ORIGINAL ARTICLE

Short-term mortality of patients with saddle pulmonary embolism: A single-center study

Eyer tipi pulmoner arter embolisi olan hastalarda kısa dönem mortalite: Tek merkezli çalışma

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ABSTRACT

Objective: Although hemodynamic instability has been identified as the most established mortality predictor in acute pulmonary embolism (PE), the debate is still open about the prognostic significance of saddle pulmonary embolism (SPE). This study determined the in-hospital mortality rate of SPE patients diagnosed via computed tomographic pulmonary angiography (CTPA) and compared these cases with non-SPE patients.

Methods: The presence of SPE observed on CTPA was used to classify 492 consecutive patients into SPE and non-SPE groups. Different features were compared between the 2 groups, and independent predictors of in-hospital mortality in acute PE were identified.

Results: A total of 70 patients (14.2%) had SPE. In univariate analysis, the SPE group was seen to have a higher in-hospital mortality rate, as well as a lower oxygen saturation level and systolic and diastolic blood pressure in comparison with the non-SPE group (all p values <0.005). Multivariate analysis revealed that SPE was an independent predictor of in-hospital mortality in acute PE patients (Odds ratio: 9.21, 95% confidence interval: 3.40-24.89; p value <0.001).

Conclusion: The results of this study indicated that SPE had a statistically significant importance in predicting in-hospital mortality and adverse events in PE patients. These findings were not consistent with many prior studies.

ÖZET

Amaç: Akut pulmoner embolide (PE) hemodinamik instabilite akut pulmoner embolinin en çok kanıtlanmış öngördürücü faktörü olmasına rağmen sağ ve sol pulmoner arter embolisinin (EPE) prognostik önemi yine de tartışmaya açıktır. Bu çalışmada bilgisayarlı tomografik pulmoner anjiyografiyle (BTPA) tanı konmuş EPE'nin hastane içi mortalitesini belirlemeyi ve bu oranları diğer tip pulmoner emboli olgularıyla karşılaştırmayı amaçladık.

Yöntemler: BTPA'da EPE varlığına göre 492 ardışık hastayı EPE ve ve EPE-dışı PE'si olan olgular olarak sınıfladık. İki grup arasında farklı özellikleri karşılaştırdıktan sonra hastane içi akut PE mortalite oranının bağımsız öngördürücü faktörlerini tanımladık.

Bulgular: Toplam 70 (%14.2) hastada EPE vardı. Tek değişkenli analizde EPE-dışı PE grubuna göre EPE grubunda hastane içi mortalite oranı daha yüksek, O_2 doygunluk oranıyla sistolik (SKB) ve diyastolik kan basınçları (DKB) daha düşüktü (tüm p değerleri <0.005). Çok değişkenli analiz akut PE hastalarında EPE'nin hastane içi mortalitesinin bağımsız bir öngördürücü faktörü olduğunu ortaya çıkartmıştır (Odds oranı =9.21, %95 GA=3.40–24.89, p değeri <0.001).

Sonuç: Çalışmamız, önceki birçok çalışma ile tutarsız olarak EPE'nin PE hastalarında hastane içi mortalite ve advers olayları öngörmede istatistiksel açıdan önemli olduğunu göstermiştir.

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Acute pulmonary embolism (PE), a potentially life-threatening condition, is one of the leading causes of cardiovascular-related mortality. It is as deadly as myocardial infarction, resulting in 100,000 to 200,000 deaths each year.[1,2] The mortality rate associated with PE ranges from less than 1% to 50% in different studies, depending on the clinical presentation.[3,4]

Saddle pulmonary embolism (SPE), an uncommon type of PE, is characterized as a visible thromboembolism straddling the bifurcation of the main pulmonary artery trunk.[5–7] The prevalence of SPE varies depending on the diagnostic method used to confirm it. SPE has been reported in 3% to 5% of all PE patients based on computed tomographic pulmonary angiography (CTPA) results[8] and in 15% when using transesophageal echocardiography (TEE).[7,9,10] The actual frequency of SPE may be underestimated utilizing TEE or CTPA, as a distinct group of SPE patients is too unstable to undergo CTPA or TEE. In these cases, the diagnosis is often confirmed via autopsy.[11,12]

