n (%)	131 (26.6)	15 (37.5)	0.01
Myocardial infarction in the history, n (%)	94 (19.1)	7 (17.5)	0.50
Arterial hypertension, n (%)	347 (70.5)	27 (67.5)	0.40
Smoking, n (%)	171 (34.8)	4 (10.0)	0.001
Killip 3 or 4 on admission, n (%)	32 (6.5)	26 (65.0)	<0.001
Out-of-hospital cardiac arrest, n (%)	13 (2.6)	11 (27.5)	<0.001
Left ventricular ejection fraction <30%, n (%)	91 (18.5)	27 (67.5)	<0.001
Heart rate, min^{-1}, median (IQR)	80 (70–90)	87 (75–108)	0.007
Systolic blood pressure, mm Hg, mean (SD)	145 (29)	119 (40)	<0.001
Diastolic blood pressure, mm Hg, median (IQR)	80 (71–90)	66.5 (51–79)	<0.001
Mean arterial pressure, mm Hg, mean (SD)	103 (18)	83 (26)	<0.001
Lactates (mmol/l), median (IQR)	1.5 (1.1–2.0)	2.4 (1.5–6.5)	<0.001
Troponin hsTnI (µg/l), median (IQR)	3589 (774–13520)	5198 (1380–25000)	0.197
eGFR, ml/min/1.73 m^2, median (IQR)	89 (68–110)	57 (37–75)	<0.001
GRACE score, median (IQR)	132 (109–155)	225 (179–257)	<0.001
Multivessel disease, n (%)	176 (35.8)	20 (50.0)	0.05

Abbreviations: eGFR, estimated glomerular filtration rate; GRACE, Global Registry of Acute Coronary Events; IQR, interquartile range; SD, standard deviation; STEMI, ST-elevation myocardial infarction.

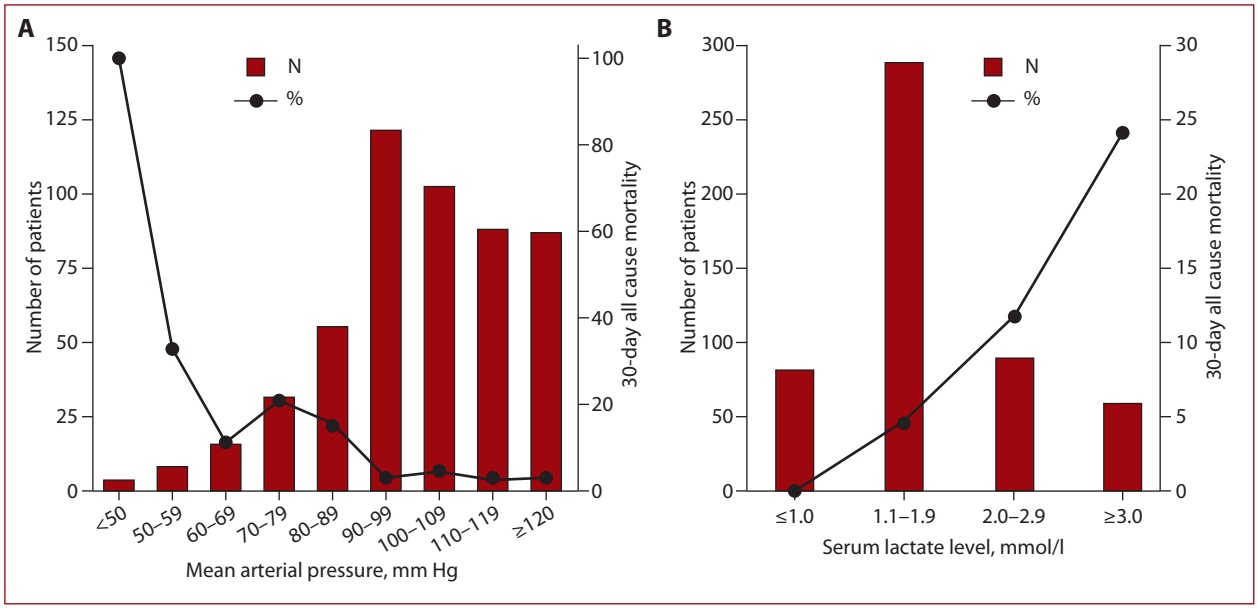

Figure 2. Number of subjects in relation to 30-day all-cause mortality according to: **A.** Decreasing values of mean arterial pressure MAP. **B.** Increasing values of serum lactate concentration

Association between MAP, serum lactate levels, and 30-day mortality

The clinical characteristics of studied patients are summarized in Table 1, Figure 2, and Supplementary material, *Figure S1*. There were 492 patients in the survivor group and 40 patients in the non-survivor group. Non-survivors had lower values of MAP and higher lactate concentrations in comparison to survivors (Table 1, Figure 2). Univariable and multivariable Cox proportional hazard models showed that both MAP and lactates are risk factors for 30-day mortality in patients with acute MI (Table 2).

