İstanbul Med J 2025; 26(2): 129-34
**DOI:** 10.4274/imj.galenos.2025.14603

# Predictive Value of Onodera's Prognostic Nutritional Index for Short-Term Mortality in Subjects with Acute Pulmonary Embolism

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## ABSTRACT

**Introduction:** The Pulmonary Embolism Severity Index (PESI) is a widely used tool for assessing prognosis and predicting 30-day mortality in acute pulmonary embolism (APE) patients. Onodera's Prognostic Nutrition Index (OPNI) is a simple tool that provides information on the nutritional status and prognosis of subjects, especially in gastrointestinal system malignancies. This academic work aims to evaluate the relationship between the OPNI score, which is a simple risk stratification tool, and the PESI score, short-term mortality (STM) in subjects diagnosed with pulmonary embolism (PE).

**Methods:** A total of 176 PE subjects were included in this retrospective academic work. The PESI scores and OPNI scores of all subjects were calculated using the formula serum albumin (g/L) + 0.005 × total lymphocyte count (cells/mm$^3$). The primary outcome of the academic work was accepted as the in-hospital mortality along with STM rate within 30 days from the time of diagnosis.

**Results:** The mean age of all subjects, PESI score, and OPNI score were 62.93±16.57, 98.83±39.35, and 42.86±8.06, respectively. There was a considerable inverse relationship between PESI score and OPNI (r=-0.401, p<0.001). In multivariate analysis, OPNI value [odds ratio (OR): 0.89, 95% confidence interval (CI): 0.83-0.96; p=0.002), cancer history (OR: 3.62, 95% CI: 1.30-10.10; p=0.014), oxygen saturation (OR: 0.86, 95% CI: 0.80-0.93; p<0.001) and male sex (OR: 2.73, 95% CI: 1.02-7.34; p=0.047) were found to be independent predictors of STM. The optimal OPNI cut-off value for predicting STM, based receiver operating characteristic curve analysis was ≤39.17, yielding a sensitivity of 75.5% and specificity of 67.6%.

**Conclusion:** OPNI appears to be a promising, easily applicable, operator-independent and cost-effective parameter for predicting STM in subjects with APE.

**Keywords:** Pulmonary embolism, Pulmonary Embolism Severity Index, Onodera's Prognostic Nutrition Index, short-term mortality

## Introduction

Pulmonary embolism (PE) is the third most common cause of mortality among cardiovascular diseases; symptoms range from dyspnea to life-threatening hemodynamic instability due to occlusion of one or more pulmonary arteries. Early diagnosis of high-risk subjects has been known to reduce the risk of death and strongly effects the selection of treatment modalitysuch as anticoagulation therapy, thrombolysis or surgical intervention (1). The risk of short-term mortality (STM) of PE by utilizing clinical parameters and/or biomarkers such as troponin and B-type natriuretic peptide, individually or in combination at the time of admission has been investigated by several scoring systems. The most widely used Pulmonary Embolism Severity Index (PESI) assesses the prognosis of PE subjects and provides a well-documented risk stratification for 30-day mortality (2).

Onodera's Prognostic Nutrition Index (OPNI) has been defined as a simple tool that provides information about the patient's nutritional status and prognosis in subjects with chronic diseases, particularly in gastrointestinal system malignancies (3). Albumin, a negative acute phase reactant, and the lymphocyte count indicating immune function are included in the formula, and OPNI can be simply calculated as: serum albumin (g/L) + 0.005 × total lymphocyte count (cells/mm$^3$). A lower OPNI score has generally been associated with worse nutritional status, chronic inflammation and poor prognosis (4-6).

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**Cite this article as:** Karaca M, Taşlıçukur Ş, Yüksel G, Tanyolaç S, Özkan F, Karabağ T, et al. Predictive value of Onodera's Prognostic Nutritional Index for short-term mortality in subjects with acute pulmonary embolism. İstanbul Med J. 2025; 26(2): 129-34

**Received:** 07.01.2025
**Accepted:** 05.04.2025
**Publication Date:** 21.05.2025

The aim of this academic work was to evaluate the association between the OPNI score, which is a simple risk stratifying tool compared to PESI, and STM in acute PE subjects.