The best known predictors of mortality and morbidity in PE are hemodynamic instability and refractory hypotension, and these indicators have widely been used for management purposes.[13,14] Specifically, in massive PE, defined as a systolic blood pressure (SBP) of less than 90 mm Hg, the 90-day mortality rate is estimated as high as 52.4%, despite a number of different treatment modalities.[15] Right ventricular (RV) dysfunction has been shown to be an important predictor of adverse events in normotensive patients.[10,16]

It is unclear whether the presence of SPE worsens hemodynamic stability. Identification of SPE on CTPA has modified management approaches, as demonstrated in studies describing diverse SPE treatments. Aggressive therapies have been recommended for patients with SPE in a previous case series.[12] However, other studies have indicated that the use of aggressive treatments in cases of PE with a large clot burden may place these patients at an increased risk for complications and prolonged hospitalization.[8]

Variations in treatment depending on the presence of SPE necessitate assessment of the prognostic aspects of SPE. There is an ongoing debate on the predictive value of SPE in the literature. Similar short-term outcomes have been demonstrated in both SPE and non-SPE groups in most studies. However, some studies have suggested a significantly higher rate of mortality and 30-day major adverse events in the SPE group.[5,11]

Abbreviations:

CI	*Confidence interval*
CTPA	*Computed tomography pulmonary angiography*
MPAP	*Mean pulmonary artery pressure*
OR	*Odds ratio*
PE	*Pulmonary embolism*
RV	*Right ventricle*
SBP	*Systolic blood pressure*
SPE	*Saddle pulmonary embolism*
sPESI	*Simplified Pulmonary Embolism Severity Index*
TEE	*Transesophageal echocardiography*

The aim of this study was to analyze the frequency, presentation, and clinical outcomes of SPE patients diagnosed via CTPA with a special focus on the in-hospital mortality rate, and to compare them with non-SPE cases.

METHODS

Study population

Upon local institutional review board approval of the design and conduct of this retrospective cohort single-center study, a list of all of the inpatients aged 18 years or older at the Cardiovascular Diseases Research Center, Shahid Madani Heart Hospital, a university-affiliated referral center in Tabriz, Iran, from July 2010 through December 2015 was obtained. The patients were selected in a computer-assisted search based on diagnosis with acute PE using appropriate International Statistical Classification of Diseases and Related Health Problems classification codes.

Study design

Patients with diagnoses of chronic PE or non-thrombotic PE, and those who did not consent to offer their personal medical records or CTPA images were excluded. The final cohort enrolled in the study consisted of 492 consecutive patients with acute PE, as diagnosed by CTPA (Siemens 32-slice computed tomography scanners). PE was defined as the presence of at least one intraluminal filling defect at the level of visualized pulmonary arteries. Close inspection of all of the CTPA images of the patients diagnosed with PE revealed that 70 (14.2%) could be identified as cases of SPE. SPE was defined as a thromboembolism extending across the bifurcation of the main pulmonary artery trunk into the right and left main pulmonary arteries. The CTPA images were reviewed by 2 expert, board-certified radiologists who were unaware of the clinical course of the patients.

In-hospital mortality was defined as death occurring due to PE during the hospitalization period, beginning from the time of admission in the emergency room, before or after treatment. Any mortality due to bleeding or non-acute PE causes was excluded. An adverse event was defined as any death, as well as any need for mechanical ventilation or a vasopressor during hospitalization.

Fibrinolytic therapy was used in those with a SBP of less than 90 mm Hg or when O_2 saturation was less than 90%, concomitant with severe RV enlargement occurring in massive, acute PE apparent on CTPA, especially in patients with a poor response to anticoagulant therapy.

The simplified Pulmonary Embolism Severity Index (sPESI) value was calculated for all the patients. In this scoring system, one point is assigned to the presence of each of these variables: age above 80 years, a history of cancer, heart rate more than 110 beats/minute, chronic cardiopulmonary disease, SBP less than 100 mm Hg, and oxyhemoglobin saturation below 90%. A score of zero is considered a low-risk patient. Even the presence of one variable put the patient in the high-risk group.[17]

Data collection

In the next step, the following characteristics were retrieved from hospital medical records for all patients: i) patients' demographic data, such as age, gender, and past medical history; ii) initial on-admission vital signs, oxygen saturation, and presenting symptoms; iii) echocardiographic findings from the cardiology reports performed within 24 hours of admission; iv) other CTPA features; v) blood sample laboratory results; vi) use of fibrinolytic drugs as a treatment option; and vii) in-hospital mortality and admission duration.

