Clinical and Applied Thrombosis/Hemostasis
Volume 26: 1-6

DOI: 10.1177/1076029620933944
journals.sagepub.com/home/cat
SAGE

Optimal Mean Arterial Pressure Within 24 Hours of Admission for Patients With Intermediate-Risk and High-Risk Pulmonary Embolism

Jialong Chen, MD[1], Jing Lin, MD[1], Danshen Wu, MD, PhD[2], Xiaolan Guo, MD[1], XiuHua Li, MD[1], and Songjing Shi, MD[1,2]

Abstract

We aimed to determine whether the average mean arterial pressure (aMAP) in the first 24 hours of hospital admission is useful in predicting short-term outcomes of patients with intermediate- and high-risk pulmonary embolism (PE). We conducted a single-center retrospective study. From May 2012 to April 2019, 122 patients with intermediate- and high-risk PE were included. The primary outcome was in-hospital mortality. The secondary outcome was adverse events. Receiver operating characteristic (ROC) curves and cutoff values for aMAP predicting in-hospital death were computed. According to cutoff values, we categorized 5 groups defined as follows: group 1: aMAP < 70 mm Hg; group 2: 70 mm Hg ≤ aMAP < 80 mm Hg; group 3: 80 mm Hg ≤ aMAP < 90 mm Hg; group 4: 90 mm Hg ≤ aMAP <100 mm Hg; and group 5: aMAP ≥ 100 mm Hg. Cox regression models were calculated to investigate associations between aMAP and in-hospital death. In the study group of 122 patients, 15 (12.30%) patients died in the hospital due to PE. The ROC analysis for MAP predicting in-hospital death revealed an area under the curve of 0.729 with a cutoff value of 79.4 mm Hg. Cox regression models showed a significant association between in-hospital death and aMAP group 1 (ref), aMAP group 2 (odds ratio [OR] = 1.680, 95% CI: 0.020-140.335), aMAP group 3 (OR = 0.003, 95% CI: 0.0001-0.343), aMAP group 4 (OR = 0.006, 95% CI: 0.0001-1.671), and aMAP group 5 (OR = 0.003, 95% CI: 0.0001-9.744). In particular, those with an aMAP of 80 to 90 mm Hg had minimum adverse events. The optimal range of MAP for patients with intermediate- and high-risk PE may be 80 to 90 mm Hg.

Keywords

pulmonary embolism, intermediate risk, high risk, mean arterial pressure, mortality, adverse events

Date received: 03 March 2020; revised: 14 May 2020; accepted: 20 May 2020.

Introduction

Acute pulmonary embolism (PE) is the third-most common diagnosis in cardiovascular disease and can be the source of significant morbidity and mortality, especially in intermediate- and high-risk patients, whose mortality in 30 days[1,2] is >10%. For low-risk patients, mortality at 30 days was rarely 1.0%. Thus, patients with intermediate- and high-risk PE should receive more attention.

The most recent guidelines emphasize the central role of the early risk assessment of patients with acute PE.[3] Systolic blood pressure (BP) and diastolic BP values are fundamental and integral to the assessment of severity. Previous articles have already proven the prognostic role of systolic BP in patients with acute PE.[4,5] Therefore, systolic BP <100 mm Hg was incorporated into the Simplified Pulmonary Embolism Severity Index (sPESI)[6] and the Pulmonary Embolism Severity Index (PESI).[1] In addition, diastolic BP ≤65 mm Hg at admission was related to in-hospital mortality.[4] In fact, mean arterial pressure (MAP), which is critical to organ and tissue perfusion, is calculated as 1/3 systolic BP plus 2/3 diastolic BP. Moreover, MAP is relevant to heart rate, left ventricular contractility, and

[1] Shengli Clinical Medical College of Fujian Medical University, China
[2] Medical Intensive Care Unit of Fujian Provincial Hospital, China

Corresponding Author:
Songjing Shi, Medical Intensive Care Unit, Fujian Provincial Hospital, 134 East Street, Fuzhou 350001, China.
Email: serena10@126.com

right heart dysfunction.[7] For critically ill patients, the MAP level was closely related to mortality.[8] However, no study to date has investigated the relationship between MAP and the outcome of patients with intermediate- and high-risk PE. In this study, we sought to determine optimal MAP within 24 hours of admission for patients with intermediate- and high-risk PE.