## Methods

A total of 221 consecutive cases with acute pulmonary embolism (APE) admitted to both clinics between November 2018 and the current year were enrolled. After excluding 45 subjects with incomplete clinical or laboratory data and missing 30-day follow-up finally 176 APE subjects were included in this retrospective acedemic work. Demographic characteristics, clinical, laboratory, electrocardiographic, and echocardiographic findings were collected from the electronic databases of the hospitals. OPNI score of all subjects was calculated using the formula serum albumin (g/L) + 0.005 × total lymphocyte count (cells/mm$^3$) based on laboratory values at first admission. All subjects were treated for PE in accordance with current guidelines. Local Ethics Committee approval (approval number: 73, date: 06.09.2024) was obtained for the study in University of Health Sciences Türkiye, İstanbul Training and Research Hospital, and the academic work was conducted in accordance with the Declaration of Helsinki Principles.

Electrocardiographic records of all subjects at the time of hospitalization or pre-assessment were reviewed, and the presence of sinus rhythm or atrial fibrillation, sinus tachycardia and negative T waves in leads V1-V4 were recorded. Echocardiographic recordings were also collected using a Philips EPIQ 7 device (Philips Healthcare, USA) with a 2.5 MHz probe. In addition to left ventricular (LV) ejection fraction measured with the Modified Simpson method, LV end-diastolic diameter, right ventricular diameter, Tricuspid Annular Plane Systolic Excursion value, and the presence of mid/advanced tricuspid valve regurgitation, were noted (7). Pulmonary artery systolic pressure was determined by adding the value obtained using the Simplified Bernoulli Equation to the right atrial pressure calculated according to inferior vena cava diameter and respiratory variation (8). APE was diagnosed by an experienced radiologist using multi-slice spiral computed tomography pulmonary angiogram (CTPA) (Toshiba Medical Systems Corporation, Japan) according to the presence of complete or partial luminal filling defects in the pulmonary artery branches. Laboratory parameters were obtained from samples taken at the time of admission to the emergency department. Hematological parameters were measured using Sysmex hematology analyzers (Sysmex Corporation, Japan) as part of an automated complete blood count. Plasma D-dimer levels were measured via an immune-turbidimetric assay, while biochemical parameters, especially albumin levels, were evaluated by Beckman Coulter kits and calibrators. Troponin I levels were quantified through a chemiluminescence immunoassay method (Access 2 Immunoassay System; Beckman Coulter, Inc.).

Subjects presenting with hemodynamic instability such as shock, hypotension, or cardiac arrest were classified as high-risk irrespective of their PESI score. In contrast, subjects who were not deemed high risk were categorized as intermediate or low risk based on their PESI score and evidence of right ventricular dysfunction on imaging or laboratory findings (1,2). In-hospital mortality and STM within 30 days from the time of diagnosis were the primary outcomes of this study. Death due to any cause within the first 30 days was considered STM. All short-term clinical events were obtained from the hospital information system or national healthcare system, and subjects were divided into two groups according to the occurrence of STM.

### Statistical Analysis

While categorical variables were given as numbers and percentages, the normality of continuous variables was evaluated with the Shapiro-Wilk test, and continuous variables were given as mean ± standard deviation. When numerical variables met the normal distribution condition, the Student's t-test was used; when the normal distribution condition was not met, the Mann-Whitney U test was used; and categorical variables were compared with the chi-square test or Fisher's exact test. Correlation analyses were calculated using Pearson or Spearman correlation tests. Univariate analyses and multivariate logistic regression analysis were used to identify independent predictors for STM, and hazard ratios (HR) and 95% confidence intervals were calculated. Receiver operating characteristic (ROC) curve analysis was performed to determine the discriminatory ability of OPNI for STM based on specificity and sensitivity. Additionally, the optimal cut-off value of OPNI was calculated from the point of maximal sensitivity and specificity using Youden's index (sensitivity + specificity - 1) (9). The effect size (Cohen's d: 0.71) and power value (1-β: 0.95) of OPNI for STM were calculated using G*Power software (version 3.1.9.2). Analyses were performed using SPSS version 23.0 (IBM, Chicago, Illinois) with a $p<0.05$ indicating statistical significance.