Statistical analysis

The patients with PE were classified into 2 groups: SPE and non-SPE. In the primary analysis, the SPE group was compared with the non-SPE group utilizing a t-test for continuous variables and a chi-square test for categorical variables. Univariate and multivariate logistic regression analyses were used to identify the independent predictors of in-hospital mortality. The final results of multivariate logistic regression analysis were reported as odds ratios (OR), and 95% confidence intervals (CI), with p values ≤0.05 considered statistically significant. All statistical analyses were performed using IBM SPSS Statistics for Windows, Version 21.0 (IBM Corp., Armonk, NY, USA).

RESULTS

A total of 492 patients with acute PE diagnosed via CTPA were enrolled in this retrospective cohort study.

Baseline characteristics

The mean age of the patients was 61.9±17.2 years. Among 492 patients, 70 (14.2%) had SPE. Table 1 presents the demographic data, clinical features, echocardiographic findings, and in-hospital mortality of patients in the SPE and non-SPE groups.

Among concurrent comorbidities and associated risk factors, only a prior history of recent immobility and congestive heart failure had a statistically significant association with SPE (p value=0.01 and 0.02, respectively). Patients with SPE had a lower systolic (p value=0.002) and diastolic (p value <0.001) blood pressure (91.4±22.3 mm Hg and 61.3±14.1 mm Hg, respectively). A lower O_2 saturation rate was also recorded (p value=0.005).

Patients' outcome

Univariate analysis revealed that the in-hospital mortality was significantly higher in the SPE group (p value <0.001). Mechanical ventilation was necessary in 20 patients (28.5%) with SPE and in 63 (14.9%) non-SPE patients (p value=0.032). A surgical embolectomy was performed in 24 (34.2%) patients with SPE and 81 (19.1%) non-SPE patients (p value=0.020). Also, the incidence of major adverse events, including in-hospital death, the need for fibrinolytic therapy, mechanical ventilation, and pulmonary embolectomy, was significantly greater in the SPE group (p value <0.001). Furthermore, white blood cell and neutrophil counts, in addition to cardiac troponin (cTnl) levels were significantly higher in SPE group (p value <0.05).

Table 2 displays the laboratory findings of the patients according to in-hospital mortality.

Multivariate logistic regression analysis was used evaluate the effect on in-hospital mortality rate of several variables, including SBP <90 mm Hg, heart rate >100 beats/minute, gender (male), RV dysfunction, sPESI, RV enlargement, O_2 saturation <90%,

Table 1. Demographic data, clinical features, echocardiographic findings, and in-hospital mortality of patients in SPE and non-SPE groups

	Total (n=492)			SPE (n=70)			Non-SPE (n=422)			*p*
	n	%	Mean±SD	n	%	Mean±SD	n	%	Mean±SD	
Gender										
Male	258	52.4		35	50		233	52.8		0.659
Female	234	47.6		35	50		199	47.2		
Age (years)			61.9±17.2			64.1±15.3			61.0±17.2	0.159
Diabetes mellitus	76	15.4		9	12.9		67	15.9		0.517
CHF	40	8.1		1	1.4		39	9.2		0.027
COPD	48	9.8		3	4.3		45	10.7		0.096
Immobility	83	16.9		19	27.1		64	15.2		0.013
RV enlargement	331	67.3		58	82.9		273	64.7		0.003
RV dysfunction,	311	63.2		57	81.4		254	60.2		0.001
TR gradient (mm Hg)			37.8±19.7			37.9±17.9			37.8±20.5	0.360
Fibrinolytic therapy	102	20.7		29	41.4		73	17.3		<0.001
Admission duration (days)			9.7±6.5			7.3±5.2			9.8±5.0	<0.001
In-hospital mortality	46	9.3		20	28.6		26	6.2		<0.001
sPESI			1.2±1.1			1.4±1.1			1.0±1.6	0.044

SPE: Saddle pulmonary embolism; SD: Standard deviation; CHF: Congestive heart failure; COPD: Chronic obstructive pulmonary disease; RV: Right ventricle; TR: Tricuspid regurgitation; sPESI: Simplified Pulmonary Embolism Severity Index.