Methods and Patients

We performed a retrospective analysis of patients with acute intermediate- and high-risk PE who were treated in the Fujian Provincial Hospital between May 2012 and April 2019. The local ethical board approved the study.

In our retrospective study, eligible patients were required to be confirmed by computed tomography pulmonary angiography (CTPA) or pulmonary angiography, the availability of patient records, and age ≥18 years. Conversely, pregnant women were excluded from the study. The classification of PE severity was performed according to the 2019 ESC Guidelines for the diagnosis and management of acute PE, developed in collaboration with the European Respiratory Society.

The final study group included 122 patients (median age 68.5 years, 60 females). The treatment methodology was the same during the study period, following the guidelines on the diagnosis and management of acute PE.[9,10] The primary outcome of this study was in-hospital mortality. The secondary outcome of this study was adverse events.

Definitions

Adverse events were defined as cardiogenic shock (systolic BP <90 mm Hg), cardiopulmonary resuscitation, mechanical ventilation, vasopressor therapy, and thrombolysis.

Clinical and Instrumental Evaluation of PE

Blood pressure measurement frequencies at the first 24 hours of hospital admission were dependent on their severity. For patients with intermediate-risk PE, BP was measured with a 6-hour interval by an automated noninvasive BP machine. For patients with high-risk PE, BP needed to be measured with a 2-hour interval by an automated invasive or noninvasive BP machine. The average mean arterial pressure (aMAP) was established by using the sum of the MAP measurements divided by the number of BP measurements during the 24 hours.

Classification of PE Severity

Hemodynamic instability, combined with PE confirmation on CTPA and/or evidence of right ventricular (RV) dysfunction on transthoracic echocardiogram (TTE), is sufficient to classify a patient into the high-risk PE category. Hemodynamic instability was determined as follows: cardiac arrest, obstructive shock (systolic BP <90 mm Hg or vasopressors required to achieve a BP ≥90 mm Hg despite an adequate filling status, in combination with end-organ hypoperfusion), or persistent hypotension (systolic BP <90 mm Hg or a systolic BP drop ≥40 mm Hg for >15 minutes, not caused by new-onset arrhythmia, hypovolemia, or sepsis). Patients with signs of RV dysfunction on TTE (or CTPA), elevated cardiac biomarker levels, and PESI classes III to V or sPESI ≥ I should be classified into the intermediate–high-risk category. Patients with signs of RV dysfunction or elevated cardiac biomarkers, despite a low PESI or an sPESI of 0, should be classified into the intermediate–low-risk category.

Statistical Analysis

The Kolmogorov-Smirnov test was performed to verify the normality of the distribution of the data. Data are presented as the mean ± SD if normally distributed or the median (interquartile range) if not normally distributed. Continuous variables were compared with Student *t* test if the data had a normal distribution or the Mann-Whitney *U* test or Wilcoxon rank-sum test for non-normally distributed variables between the 2 groups. The Kruskal-Wallis (non-normal distribution) test was used to compare >2 groups. Categorical variables, presented as a percentage, were compared by Pearson χ^2 test. We calculated a receiver operating characteristic (ROC) curve with the area under the curve (AUC) to establish the optimal cutoff for aMAP as a predictor of in-hospital mortality. In particular, Youden index (YI = sensitivity + specificity − 1) was also adopted to estimate and confirm the optimal cutoff value from the ROC curve analysis. Univariate and multivariate analyses for the assessment of independent-risk predictors were performed using the Cox regression model.

We performed statistical analyses with SPSS version 24 (SPSS). All hypothesis tests were 2 sided, with a significance level of .05.

Results

In the study group of 122 patients, 15 (12.30%) patients died in the hospital due to PE. The baseline characteristics of the study groups are outlined in Table 1.