## Results

A total of 176 subjects (n=97, 55.1% female) were evaluated. The mean age and PESI score of all subjects were 62.93±16.57 and 98.83±39.35, respectively. The proportion of subjects with PESI class IV-V was 36.4%. Demographic characteristics, admission clinical parameters, and electrocardiogram characteristics of all subjects were presented in Table 1. While male sex, congestive heart failure, history of cancer, and PESI class-V were substantially higher in the STM group, the rate of PESI class I-II was lower. There was no significant difference in the admission electrocardiographic characteristics. Laboratory and echocardiographic parameters at admission are presented in Table 2. The mean OPNI score of all subjects was 42.86±8.06. Lymphocyte count, albumin values, and OPNI score were statistically significantly lower in the STM group. No significant difference was found between the groups among echocardiographic parameters except LV ejection fraction. The correlation analysis between admission PESI score and admission characteristics, echocardiographic parameters and OPNI is given in Table 3. A statistically negative relationship was detected between PESI score and OPNI ($r=-0.401$, $p<0.001$). In multivariate analysis, the independent predictors of STM were history of cancer (HR: 2.25, $p=0.034$), oxygen saturation (HR: 0.92, $p=0.001$), and OPNI value (HR: 0.92, $p=0.005$), and these variables are shown in Table 4. The ROC analysis is given in Figure 1. The ROC curve analysis determined that OPNI ≤39.3 was the optimal cut-off value for discriminating between high-risk and low-risk subjects for STM, with a sensitivity of 70% and a specificity of 73%, respectively.

The Kaplan-Meier curves in Figure 2 represent the STM in subjects divided into low and high-risk groups, according to the determined cut-off value of OPNI.

## Discussion

The present acedemic work aimed to investigate the prognostic value of OPNI, a simple and easily calculable index based on serum albumin levels and lymphocyte count, instead of PESI which is a more complex assessment score to demonstrate 30-day mortality in subjects with APE. Our results showed that subjects with lower OPNI scores had significantly higher STM, suggesting that it can be used as a prognostic tool in the clinical management of APE.

In recent years, numerous tools have been investigated to assess the short-term risk of death in APE subjects, including various risk scores, imaging modalities, and biomarkers, the most commonly used of which is the PESI. The role of biomarkers such as troponins, brain natriuretic peptides, and D-dimer is well established in determining right ventricular dysfunction, patient prognosis, and hemodynamic impairment in APE subjects. These biomarkers reflect myocardial damage, right ventricle (RV) dysfunction and hemodynamic instability, all of which are important factors in determining patient prognosis (10). In addition to biomarkers, imaging modalities such as echocardiography and CTPA, which are important in assessing RV function and pulmonary artery pressure, provide insights about mortality. Previous studies have