Table 2. Laboratory findings of patients and in-hospital mortality

Value	Hospital death (yes) 46	Hospital death (no) 446	*p*
Neutrophil (%)	79.7±13.4	74.4±11.0	0.041
Lymphocyte (%)	16.8±3.1*	18.8±0.7*	0.550
White blood cell /L	12082.9±386*	12127.7±265.5*	0.300
O_2 saturation (%)	78.9±12.2	85.7±10.4	<0.001
Blood sugar	137.8±84.8	142.4±71.0	0.763
Creatinine	1.54±1.07	1.21±0.95	0.062
Total cholesterol	157.8±43.7	170.5±48.7	0.239
Hemoglobin (g/dL)	12.6±2.5	12.8±2.2	0.927
Creatine kinase	104.8±19.1*	162±25.4	0.402
Right ventricular dysfunction&dysfunction, n (%)	9 (19.5)	26 (5.8)	0.001
Saddle emboli, n (%)	20 (43.4)	26 (5.8)	0.001
Simplified Pulmonary Embolism Severity Index	1.54±1.01	1.14±1.08	0.016

*Mean±standard error.

and SPE. The analysis revealed that sPESI and SPE were independent predictors of in-hospital mortality. Notably, the presence of SPE was associated with an average 9.2-fold greater incidence of in-hospital mortality (95% CI=3.40-24.89; p value <0.001) (Table 3).

DISCUSSION

The noninvasive nature of CTPA as well as a clinical equivalence with invasive methods, and greater availability have made it the first diagnostic imaging test

Table 3. Factors affecting in-hospital mortality according to logistic regression analysis

Variables	OR	95% CI for OR		p
		Lower	Upper	
Saddle emboli	9.212	3.409	24.892	<0.001
Gender (male)	0.339	0.124	0.932	0.036
sPESI	0.494	0.294	0.831	0.008
O_2 saturation <90%	1.043	0.998	1.090	0.061

CI: Confidence interval; OR: Odds ratio; sPESI: Simplified Pulmonary Embolism Severity Index.

for most of patients with a high suspicion of PE.[18] Yet, despite its proven ability to identify patients with PE, the strength of CTPA in predicting the mortality of these patients still remains an area of debate.[19] CTPA results have been both a dual diagnostic/predictive tool in some instances,[20] and has guided some physicians in making clinical decisions.[18] We particularly tried to discover whether the presence of SPE observed on CTPA could predict in-hospital mortality.

Our study revealed an SPE frequency of 14.2%, which is far higher than that seen in similar research, making this study one of the largest available investigations to compare the mortality rates of SPE and non-SPE groups.[11,21,22] Our institution is the only referral center for massive PE in the province, which is a likely reason for the higher rate of SPE in our sample. A mortality rate of 28.6% in SPE patients in our investigation was close to the 18.5% mortality rate reported by Kwak et al.[11] and the 20.4% observed by Liu et al.[28] Nevertheless, it is still far higher than the values seen in other similar available studies with smaller sample sizes and low-risk patients.[21]

The results of this study suggested that finding SPE on CTPA was an independent predictor of in-hospital mortality. This was also indicated by the fact that the SPE group had a significantly shorter admission duration (7.3±5.2 days) than the non-SPE counterparts (9.8±5.0 days) (p value <0.05), which can be explained by higher early mortality rate in these patients. This finding is, however, different from that of previous reports. In a study performed by Pruszczyk et al.,[21] no significant difference in the mortality rate between the SPE and non-SPE groups was observed. Ryu et al.[8] demonstrated that the short-term mortality rate for patients with SPE was not higher than that of other patients with PE. In a nested, case-control investigation, Gandara et al.[23] found that SPE did not seem to be associated with a poorer 30-day outcome compared with matched patients with proximal PE. Sardi et al.[22] observed that the presence of SPE did not indicate a more unfavorable clinical outcome and may not require different management than that used for standard PE. On the other hand, a study performed by Yusuf et al.[24] revealed a higher mortality rate for SPE patients after 1 year despite no significant difference in short-term mortality. All of the aforementioned studies had fewer SPE cases and a lower mortality rate, which may have contributed to their results. The research conducted by Kwak et al.,[11] which had a comparatively larger number SPE cases, suggested that while the short-term mortality alone was not significantly higher in SPE patients, they had more major adverse events in total (which included short-term mortality), which is similar to our results. The discrepancy between our results and the findings of previous investigations can partially be explained by the small sample size of those studies and the inclusion of only normotensive patients in some of the investigations.