The survival group and nonsurvival group had comparable rates of previous conditions, such as hypertension, diabetes, and chronic heart failure. Compared to the survival group, the nonsurvival group had higher white blood cell count, aspartate aminotransferase (AST), alanine aminotransferase (ALT), and D-dimer. Average mean arterial pressure, systolic BP, diastolic BP, and the estimate glomerular filtration rate were significantly lower in the nonsurvival group. The nonsurvival group had a higher percentage of high-risk (53.3% vs 11.2%, $P < .001$) and a lower percentage of intermediate–low-risk patients (20.0% vs 75.7%, $P < .001$) as well as a more severe classification.

Calculated ROC analysis for aMAP predicting in-hospital death revealed an AUC of 0.729 with an aMAP cutoff value of 79.4 mm Hg, with patients with PE included. The percentages of sensitivity, specificity, and Youden index were calculated as 80.5%, 60.0%, and 0.405, respectively (Figure 1).

Table 1. Baseline Characteristics of Overall, Survival, and Nonsurvival Groups.

	Overall, N = 122	Survival, N = 107	Nonsurvival, N = 15	*P* value
Female, n (%)	60 (49.2)	50 (46.7)	10 (66.7)	.242
Age (years)	68.50 (54.00-79.00)	70.00 (56.00-79.00)	60.00 (44.00-71.00)	.058
Previous conditions				
Diabetes, n (%)	13 (10.7)	11 (10.3)	2 (13.3)	1.000
Hypertension, n (%)	46 (37.7)	42 (39.3)	4 (26.7)	.511
Stroke, n (%)	17 (13.9)	16 (15.0)	1 (6.7)	.638
Chronic heart failure, n (%)	21 (17.2)	18 (16.8)	3 (20.0)	1.000
Cancer, n (%)	23 (18.9)	21 (19.6)	2 (13.3)	.817
Arterial fibrillation, n (%)	16 (13.1)	13 (12.1)	3 (20.0)	.663
Clinical data				
White blood count ($\times 10^9$/L)	8.10 (5.60-11.77)	7.80 (5.40-11.00)	13.20 (7.40-17.50)	<.001
Hemoglobin (g/L)	125.31 ± 20.25	126.81 ± 19.20	114.57 ± 24.71	.084
Platelet ($\times 10^9$/L)	204.00 (157.00-258.00)	204.00 (165.00-262.00)	167.00 (132.00-252.00)	.133
Albumin (g/L)	36.00 (33.00-41.00)	37.00 (33.00-41.00)	32.00 (27.00-43.00)	.103
ALT (U/L)	23.00 (13.00-47.00)	21.00 (12.00-38.00)	41.00 (28.00-235.00)	.002
AST (U/L)	25.00 (18.00-45.00)	23.00 (17.00-39.00)	48.00 (27.00-245.00)	.003
Uric acid (mmol/L)	347.25 ± 134.81	336.34 ± 123.30	425.07 ± 185.92	.189
Estimated glomerular filtration rate (mL/(min $\times$ 1.73 m^2)	84.90 (68.55-102.78)	85.44 (70.72-104.49)	73.15 (34.46-89.73)	.026
HDL (mmol/L)	1.02 (0.77-1.22)	1.04 (0.80-1.22)	0.93 (0.59-1.34)	.326
LDL (mmol/L)	2.75 (2.02-3.44)	2.78 (2.07-3.69)	2.20 (1.25-3.05)	.080
Troponin I (pg/mL)	0.02 (0.01-0.16)	0.02 (0.01-0.15)	0.06 (0.01-1.17)	.112
D-Dimer (mg/L)	4.11 (1.77-8.27)	4.00 (1.77-7.16)	9.88 (0.93-17.41)	.037
MAP (mm Hg)	89.50 (80.67-96.44)	90.00 (83.67-97.17)	77.17 (64.33-90.67)	.004
Systolic BP (mm Hg)	121.05 ± 19.46	122.51 ± 19.44	110.66 ± 16.73	.021
Diastolic BP (mm Hg)	74.00 (65.00-80.00)	74.25 (67.00-80.00)	64.40 (58.00-77.50)	.051
Classification of pulmonary embolism severity				
Intermediate-low risk, n (%)	84 (100)	81 (75.7)	3 (20.0)	<.001
Intermediate-high risk, n (%)	18 (100)	14 (13.1)	4 (26.7)	.317
High risk, n (%)	20 (100)	12 (11.2)	8 (53.3)	<.001

Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; BP, blood pressure; HDL, high-density lipoprotein; LDL, low-density lipoprotein; MAP, mean arterial pressure.