The predictive value of MAP and lactates

Based on the ROC curve, MAP and lactate concentration on admission had a moderately strong predictive value for 30-day mortality (AUC 0.74; *P* <0.001 and AUC 0.75; *P* <0.001, respectively). The AUC for the combination of both lactates and MAP was 0.78 (*P* <0.001) (Supplementary material, *Figure S2*). We then compared the AUCs and confirmed that there was no difference between MAP and lactates in predictive performance for 30-day mortality. However, there was a significant increase in prognostic performance in relation to 30-day mortality for the combination of both

Table 2. Univariable and multivariable Cox regression model assessing the risk of death at a 30-day follow-up

Risk factor	Univariable Cox regression		Multivariable Cox regression	
	HR (95% CI)	*P*-value	HR (95% CI)	*P*-value
Age (per 1 year)	1.09 (1.06–1.12)	<0.001	1.06 (1.03–2.67)	<0.001
Male sex	1.27 (0.66–2.44)	0.47		
STEMI	2.01 (1.07–3.79)	0.03	1.96 (1.01–3.82)	0.047
Out-of-hospital cardiac arrest	9.56 (4.77–19.16)	<0.001	3.91 (1.77–8.65)	<0.001
Arterial hypertension	0.88 (0.45–1.70)	0.69		
Smoking	0.22 (0.08–0.61)	0.004	0.87 (0.28–2.70)	0.81
Diabetes mellitus	1.62 (0.86–3.08)	0.14		
History of MI	0.91 (0.40–2.06)	0.82		
LVEF <30%	8.22 (4.24–15.93)	<0.001	3.52 (1.67–7.40)	0.001
Heart rate on admission (per 1 min^{-1})	1.03 (1.01–1.05)	<0.001	1.02 (1.001–1.03)	0.04
MAP on admission (per 10 mm Hg)	0.63 (0.55–0.73)	<0.001	0.83 (0.71–0.97)	0.02
Lactates level on admission (per 1 mmol/l)	1.28 (1.21–1.36)	<0.001	1.16 (1.06–1.27)	0.01
eGFR on admission, (per 1 ml/min/1.73 m^2)	0.97 (0.96–0.98)	<0.001	0.97 (0.973–0.999)	0.03

Abbreviations: CI, confidence interval; HR, hazard ratio; LVEF, left ventricular ejection fraction; other — see Figure 1 and Table 1

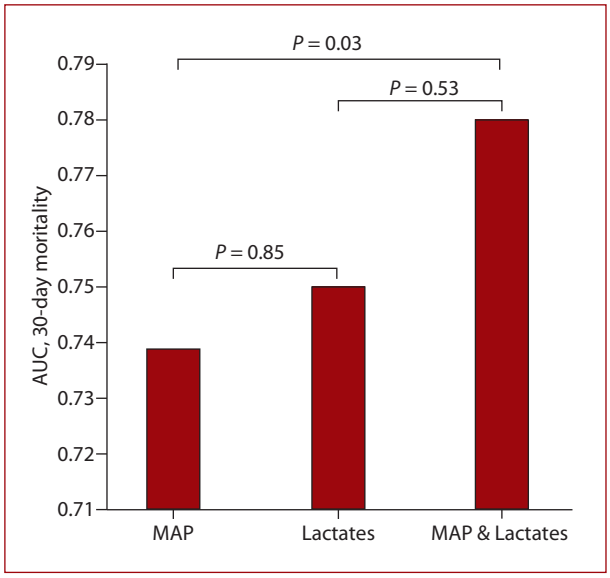

Figure 3. Comparison of area under the curve for mean arterial pressure (MAP), serum lactate levels and combination of MAP and serum lactate levels on admission

MAP and lactates in comparison to MAP alone. On the contrary, adding MAP to lactates and studying the combination of both MAP and lactates did not significantly improve the prognostic performance in comparison to an analysis of lactates alone (Figure 3).