The inconsistencies between our study and available literature can also be interpreted in light of multiple clinical and imaging aspects. The presence of shock in PE patients increases mortality, with most deaths occurring during the first hour of admission.[25] The time interval between the initiation of symptoms and diagnosis plus management of patients may vary in different countries according to the available equipment and protocols. The incidence of SPE observed in necropsies confirms its life-threatening power.[21] Concurrent right or left PE in segmental arteries and the proximity of the PE can also make a big difference. A study by Choi et al.[26] indicated that central PE (including both SPE and left/right pulmonary artery embolus groups), rather than SPE, was an independent predictor of an adverse outcome. Ghanima et al.[27] found a significant association between the pulmonary artery obstruction index and the proximal extension of the clot (p value <0.001). Interestingly, PaO_2 and cTnI levels were significantly different in patients with a clot in the main pulmonary artery. The authors suggested that the most proximal locations of PE are valuable as a prognostic factor.

Although the cTnI level was significantly higher (p value <001) and the O_2 saturation was significantly

lower (p=0.005) in the SPE group, our study did not show O_2 saturation or cTnI to be independent predictors of short-term mortality. Liu et al.[28] found that the cTnI level was higher in the non-survivor SPE group. As with previous investigations,[29] our logistic regression analysis also showed that O_2 saturation of <90% on admission could increase the mortality rate in SPE group, though it was not statistically significant (OR: 1.04, Cl 95%: 0.99–1.09; p value=0.061). As in previous studies,[2] logistic regression analysis indicated that the sPESI value was an independent predictor of mortality in patients with SPE.

Significantly higher rates of fibrinolytic therapy in the SPE group in comparison with the non-SPE group (p value <0.05) have also been reported in prior studies.[11] This finding is somewhat compatible with the significantly lower blood pressures seen in the SPE group. Based on the currently available guidelines, it appears that low blood pressure plays a far greater role in initiating fibrinolytic therapy than the simple presence of SPE.

Although there were significantly more echocardiographic findings of RV enlargement in the SPE group (p value <0.05), it was not an independent factor for predicting in-hospital mortality. It seems that RV dysfunction by itself is not an immediate factor of mortality.[30] Indeed, it may cause more harm when PE remains untreated or is combined with low blood pressure. One challenging issue in patients with massive PE is to know whether the degree of pulmonary vascular obstruction or RV function plays a more important role in short-term mortality. It seems that in patients without previous pulmonary vascular disease, the degree of vascular obstruction has a higher correlation with short-term mortality, where sudden obstruction leads to sudden death. On the other hand, in patients with previous pulmonary vascular disease and RV hypertrophy, the RV is trained to tolerate a sudden rise in pulmonary vascular resistance. It has been shown that even the mean pulmonary artery pressure (mPAP) and the severity of pulmonary vascular obstruction do not have any correlation in these patients.[31–33]

Significantly higher white blood cell and neutrophil counts in the SPE group suggested the possible role of inflammation in this group of patients. Recently, an association between the neutrophil-to-lymphocyte ratio and PE mortality has been demonstrated in several investigations.[34,35]

Overall, the use of CTPA for PE diagnosis and the prediction of mortality in these patients still needs further investigation. SPE should prompt concern as for a large clot, since small clots will pass from left and right pulmonary arteries (big arteries) to distal branches with the force of RV blood flow. The presence of SPE in the main pulmonary artery indicates the inability of the RV to push the clot to distal parts. Hence, SPE can be a sign of a large clot and susceptibility to hemodynamic instability.

Treatment and follow-up suggestions

We suggest that patients with SPE and RV enlargement observed on echocardiography without a previous history of pulmonary vascular disease should be admitted to intensive care units. Also, early invasive therapy with fibrinolytic drugs should be started as soon as possible. Since an mPAP >50 mm Hg has been shown to have a correlation with 2-year mortality,[35] when an mPAP >50 mm Hg concomitant with RV enlargement remains after fibrinolytic therapy, surgical embolectomy may be useful to improve patient survival.

Conclusion

Our study results demonstrated that the presence of SPE had a statistically significant importance in predicting the in-hospital mortality and in-hospital adverse events of PE patients, a finding that was not consistent with many prior reports.