**Table 1. Baseline demographic characteristics findings of all patients**

| | All patients, (n=176) | Mortality, (n=37) | Survivor, (n=139) | p |
|---|---|---|---|---|
| Age, years | 62.93±16.57 | 66.70±15.87 | 61.93±16.66 | 0.148 |
| Female gender, n (%) | 97 (55.1) | 14 (37.8) | 83 (59.7) | 0.017 |
| Hypertension, n (%) | 71 (40.3) | 11 (29.7) | 60 (43.2) | 0.139 |
| Diabetes mellitus, n (%) | 39 (22.2) | 10 (27.0) | 29 (20.9) | 0.422 |
| Congestive heart failure, n (%) | 21 (11.9) | 9 (24.3) | 12 (8.6) | 0.019 |
| Cerebrovascular disease, n (%) | 11 (6.3) | 5 (13.5) | 6 (4.3) | 0.055 |
| COPD, n (%) | 19 (10.8) | 6 (16.2) | 13 (9.4) | 0.240 |
| Acute kidney failure, n (%) | 3 (1.7) | 1 (2.7) | 2 (1.4) | 0.510 |
| Cancer, n (%) | 38 (21.6) | 16 (43.2) | 22 (15.8) | <0.001 |
| **On admission** | | | | |
| PESI score | 98.83±39.35 | 130.75±42.08 | 90.21±33.89 | <0.001 |
| **PESI class, n (%)** | | | | |
| I | 36 (20.5) | 2 (5.4) | 34 (24.5) | <0.001 |
| II | 36 (20.5) | 2 (5.4) | 34 (24.5) | |
| III | 40 (22.7) | 9 (24.3) | 31 (22.3) | |
| IV | 31 (17.6) | 7 (18.9) | 24 (17.3) | |
| V | 33 (18.8) | 17 (45.9) | 16 (11.5) | |
| Systolic blood pressure, mmHg | 127.97±27.04 | 119.09±29.60 | 130.23±25.98 | 0.002 |
| Diastolic blood pressure, mmHg | 73.47±14.02 | 72.57±17.29 | 73.69±13.12 | 0.234 |
| Heart rate, beats per minute | 97.38±18.92 | 102.89±19.98 | 95.99±18.46 | 0.083 |
| $SaO_2$, (%) | 90.69±6.01 | 87.41±7.42 | 91.57±5.26 | 0.001 |
| **Admission electrocardiogram** | | | | |
| Normal electrocardiogram, n (%) | 90 (51.1) | 16 (43.2) | 74 (53.2) | 0.748 |
| Sinus tachycardia, n (%) | 56 (31.8) | 14 (37.8) | 42 (30.2) | |
| Atrial fibrillation, n (%) | 22 (12.5) | 5 (13.5) | 17 (12.2) | |
| Negative T wave in V1-V4, n (%) | 8 (4.5) | 2 (5.4) | 6 (4.3) | |
| **Admission echocardiography** | | | | |
| Left ventricular ejection fraction, (%) | 55.42±9.79 | 51.14±11.57 | 56.72±8.84 | 0.003 |
| LV end-diastolic diameter, mm | 47.85±5.02 | 49.24±4.96 | 47.41±4.99 | 0.130 |
| Left atrial diameter, mm | 38.51±7.05 | 38.63±6.85 | 38.47±7.17 | 0.994 |
| Right ventricular diameter, mm | 28.60±6.42 | 28.83±5.98 | 28.54±6.58 | 0.764 |
| TAPSE, mm | 2.20±0.47 | 2.14±0.40 | 2.21±0.49 | 0.495 |
| sPAP, mmHg | 37.30±13.20 | 39.74±11.14 | 36.71±13.65 | 0.463 |
| Moderate/severe TR, n % | 44 (25.0) | 25 (67.6) | 12 (32.4) | 0.240 |

Continuous variables are presented as mean ± standard deviation; nominal variables are presented as frequency (%). COPD: Chronic obstructive pulmonary disease, PESI: Pulmonary embolism severity index, $SaO_2$: Arterial oxygen saturation, TAPSE: Tricuspid Annular Plane Systolic Excursion, sPAP: Systolic pulmonary artery pressure, TR: Tricuspid regurgitation