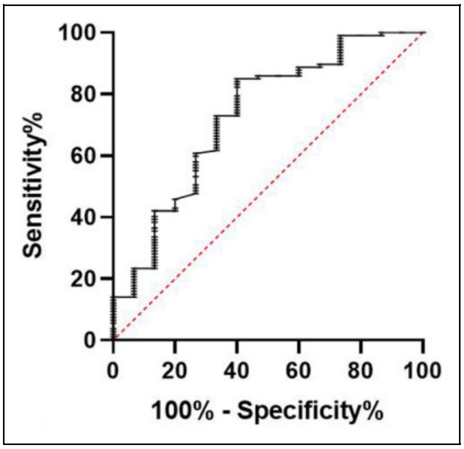

Figure 1. Receiver operating characteristic curve with area under the curve and Youden index were calculated to the test the effectiveness of mean arterial pressure to predict in-hospital death.

According to the aMAP cutoff value, we categorize 5 groups defined as follows: group 1: aMAP < 70 mm Hg; group 2: 70 mm Hg ≤ aMAP <80 mm Hg; group 3: 80 mm Hg ≤ aMAP < 90 mmHg; group 4: 90 mm Hg ≤ aMAP < 100 mm Hg; and group 5: aMAP ≥ 100 mm Hg. Comparing the number of adverse events among the different groups, we found that group 1 had the most adverse events. Conversely, the number of adverse events for group 2 was the lowest. Comparing to group 1 and group 2, MAP from 80 to 90 mm Hg can significantly reduce adverse events. However, MAP > 90 mm Hg seems not helpful in reducing adverse events (Figure 2).

The characteristics of the MAP groups are outlined in Table 2. Group 4 was the oldest of all the groups (P = .023). Among the groups above, the albumin level in group 1 was the lowest. There were no differences in hemoglobin, ALT, AST, uric acid, estimate glomerular filtration rate, or troponin I among the groups described above.

The Cox proportional hazards regression method was used for univariable and multivariable analysis. For univariable analysis, group 3 was a protective factor against in-hospital death (odds ratio [OR] = 0.051, 95% CI: 0.006-0.460). In multivariable analysis (model 2 and model 3 in Table 3), group 3 remained a protector factor for in-hospital death (OR = 0.011, 95% CI: 0.0001-0.442 and OR = 0.003, 95% CI: 0.0001-0.343, respectively) compared with group 1. In model 2 and model 3, group 1, group 4, and

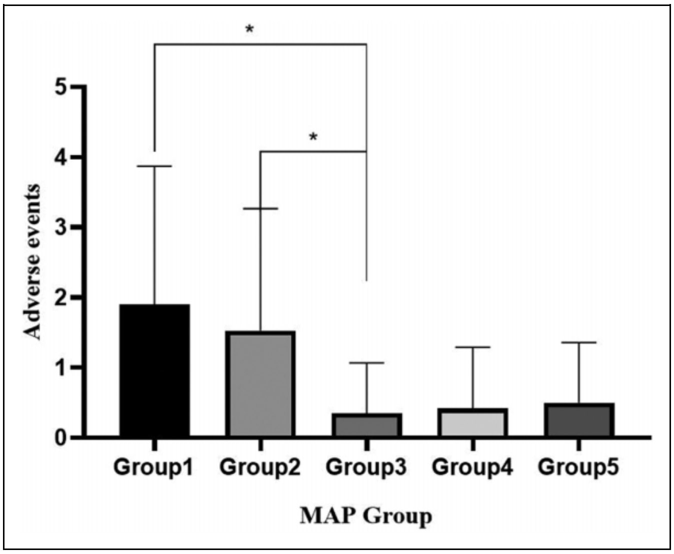

Figure 2. The adverse events according to mean arterial pressure group (*P < .05).

group 5 were not protective or hazardous factors for in-hospital mortality.