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REFERENCES

1. Park B, Messina L, Dargon P, Huang W, Ciocca R, Anderson FA, et al. Recent trends in clinical outcomes and resource utilization for pulmonary embolismin the United

States: findings from the nationwide inpatient sample. Chest 2009;136:983–90. [CrossRef]

2. Ostovan MA, Ghaffari S, Pourafkari L, Dehghani P, Hajizadeh R, Nadiri M, et al. Modification of Simplified Pulmonary Embolism Severity Index and its PrognosticValue in Patients with Acute Pulmonary Embolism. Heart Lung Circ 2016;25:184–90. [CrossRef]
3. Arcasoy SM, Kreit JW. Thrombolytic therapy of pulmonary embolism: a comprehensive review of currentevidence. Chest 1999;115:1695–707. [CrossRef]
4. de Bonis S, Rendina D, Vargas G, Di Minno D, Piedimonte V, Gallotta G, et al. Predictors of in-hospital and long-term clinical outcome in elderly patients with massive pulmonary embolism receiving thrombolytic therapy. J Am Geriatr Soc 2008;56:2273–7. [CrossRef]
5. Torbicki A, Pacho R, Jedrusik P, Pruszczyk P. Noninvasive diagnosis and treatment of a saddle pulmonary embolism. A case report in support of new trends in management of pulmonary embolism. Chest 1996;109:1124–6. [CrossRef]
6. Mabee SW, Mabee CL, Pacht ER. Normal arterial blood gas in a patient with saddle pulmonary artery embolus: diagnosis by transesophageal echocardiography. J Natl Med Assoc 1995;87:717–9.
7. Pruszczyk P, Torbicki A, Pacho R, Chlebus M, Kuch-Wocial A, Pruszynski B, et al. Noninvasive diagnosis of suspected severe pulmonary embolism: transesophageal echocardiography vs spiral CT. Chest 1997;112:722–8. [CrossRef]
8. Ryu JH, Pellikka PA, Froehling DA, Peters SG, Aughenbaugh GL. Saddle pulmonary embolism diagnosed by CT angiography: frequency, clinicalfeatures and outcome. Respir Med 2007;101:1537–42. [CrossRef]
9. Pruszczyk P, Torbicki A, Kuch-Wocial A, Szulc M, Pacho R. Diagnostic value of transoesophageal echocardiography in suspectedhaemodynamically significant pulmonary embolism. Heart 2001;85:628–34. [CrossRef]
10. Kucher N, Rossi E, De Rosa M, Goldhaber SZ. Prognostic role of echocardiography among patients with acute pulmonary embolismand a systolic arterial pressure of 90 mm Hg or higher. Arch Intern Med 2005;165:1777–81.
11. Kwak MK, Kim WY, Lee CW, Seo DW, Sohn CH, Ahn S, et al. The impact of saddle embolism on the major adverse event rate of patients with non-high-risk pulmonary embolism. Br J Radiol 2013;86:20130273. [CrossRef]
12. Sweet PH 3rd, Armstrong T, Chen J, Masliah E, Witucki P. Fatal pulmonary embolism update: 10 years of autopsy experience at an academic medical center. JRSM Short Rep 2013;4:2042533313489824. [CrossRef]
13. Jaff MR, McMurtry MS, Archer SL, Cushman M, Goldenberg N, Goldhaber SZ, et al. Management of massive and submassive pulmonary embolism, iliofemoral deep vein thrombosis, and chronic thromboembolic pulmonary hypertension: a scientificstatement from the American Heart Association. Circulation 2011;123:1788–830. [CrossRef]
14. Goldhaber SZ, Visani L, De Rosa M. Acute pulmonary embolism: clinical outcomes in the International CooperativePulmonary Embolism Registry (ICOPER). Lancet 1999;353:1386–9. [CrossRef]
15. Kucher N, Rossi E, De Rosa M, Goldhaber SZ. Massive pulmonary embolism. Circulation 2006;113:577–82. [CrossRef]
16. Sanchez O, Trinquart L, Colombet I, Durieux P, Huisman MV, Chatellier G, et al. Prognostic value of right ventricular dysfunction in patients with haemodynamicallystable pulmonary embolism: a systematic review. Eur Heart J 2008;29:1569–77.
17. Jiménez D, Aujesky D, Moores L, Gómez V, Lobo JL, Uresandi F, et al. Simplification of the pulmonary embolism severity index for prognostication in patients with acute symptomatic pulmonary embolism. Arch Intern Med 2010;170:1383–9.
18. Doğan H, de Roos A, Geleijins J, Huisman MV, Kroft LJ. The role of computed tomography in the diagnosis of acute and chronic pulmonary embolism. Diagn Interv Radiol 2015;21:307–16. [CrossRef]
19. Masotti L, Righini M, Vuilleumier N, Antonelli F, Landini G, Cappelli R, et al. Prognostic stratification of acute pulmonary embolism: focus on clinical aspects, imaging, and biomarkers. Vasc Health Risk Manag 2009;5:567–75. [CrossRef]
20. Qanadli SD, El Hajjam M, Vieillard-Baron A, Joseph T, Mesurolle B, Oliva VL, New CT index to quantify arterial obstruction in pulmonary embolism: comparisonwith angiographic index and echocardiography. AJR Am J Roentgenol 2001;176:1415–20. [CrossRef]
21. Pruszczyk P, Pacho R, Ciurzynski M, Kurzyna M, Burakowska B, Tomkowski W, et al. Short term clinical outcome of acute saddle pulmonary embolism. Heart 2003;89:335–6.
22. Sardi A, Gluskin J, Guttentag A, Kotler MN, Braitman LE, Lippmann M. Saddle pulmonary embolism: is it as bad as it looks? A community hospitalexperience. Crit Care Med 2011;39:2413–8. [CrossRef]
23. Gandara E, Bose G, Erkens P, Rodgers M, Carrier M, Wells P. Outcomes of saddle pulmonary embolism: a nested case-control study. J Thromb Haemost 2011;9:867–9. [CrossRef]
24. Yusuf SW, Gladish G, Lenihan DJ, Lei X, Durand JB, Swafford J, et al. Computerized tomographic finding of saddle pulmonary embolism is associated with high mortality in cancer patients. Intern Med J 2010;40:293–9. [CrossRef]
25. Wood KE. Major pulmonary embolism: review of a pathophysiologic approach to the goldenhour of hemodynamically significant pulmonary embolism. Chest 2002;121:877–905.
26. Choi KJ, Cha SI, Shin KM, Lim JK, Yoo SS, Lee J, et al. Central emboli rather than saddle emboli predict adverse outcomes in patients with acute pulmonary embolism. Thromb Res 2014;134:991–6. [CrossRef]
27. Ghanima W, Abdelnoor M, Holmen LO, Nielssen BE, Sandset PM. The association between the proximal extension of the clot and the severity of pulmonary embolism (PE): a proposal for a new radiological score for PE. J Intern Med 2007;261:74–81. [CrossRef]

