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ПРОГНОСТИЧЕСКАЯ МОДЕЛЬ 12-МЕСЯЧНОЙ ЛЕТАЛЬНОСТИ У ПАЦИЕНТОВ, ВЫПИСАННЫХ ИЗ СТАЦИОНАРА ПОСЛЕ ПЕРЕНЕСЕННОЙ ТРОМБОЭМБОЛИИ ЛЁГОЧНОЙ АРТЕРИИ

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The Prognostic Model Of 12-Month Mortality in Patients Discharged from The Hospital After Pulmonary Embolism

Резюме

Цель. Определить клинико-лабораторные и инструментальные предикторы 12-месячной летальности у пациентов, выписанных из стационара после перенесенной тромбоэмболии лёгочной артерии (ТЭЛА) и разработать прогностическую модель. **Материал и методы.** В исследование включены 150 пациентов, выписанных из стационара после эпизода ТЭЛА. Оценивали демографические, анамнестические, клинические, лабораторные и эхокардиографические показатели. За конечную точку принимали смерть от любой причины в течение 12 месяцев после ТЭЛА. Для поиска независимых предикторов применяли одно- и многофакторную логистическую регрессию, дискриминацию модели оценивали по AUC, калибровку — по критерию Хосмера–Лемешоу и calibration plot; внутреннюю валидацию выполняли методом bootstrap. **Результаты.** За период наблюдения умерли 20 (13,3 %) пациентов. В многофакторную модель прогнозирования 12-месячной летальности вошли три независимых предиктора: уровень гемоглобина, расчетная скорость клубочковой фильтрации (рСКФ) и фракция выброса лево-

го желудочка (ФВ ЛЖ). Модель показала высокую дискриминационную способность (AUC 0,906; 95 % ДИ 0,852–0,960; p <0,001) и хорошую калибровку (χ^2=4,009; p=0,856). При пороговом значении p=0,08 чувствительность модели составила 100 %, специфичность — 69,2 %. Предложенная модель (шкала Mezo) продемонстрировала преимущество по AUC по сравнению со шкалами sPESI, ICOPER, GPS и Yamaki. **Заключение.** Шкала Mezo, включающая уровень гемоглобина, рСКФ и ФВ ЛЖ, обеспечивает высокую точность прогнозирования 12-месячной летальности у пациентов, выписанных из стационара после перенесенной ТЭЛА, и может использоваться после проведения внешней валидизации для ранней стратификации риска.

Ключевые слова: *тромбоэмболия лёгочной артерии, прогностическая шкала, летальность, фракция выброса левого желудочка, скорость клубочковой фильтрации, гемоглобин*

Конфликт интересов

Авторы заявляют, что данная работа, её тема, предмет и содержание не затрагивают конкурирующих интересов

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Abstract

Objective. To identify clinical, laboratory and instrumental predictors of 12-month mortality in patients after PE and to develop a prognostic model. **Material and methods.** This retrospective study included 150 patients discharged after an episode of PE (2021–2024). The diagnosis was confirmed predominantly by CT pulmonary angiography. Demographic, clinical, laboratory and echocardiographic parameters were assessed. The primary endpoint was death within 12 months after PE (excluding in-hospital and early mortality within 30 days). Univariable and multivariable logistic regression were used to identify independent predictors. Model discrimination was evaluated using the AUC, and calibration using the Hosmer–Lemeshow test and a calibration plot; internal validation was performed by bootstrap resampling. **Results.** During follow-up, 20 patients (13.3 %) died. Three independent predictors of 12-month mortality were included in the multivariable model: hemoglobin level, estimated glomerular filtration rate (eGFR) and left ventricular ejection fraction (LVEF). The model demonstrated high discriminatory ability (AUC 0.906; 95 % CI 0.852–0.960; p <0.001) and good calibration (χ^2=4.009; p=0.856). At the probability threshold p=0.08, sensitivity of the model was 100 % and specificity 69.2 %. The Mezo score showed higher AUC values compared with sPESI, ICOPER, GPS and the Yamaki scores. **Conclusion.** The Mezo score, based on hemoglobin level, eGFR and LVEF, provides high accuracy in predicting 12-month mortality in patients after PE and, after external validation, may be used for early risk stratification.

Key words: *pulmonary embolism, prognostic score, mortality, left ventricular ejection fraction, estimated glomerular filtration rate, hemoglobin*

Conflict of Interest

The authors declare that this work, its topic, subject matter, and content do not affect any competing interests.

Funding Sources

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Compliance with the principles of ethics

The study was approved by the Ethics Committee of the Medical Institute of the Peoples' Friendship University of Russia named after Patrice Lumumba (Protocol No. 1, January 17, 2025). Written informed consent was obtained from all participants.