**Table 2. Laboratory and echocardiographic findings of all patients**

| | All patients, (n=176) | Mortality, (n=37) | Survivor, (n=139) | p |
|---|---|---|---|---|
| **Laboratory variables** | | | | |
| D-dimer, ng/mL | 5.53±5.54 | 5.36±5.32 | 5.58±5.65 | 0.907 |
| Troponin I, pg/mL | 65.55±120.54 | 80.28±133.34 | 61.69±117.34 | 0.759 |
| Creatinine, mg/dL | 0.90±0.48 | 0.91±0.46 | 0.89±0.49 | 0.820 |
| Glucose, mg/dL | 146.77±73.65 | 154.14±60.16 | 144.77±76.99 | 0.495 |
| ALT, U/L | 29.51±39.67 | 44.89±71.14 | 25.41±24.40 | 0.008 |
| AST, U/L | 35.24±43.84 | 58.51±85.92 | 29.00±17.77 | <0.001 |
| White blood cell, cells/µL | 10.66±4.69 | 11.37±5.31 | 10.47±4.52 | 0.305 |
| Hemoglobin, g/dL | 11.89±2.13 | 11.40±1.73 | 12.02±2.21 | 0.119 |
| Platelets, cells/µL | 250.59±106.82 | 222.86±83.08 | 257.96±111.39 | 0.076 |
| Lymphocyte | 1.62±0.86 | 1.39±0.86 | 1.68±0.85 | 0.027 |
| Albumin | 34.76±6.26 | 29.95±5.91 | 36.04±5.72 | <0.001 |
| OPNI score | 42.86±8.06 | 36.92±7.05 | 44.44±7.58 | <0.001 |
| **Admission echocardiography** | | | | |
| Left ventricular ejection fraction, (%) | 55.42±9.79 | 51.14±11.57 | 56.72±8.84 | 0.003 |
| LV end-diastolic diameter, mm | 47.85±5.02 | 49.24±4.96 | 47.41±4.99 | 0.130 |
| Left atrial diameter, mm | 38.51±7.05 | 38.63±6.85 | 38.47±7.17 | 0.994 |
| Right ventricular diameter, mm | 28.60±6.42 | 28.83±5.98 | 28.54±6.58 | 0.764 |
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Continuous variables are presented as mean ± standard deviation, nominal variables presented as frequency (%). ALT: Alanine aminotransferase, AST: Aspartate aminotransferase, OPNI: Onodera's Prognostic Nutritional Index, sPAP: Systolic pulmonary artery pressure, TAPSE: Tricuspid Annular Plane Systolic Excursion, TR: Tricuspid regurgitation

**Table 3. Correlation of PESI score with baseline characteristics, echocardiographic findings, OPNI**

| Correlation between | R-value | p |
|---|---|---|
| Age | 0.597** | <0.001 |
| Systolic blood pressure | -0.222** | 0.004 |
| Diastolic blood pressure | -0.152* | 0.047 |
| Heart rate | 0.283** | <0.001 |
| RV TAPSE | -0.279 | 0.129 |
| LVEF | -0.420** | <0.001 |
| PASP | 0.312** | 0.002 |
| Troponin I | 0.182* | 0.035 |
| OPNI | -0.401** | <0.001 |

LVEF: Left ventricular ejection fraction, OPNI: Onodera's Prognostic Nutritional Index, PASP: Pulmonary artery systolic pressure, PESI: Pulmonary Embolism Severity Index, RV: Right ventricle, TAPSE: Tricuspid Annular Plane Systolic Excursion, *p<0.05, **p<0.01

shown that subjects with RV enlargement and dysfunction, as well as high pulmonary artery pressure, have a poor prognosis (11).

Instead of body mass index or serum albumin levels, which indicate nutritional status, physical, and immunologic competence, various scoring systems such as CONUT and OPNI have been developed (12,13). The fact that these scoring systems can be calculated simply and cost-effectively seems to be an advantage. The finding in this analysis suggests that lower OPNI values, which were significantly related to STM, indicate that nutritional status and immune function may play an important role in APE patients' outcomes.