Discussion

In this study, we found that the in-hospital mortality of patients with aMAP < 80 mm Hg was considerably higher than that of patients with aMAP ≥ 80 mm Hg. It seems that aMAP ranging from 80 to 90 mm Hg was beneficial to patients with PE.

Pulmonary embolism is currently one of the most common critical diseases. Longitudinal studies have indicated that the incidence of PE continues to increase.[11,12] However, with different severities, mortality varied widely. For patients with intermediate- and high-risk PE, mortality was more than 10-fold that of low-risk patients.[1] That is, they accounted for most patients with PE who died within 30 days. In our view, these patients warrant more attention. Therefore, only patients with intermediate- and high-risk PE were included in this study.

To the best of our knowledge, this is the first study to investigate the relationship between aMAP and in-hospital death in patients with intermediate- and high-risk PE. Previous studies have indicated that MAP is involved in right heart dysfunction, which is generally acknowledged to be pertinent to adverse outcomes. When the pulmonary arterial bed is occluded by thromboembolism, pulmonary vascular resistance, and RV afterload increase. As a result, the RV contraction time was prolonged, and leftward bowing of the interventricular septum occurred. This may result in a decrease in cardiac output and contribute to systemic hypotension and hemodynamic instability. In the PESI and the sPESI, a systolic BP of <100 mm Hg is one of the parameters predicting adverse outcomes.[1] However, limited studies have focused on diastolic BP. Coronary arteries perfuse cardiac myocytes during diastole. Diastolic BP is essential to coronary flow. Patients with PE often have myocardial infarction. Waits et al[13] found that low diastolic BP < 70 mm Hg in participants without a history of cardiovascular disease whose systolic BP > 140 mm Hg still carries an increased risk of subclinical myocardial injury. There is a J-shaped curve relationship between diastolic BP during treatment and myocardial infarction, and the optimal diastolic BP was between 85 and 90 mm Hg.[14] A recent study demonstrated that patients with PE with diastolic BP ≤65 mm Hg at admission are susceptible to in-hospital death.[4]

Mean arterial pressure was determined as 1/3 systolic BP plus 2/3 diastolic BP. The evaluation of MAP has turned out to improve the outcome in different life-threatening illnesses, such as sepsis resuscitation, ischemic stroke, and distributive shock.[15] Moreover, MAP fluctuations between −5% and 5% were significantly related to intensive care unit (ICU) mortality (OR = 1.296; 95% CI: 1.103-1.521; P = 0.002) and hospital mortality (OR = 1.323; 95% CI: 1.142-1.531; P < .001).[16] Consequently, MAP may be a better indicator than systolic BP that reflects the hemodynamic profile.

Mean arterial pressure at admission has been identified as a risk factor for ICU mortality and an indicator of kidney perfusion.[8,17] Computed ROC analysis showed an optimal aMAP cut-off value of 79.4 mm Hg for the prediction of in-hospital death in patients with PE (specificity 0.805, sensitivity 0.600, AUROC 0.729). Current guidelines recommend targeting an MAP ≥ 65 mm Hg in patients with sepsis.[18] However, the optimal MAP remains an area of debate. In this study, we found that the optimal aMAP for patients with PE was 80 to 90 mm Hg. In group 4 and group 5, 4 patients died in the hospital (2 received vasopressor therapy, 1 had uncontrolled hypertension). It seems that keeping aMAP ≥ 90 mm Hg would not benefit patients with PE. It has been proven that a higher MAP is detrimental to patients, especially to patients without hypertension.[19,20] Too many doses of vasopressors, such as norepinephrine and dobutamine, may contribute to higher MAP levels. Excessive vasoconstriction may deteriorate tissue perfusion and trigger or aggravate arrhythmias.[21] Vasopressors could increase RV inotropy and systemic BP, promote positive ventricular interactions, and lower filling pressures[3]; however, raising cardiac output may aggravate the ventilation/perfusion mismatch by further redistributing flow from (partly) occluded to unoccupied arteries.[22] Uncontrolled hypertension could increase left ventricular afterload.