28. Liu M, Miao R, Guo X, Zhu L, Zhang H, Hou Q, et al. Saddle Pulmonary Embolism: Laboratory and Computed Tomographic PulmonaryAngiographic Findings to Predict Short-term Mortality. Heart Lung Circ 2017;26:134–42. [CrossRef]
29. 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;33:1617–21. [CrossRef]
30. Stein PD, Beemath A, Matta F, Goodman LR, Weg JG, Hales CA, et al. Enlarged right ventricle without shock in acute pulmonary embolism: prognosis. Am J Med 2008;121:34–42.
31. Gerges C, Skoro-Sajer N, Lang IM. Right ventricle in acute and chronic pulmonary embolism (2013 Grover Conference-series). Pulm Circ 2014;4:378–86. [CrossRef]
32. Delcroix M, Mélot C, Vachiéry JL, Lejeune P, Leeman M, Vanderhoeft P, et al. Effects of embolus size on hemodynamics and gas exchange in canine embolicpulmonary hypertension. J Appl Physiol (1985) 1990;69:2254–61. [CrossRef]
33. Sharma GV, McIntyre KM, Sharma S, Sasahara AA. Clinical and hemodynamic correlates in pulmonary embolism. Clin Chest Med 1984;5:421–37.
34. Kayrak M, Erdoğan HI, Solak Y, Akilli H, Gül EE, Yildirim O, et al. Prognostic value of neutrophil to lymphocyte ratio in patients with acute pulmonary embolism: a restrospective study. Heart Lung Circ 2014;23:56–62. [CrossRef]
35. Ribeiro A, Lindmarker P, Johnsson H, Juhlin-Dannfelt A, Jorfeldt L. Pulmonary embolism: one-year follow-up with echocardiography doppler and five-year survival analysis. Circulation 1999;99:1325–30. [CrossRef]

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