DISCUSSION

Our study confirms that among patients with acute MI, non-survivors have on average lower MAP and higher levels of serum lactate concentrations on admission. Based on the multivariable analysis, we confirmed that lower MAP values and higher serum lactate levels independently increased the risk of death at 30-day follow-up. Based on the ROC curves, there is no difference between MAP alone and lactates alone in predictive performance for 30-day mortality. However, the combination of both MAP and lactates has improved predictive value in comparison to MAP alone. On the contrary, the combination of MAP and lactates did not add the prognostic value significantly in comparison to lactates alone. We can conclude that lactates seem to have a superior prognostic value in comparison to MAP in an unselected group of patients with acute MI. In our opinion, this supports the thesis that MAP corresponds to the state of perfusion in the macrovascular circulation only. Thus, we should reconsider our beliefs on the purpose of monitoring hemodynamics, and instead of trying to achieve ideal macro-hemodynamic norms, we should strive to normalize surrogates of cellular perfusion and consider it our superior therapeutic target.

Over the years several studies have confirmed the importance of BP on admission as a prognostic factor in acute MI — for example, this parameter has been included in such risk scales as GRACE, or TIMI Risk Score [17]. Moreover, the prognostic value of other BP indexes, such as SBP, DBP, pulse pressure, or MAP alone, has been demonstrated in many clinical scenarios [18]. Additionally, guidelines for the initial management of patients in unstable condition due to MI stressed the importance of maintaining adequate systemic hemodynamic function through the optimization of BP and cardiac output in addition to immediate revascularization of the infarct-related artery [19]. Moreover, most practitioners consider low BP in patients with acute MI as undesirable, even though patients often do not display any other symptoms of direct endangerment of their circulatory system. However, there is more and more evidence indicating that the correlation between macro- and microcirculation in unstable patients is rather poor [20]. Additionally, some experimental studies have shown that vasodilation-induced arterial hypotension can even lead to the improvement of microcirculatory blood flow instead of decreasing the microcirculatory flow [21]. It has been also shown that in patients with reduced left ventricular ejection fraction, any vasopressor-mediated increase in vascular resistance augments left ventricular afterload and may reduce cardiac output leading to little or no benefit

for global tissue perfusion [22]. All the above investigations suggest that we should reconsider our approach to the assessment of the cardiovascular system in patients with acute MI, and we should strive to obtain a MAP that is sufficient to provide adequate tissue perfusion. It has been indisputably confirmed that lactate, which is the metabolic end-product of anaerobic glycolysis, may serve as the most basic and readily available marker of tissue perfusion at the microcirculation level [10].

Hyperlactatemia is a well well-known prognostic marker in patients with acute cardiac conditions [23, 24], but the results of our work provide new insights into this issue: in contrast to the aforementioned studies, we attempted a simultaneous comparative assessment of the prognostic significance of markers of circulatory perfusion at both macro- and microperfusion levels. We postulate that abnormally low MAP values may be an indication to expand the diagnostic workup to include biochemical confirmation of tissue perfusion impairment using the lactate measurement. Once hyperlactatemia is confirmed, prompt efforts should be taken to restore the abnormality. In the context of acute MI patients, this means not only effective revascularization of the infarct-related artery without unnecessary delay using novel diagnostic and/or therapeutic devices [25], but also more rapid introduction of interventions to optimize cardiac output or improve oxygen delivery to tissues (catecholamines, diuretics, or fluid supplementation, oxygen therapy, correction of hemoglobin level, etc.). Based on the results of our study, we would like to introduce a wider perspective on the perception of hemodynamics in which BP measurement should only be treated as an indirect indicator of the instability of circulation, and serum lactate levels should be used to fully assess microcirculatory perfusion efficiency.

Limitations

Firstly, this study was a single-center observational and retrospective study. This method of data collection is prone to misclassification and selection bias. Further validation from multiple centers can help ascertain that these results are consistent. Single serum lactate concentrations and MAP assessments carried out on admission may reduce the reliability of our data, but in the real-world emergency setting, physicians often do not have time or diagnostic capabilities to reassess these parameters multiple times due to the patient's condition. Moreover, we did not include in our analysis the potential relationship between medications used by patients, such as vasopressors, and their impact on MAP and lactate values. Moreover, MAP was calculated with the use of non-invasive techniques and mathematical formulas. Additionally, the size of our study sample was insufficient to perform a detailed sub-analysis of patients with shock and patients who suffered from out-of-hospital cardiac arrest. Due to lack of data on the time from symptom onset to revascularization, we did not analyze the correlation between the time of ischemia and lactate levels.

CONCLUSIONS

In unselected patients with acute MI, the prognostic value of lactates seems to be superior in comparison to MAP in relation to 30-day mortality. Our observations support the hypothesis that we should reconsider our goal to monitor hemodynamic parameters in patients with acute MI and instead of striving to achieve the optimal blood pressure, we should aim to obtain blood pressure levels sufficient to restore adequate tissue perfusion at the microcirculatory level.

Supplementary material

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

Article information

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