Previous studies have demonstrated no significant improvement in renal function when targeting higher MAP values in septic shock.[23,24] We also found no differences in kidney function among different MAP groups in this article.

We defined adverse clinical events as cardiogenic shock, cardiopulmonary resuscitation, mechanical ventilation, vasopressor therapy, and thrombolysis. A low level of MAP may in fact be a presentation of cardiogenic shock and an indication of vasopressor therapy. This is the reason why patients with aMAP < 70 mm Hg had maximum adverse events. However, in particular, those with an aMAP of 80 to 90 mm Hg had minimum adverse events. Interestingly, an aMAP from 80 to 90 mm Hg was also a protective factor against in-hospital mortality.

There are some limitations to this study that should be considered. First, this was a single-center study conducted in a

Table 2. Characters Between MAP Groups.[a]

Characterize	MAP					
	Group 1, n = 10	Group 2, n = 16	Group 3, n = 38	Group 4, n = 36	Group 5, n = 21	*P* value
In-hospital mortality, n (%)	4 (40.00)	5 (31.25)	2 (5.26)	3 (8.33)	1 (4.45)	<.001
Inotropic agents, n (%)	7 (70)	7 (43.8)	3 (7.9)	2 (5.6)	1 (4.8)	<.001
Female, n (%)	3 (30.0)	8 (47.1)	20 (54.1)	20 (55.6)	9 (40.9)	.555
Age (years)	59.50 (49.006-7.00)	60.00 (43.00-78.00)	69.00 (44.00-76.00)	77.00 (62.50-81.00)	68.50 (64.00-79.00)	.023
Previous conditions						
Diabetes, n (%)	0 (0.0)	0 (0.0)	4 (10.8)	5 (13.9)	4 (18.2)	.339
Hypertension, n (%)	3 (30.0)	3 (17.6)	10 (27.0)	15 (41.7)	15 (68.2)	.008
Stroke, n (%)	1 (10.0)	1 (5.9)	4 (10.8)	7 (19.4)	4 (18.2)	.692
Chronic heart failure, n (%)	0 (0.0)	7 (41.2)	6 (16.2)	3 (8.3)	5 (22.7)	.030
Cancer, n (%)	1 (10.0)	4 (23.5)	9 (24.3)	6 (16.7)	3 (13.6)	.795
Atrial fibrillation, n (%)	0 (0.0)	4 (23.5)	4 (10.8)	8 (22.2)	0 (0.0)	.042
Laboratory test						
Hemoglobin (g/L)	121.46 ± 24.82	119.65 ± 31.75	126.16 ± 20.69	125.03 ± 14.75	130.45 ± 13.09	.534
Albumin (g/L)	30.00 (26.00-34.00)	36.00 (34.00-37.00)	37.00 (33.00-42.00)	37.00 (33.50-39.00)	40.00 (35.00-42.00)	.038
ALT (U/L)	25.00 (11.00-78.00)	33.00 (14.00-72.00	21.00 (12.00-41.00)	20.50 (13.00-42.50)	22.00 (13.00-38.00)	.909
AST (U/L)	36.00 (12.00-52.00)	31.00 (25.00-62.00)	25.00 (18.00-41.00)	22.50 (18.00-62.00)	21.00 (17.00-38.00)	.640
Uric acid (mmol/L)	351.00 ± 271.34	380.53 ± 125.68	343.14 ± 127.16	331.58 ± 123.08	352.39 ± 104.74	.805
Estimated glomerular filtration rate (mL/(min × 1.73 m^2)	79.95 (34.46-96.80)	89.73 (76.17-131.61)	90.46 (73.15-105.80)	81.53 (69.16-92.71)	79.35 (64.91-108.52)	.325
Troponin I (pg/mL)	0.01 (0.01-1.16)	0.04 (0.01-0.92)	0.02 (0.01-0.13)	0.02 (0.01-0.06)	0.03 (0.01-0.26)	.510

Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; MAP, mean arterial pressure.
[a] Group 1: MAP < 70 mm Hg; group 2:70 mm Hg ≤ MAP < 80 mm Hg; group 3: 80 mm Hg ≤ MAP < 90 mm Hg; group 4: 90 mm Hg ≤ MAP < 100 mm Hg; group 5: MAP ≥ 100 mm Hg.