Studies have shown that low albumin levels, usually an indicator of malnutrition or systemic inflammation, and decreased lymphocyte counts, an indicator of immunosuppression, affect mortality or poor survival in some diseases (14,15). A review of 29 studies showed that pre-treatment albumin levels below 35 g/L were associated with poor outcomes in almost all cancer types. In our study, the albumin level was 36.04±5.72 in the surviving group and 29.95±5.91 in the group with STM, values which are similar to those reported in the literature (16). It has been shown that decreased lymphocyte counts, which are an indicator of immunosuppression, are associated with mortality in subjects with pneumonia; with mortality at 13.6% between lymphocyte values 0-1 and 9.2% between lymphocyte values 1-2, and mortality decreased further with an increase in lymphocyte values (17). In our study, similar to the literature, lymphocyte values were found to be 1.39±0.86 lower in the STM group. In 220 small cell lung cancer subjects, an OPNI score <40 derived from pre-treatment albumin and lymphocyte values has been shown to be associated with low tolerance to chemotherapy and poor prognosis (4). From another perspective, the fact that it emerged as an independent predictor of STM in regression analysis and that a lower OPNI score (≤39.17) was related to an increased probability of STM supports the link between OPNI and poor outcome in APE patients in our study.

History of cancer and low oxygen saturation were found to be other important predictors of STM. In a cross-sectional study with 5,152 participants to determine whether low oxygen saturation was associated with increased mortality in the general adult population, $SpO_2$

**Table 4. The independent effects of some possible predictors in relation to short-term mortality according to univariate/multivariate analysis**

| | Univariate | | Multivariate | |
|---|---|---|---|---|
| | HR (95% CI) | p | HR (95% CI) | p |
| Age, years | 1.02 (0.99-1.04) | 0.167 | | |
| Female, gender | 2.02 (1.04-3.92) | 0.038 | 1.85 (0.88-3.88) | 0.102 |
| CHF | 2.37 (1.12-5.03) | 0.024 | 1.83 (0.76-4.42) | 0.181 |
| Malignite | 2.77 (1.44-5.30) | 0.002 | 2.25 (1.06-4.75) | 0.034 |
| $SaO_2$ | 0.93 (0.89-0.97) | 0.001 | 0.92 (0.87-0.96) | 0.001 |
| Systolic blood pressure | 0.99 (0.97-1.00) | 0.137 | | |
| Heart rate | 1.01 (1.99-1.03) | 0.085 | | |
| Troponin | 1.00 (0.99-1.00) | 0.471 | | |
| LVEF | 0.97 (0.94-0.99) | 0.011 | 0.99 (0.96-1.02) | 0.473 |
| PABS | 1.01 (0.98-1.05) | 0.418 | | |
| OPNI | 0.91 (0.87-0.95) | <0.001 | 0.93 (0.89-0.98) | 0.005 |

HR: Hazard ratios, CI: Confidence interval, CHF: Congestive heart failure, $SaO_2$: Arterial oxygen saturation, LVEF: Left ventricular ejection fraction, PASP: Pulmonary artery systolic pressure, OPNI: Onodera's Prognostic Nutritional Index

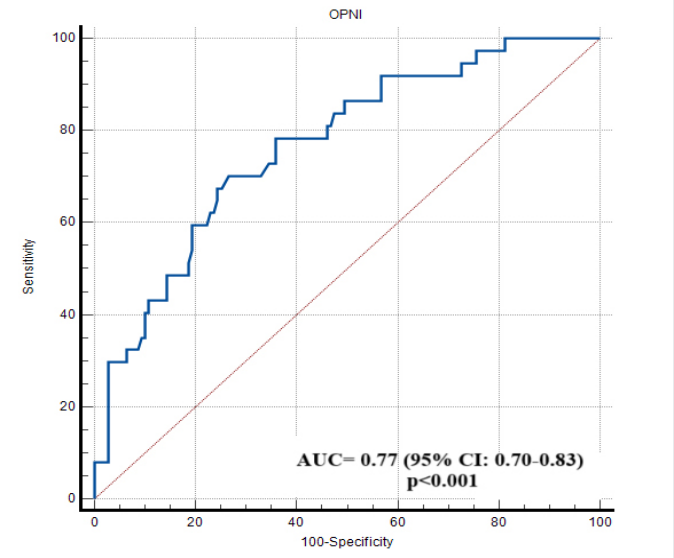


**Figure 1.** On receiver operating characteristic (ROC) analysis, area under the ROC curve (AUC) value of OPNI for short-term mortality was 0.77 (95% CI: 0.70-0.83, p<0.001)
CI: Confidence interval, OPNI: Onodera's Prognostic Nutrition Index