Table 3. Odds Ratio for In-Hospital Mortality According to MAP Group.[a,b,c]

	Model 1	Model 2	Model 3
Group 1 (OR, 95% CI)	Reference	Reference	Reference
Group 2 (OR, 95% CI)	0.898 (0.239-3.373)	0.808 (0.078-8.340)	1.680 (0.020-140.335)
Group 3 (OR, 95% CI)	0.051 (0.006-0.460)[d]	0.011 (0.0001-0.442)[d]	0.003 (0.0001-0.343)[d]
Group 4 (OR, 95% CI)	0.188 (0.041-0.862)[d]	0.029 (0.0001-1.987)	0.006 (0.0001-1.671)
Group 5 (OR, 95% CI)	0.109 (0.012-0.992)[d]	0.007 (0.0001-2.012)	0.003 (0.0001-9.744)

[a] Cox proportional hazards regression method was used for univariable and multivariable analysis.
[b] Model 1: unadjust, model 2:adjust by gender, hypertension, diabetes, atrial fibrillation, chronic heart failure, stroke, cancer, D-dimer, white blood cells, estimated glomerular filtration rate, hemoglobin, ALT, systolic BP.
[c] Model 3: adjust by model 2, age, albumin, troponin I, uric acid, platelets, albumin, HDL, LDL, troponin I, diastolic BP, uric acid, platelet.
[d] $P < .05$.

hospital that was susceptible to sampling bias. Second, this study was retrospectively conducted with reference to the electronic medical record. The aMAP values may not reflect actual hemodynamic status. Third, we only included the first 24 hours of MAP for analysis, but subsequent MAP was not considered. Fourth, MAP between patients with hypertension and without hypertension was not compared.

Conclusion

In conclusion, the prognostic role of MAP during the first 24 hours of hospital admission should be emphasized in patients with PE. The optimal range of MAP for patients with intermediate- and high-risk PE may be 80 to 90 mm Hg.

Authors' Note

JL C and J L contributed equally to this work, acquired the data, and drafted and revised the manuscript. SJ S and DS W designed the study, provided supervision, and critically revised the manuscript. All authors approved the final version of the manuscript and agreed to be accountable for all aspects of the study. The data sets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Ethics Approval and Consent to Participate

The study protocol was approved by the ethics committee of Fujian Provincial Hospital (Fujian, China) in accordance with the 1964 Declaration of Helsinki. Consent was not required due to the retrospective nature of the study (K2019-12-017).

Funding

The author(s) disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: High-level hospital grants from the Fujian Provincial Hospital, Fujian province, China (grant/award number 2017-510#) and Fujian Provincial Hospital (grant/award number: 2017-510#).

ORCID iD

Songjing Shi https://orcid.org/0000-0001-5465-2969

References

1. Jimenez D, Kopecna D, Tapson V, et al. Derivation and validation of multimarker prognostication for normotensive patients with acute symptomatic pulmonary embolism. *Am J Resp Crit Care Med*. 2014;189(6):718-726.
2. Raskob GE, Angchaisuksiri P, Blanco AN, et al. Thrombosis: a major contributor to global disease burden. *Arterioscler Thromb Vasc Biol*. 2014;34(11):2363-2371.
3. Konstantinides SV, Meyer G, Becattini C, et al. 2019 ESC Guidelines for the diagnosis and management of acute pulmonary embolism developed in collaboration with the European Respiratory Society (ERS): the task force for the diagnosis and management of acute pulmonary embolism of the European Society of Cardiology (ESC). *Eur Respir J*. 2019;54(3):1901647.
4. Keller K, Beule J, Balzer JO, Dippold W. Blood pressure for outcome prediction and risk stratification in acute pulmonary embolism. *Am J Emerg Med*. 2015;33(11):1617-1621.
5. Bach AG, Taute BM, Baasai N, et al. 30-Day mortality in acute pulmonary embolism: prognostic value of clinical scores and anamnestic features. *PLoS One*. 2016;11(2):e0148728.
6. Jimenez D, Aujesky D, Moores L, et al. Simplification of the Pulmonary Embolism Severity Index for prognostication in patients with acute symptomatic pulmonary embolism. *Arch Intern Med*. 2010;170(15):1383-1389.
7. Sesso HD, Stampfer MJ, Rosner B, et al. Systolic and diastolic blood pressure, pulse pressure, and mean arterial pressure as predictors of cardiovascular disease risk in men. *Hypertension*. 2000;36(5):801-807.
8. Sakr Y, Dubois MJ, De Backer D, et al. Persistent microcirculatory alterations are associated with organ failure and death in patients with septic shock. *Crit Care Med*. 2004;32(9):1825-1831.
9. Konstantinides SV.2014 ESC Guidelines on the diagnosis and management of acute pulmonary embolism. *Eur Heart J*. 2014;35(45):3145-3146.
10. Torbicki A. Pulmonary thromboembolic disease. Clinical management of acute and chronic disease. *Rev Esp Cardiol*. 2010;63(7):832-849.
11. Lehnert P, Lange T, Moller CH, Olsen PS, Carlsen J. Acute pulmonary embolism in a National Danish Cohort: increasing incidence and decreasing mortality. *Thromb Haemost*. 2018;118(3):539-546.
12. Keller K, Hobohm L, Ebner M, et al. Trends in thrombolytic treatment and outcomes of acute pulmonary embolism in Germany. *Eur Heart J*. 2019;41(4):522-529.
13. Waits GS, O'neal WT, Sandesara PB, et al. Association between low diastolic blood pressure and subclinical myocardial injury. *Clin Res Cardiol*. 2018;107(4):312-318.
14. Cruickshank JM. Coronary flow reserve and the J curve relation between diastolic blood pressure and myocardial infarction. *BMJ*. 1988;297(6658):1227-1230.
15. Vincent JL, Nielsen ND, Shapiro NI, et al. Mean arterial pressure and mortality in patients with distributive shock: a retrospective analysis of the MIMIC-III database. *Ann Intensive Care*. 2018;8(1):107.
16. Gao Y, Wang Q, Li J, et al. Impact of mean arterial pressure fluctuation on mortality in critically Ill patients. *Crit Care Med*. 2018;46(12):e1167-e1174.
17. Mathis MR, Naik BI, Freundlich RE, et al. Preoperative risk and the association between hypotension and postoperative acute kidney injury. *Anesthesiology*. 2019;132(3):461-475.
18. Rhodes A, Evans LE, Alhazzani W, et al. Surviving sepsis campaign: international guidelines for management of sepsis and septic shock: 2016. *Intensive Care Med*. 2017;43(3):304-377.
19. Lamontagne F, Meade MO, Hebert PC, et al. Higher versus lower blood pressure targets for vasopressor therapy in shock: a multicentre pilot randomized controlled trial. *Intensive Care Med*. 2016;42(4):542-550.
20. Lamontagne F, Day AG, Meade MO, et al. Pooled analysis of higher versus lower blood pressure targets for vasopressor therapy septic and vasodilatory shock. *Intensive Care Med*. 2018;44(1):12-21.
21. Asfar P, Meziani F, Hamel JF, et al. High versus low blood-pressure target in patients with septic shock. *N Engl J Med*. 2014;370(17):1583-1593.
22. Manier G, Castaing Y. Influence of cardiac output on oxygen exchange in acute pulmonary embolism. *Am Rev Respir Dis*. 1992;145(1):130-136.
23. Correa TD, Vuda M, Takala J, et al. Increasing mean arterial blood pressure in sepsis: effects on fluid balance, vasopressor load and renal function. *Crit Care*. 2013;17(1):R21.
24. Jacobs FM. Relation between mean arterial pressure and renal function in the early phase of shock: a prospective, explorative cohort study. *Crit Care*. 2011;15(5):442.