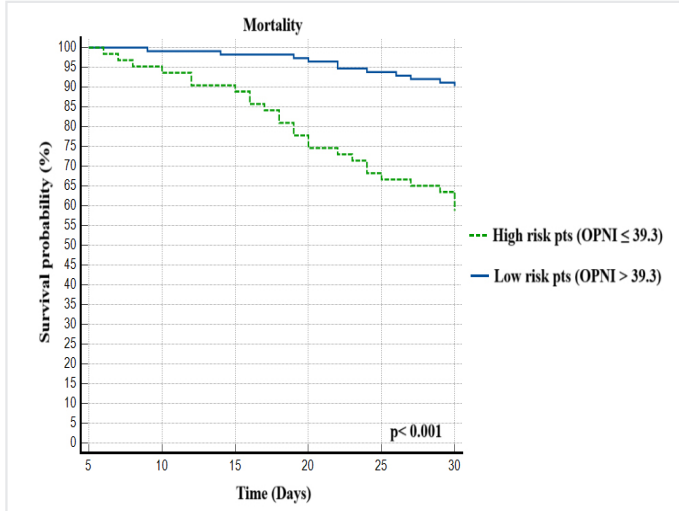


**Figure 2.** Kaplan-Meier plots show survival curves for low (blue line) and high (green line) risk patients based on the established OPNI cut-off value
OPNI: Onodera's Prognostic Nutrition Index

≤92% increased mortality by a factor of 1.73 times in a multivariable regression model for all-cause mortality. It has also been shown that in various diseases, which cause low oxygen levels, with or without lung pathology, increase mortality by worsening the burden on the RV and overall hemodynamic stability (18). In our study, oxygen saturation, a critical marker of lung function that can be evaluated quickly and easily, was also shown to be an important determinant of mortality. It is well known that a history of cancer is both a risk factor for PE and a predictor of mortality (19,20).

In a single-center registry study of 896 consecutive PE subjects followed for up to 14 years, Eckelt et al. (21) showed that a history of cancer was the strongest predictor of mortality compared to the general population, which is in line with our results.

### Study Limitations

Despite the promising results, our acedemic work has several limitations. First, the retrospective design, relatively small sample size, and small number of participating centers limit the study's generalizability. It needs to be validated with future appropriately sized, randomized, prospective studies. Second, while our study focused on STM, investigating the effect of OPNI on long-term mortality, may enhance the study's outcomes. Finally, OPNI was evaluated as a single parameter rather than an index; however, it can be used as an index with the inclusion of additional parameters such as echocardiographic findings and more detailed clinical characteristics.

## Conclusion

OPNI appears to be a promising, easily applicable, practitioner-independent and cost-effective parameter for predicting STM in subjects

with APE. Based on the findings from our study, the OPNI may be an effective complement or alternative to the PESI, especially in settings with limited access to comprehensive clinical data and when patient communication is difficult.

### Ethics

**Ethics Committee Approval:** This study was approved by the University of Health Sciences Türkiye, İstanbul Training and Research Hospital Ethics Committee (approval number: 73, date: 06.09.2024).

**Informed Consent:** Retrospective study.

### Footnotes

**Authorship Contributions:** Concept - M.K., Ş.T., G.Y., S.T., F.Ö., T.K., A.Ö.; Design - M.K., Ş.T., G.Y., S.T., F.Ö., T.K., A.Ö.; Data Collection or Processing - M.K., Ş.T., G.Y., S.T., F.Ö., T.K., A.Ö.; Analysis or Interpretation - T.K., A.Ö.; Literature Search - M.K.; Writing - M.K., T.K., A.Ö.

**Conflict of Interest:** No conflict of interest was declared by the authors.

**Financial Disclosure:** The authors declared that this study received no financial support.

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