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ALT — alanine aminotransferase, AST — aspartate aminotransferase, aPPT — activated partial thromboplastin time, DBP — diastolic blood pressure, CI — confidence interval, BMI — body mass index, INR — international normalised ratio, MSCT — multispiral computed tomography, OR — odds ratio, eGFR — estimated glomerular filtration rate, SBP — systolic blood pressure, sPAP — systolic pulmonary artery pressure, PATE — pulmonary artery thromboembolia, LVEF — left ventricle ejection fraction, echoCG — echocardiography, AUC — area under the curve, BNP — brain natriuretic peptide, ROC — receiver operating characteristic, SpO_2 — peripheral blood oxygen saturation

Introduction

Pulmonary artery thromboembolia (PATE) remains one of the leading causes of hospital deaths and plays a significant role in the structure of cardiovascular diseases [1–3]. Despite advances in diagnostic and therapeutic approaches, mortality associated with PATE remains high, including deaths occurring during the intermediate- and long-term periods following the acute event [4, 5].

A key objective of contemporary clinical practice is the early risk stratification of patients with a history of PATE, as this enables optimisation of follow-up, determination of the need for extended anticoagulant therapy, and prevention of adverse outcomes [6]. Long-term outcomes remain insufficiently studied [7, 8]. Existing prognostic scores demonstrate limited predictive value for assessing long-term outcomes after pulmonary embolism. The simplified Pulmonary Embolism Severity Index (sPESI) is primarily designed to estimate short-term risk and does not always capture the characteristics of disease progression in the long-term period [9–11]. The ICOPER model, which relies mainly on demographic and clinical variables, does not fully reflect the extent of organ dysfunction [12]. The GPS and Yamaki scores incorporate composite endpoints, which may reduce the accuracy of mortality prediction [13, 14].

In this context, the development of novel prognostic scores capable of estimating the risk of mortality during the first year of follow-up in patients discharged after an episode of PATE appears particularly relevant. Such scoring systems may serve as valuable tools for more accurate prognostication, risk stratification, and the individualisation of outpatient management.

Study objective

To identify clinical, laboratory, and instrumental predictors and to develop a predicative model for the 12-month mortality in patients discharged from the hospital after an episode of PATE.

Materials and methods

A total of 150 patients discharged after an episode of PATE between January 1, 2021 and December 30, 2024 were retrospectively included in the study. Patient identification and selection were performed at Kolomna Hospital, Outpatient Clinic No. 2 (84 patients), and Voskresensk Hospital, Outpatient Clinic No. 4 (66 patients).

The study was conducted in accordance with the ethical standards set forth in the Declaration of Helsinki and was approved by the Ethics Committee of the Medical Institute of RUDN University on January 17, 2025. All patient data were anonymised.

Study population and identification of outcomes

PATE was diagnosed based on clinical findings and confirmed by instrumental investigations. In the majority of cases, involving 139 (88.4 %) patients, the diagnosis was verified by contrast-enhanced multispiral computed tomography (MSCT) of the chest with visualisation of the pulmonary artery and its branches. In 11 (11.6 %) cases, when contrast administration was contraindicated or MSCT was technically unfeasible, the diagnosis was confirmed by echocardiographic (echoCG) findings demonstrating signs of right heart overload and pulmonary hypertension.

All patients received anticoagulant therapy and, when indicated, thrombolytic treatment during hospitalisation in accordance with the current clinical practice guidelines of the European Society of Cardiology (2019). Following discharge, the choice of anticoagulant, as well as the duration and regimen of therapy, were determined individually based on the assessment of the risks of recurrent venous thromboembolism and bleeding.

Within the framework of the study, demographic data, clinical characteristics, and laboratory and instrumental parameters were analysed. Demographic and clinical variables were assessed at the time of hospital admission. Laboratory and instrumental investigations were performed during hospitalisation before discharge.

Twelve months later, patients were contacted to assess outcomes. Patients were divided into groups according to the occurrence or absence of all-cause mortality during the 12-month follow-up period after hospital discharge.

Statistical analysis

Categorical variables are presented as absolute values and percentages, whereas quantitative variables are expressed as either the median and interquartile range (Me [IQR]) or the mean ± standard deviation (M ± SD), depending on the distribution of the data. The normality of quantitative attributes was assessed using the Kolmogorov-Smirnov test. Comparisons of quantitative variables between two independent groups were performed using the Mann-Whitney U test for non-normally distributed data and Student's t-test for normally distributed data. Differences in categorical variables were analysed using the χ^2 (chi-square) test or Fisher's exact test when expected frequencies were small. Differences were considered statistically significant at $p < 0.05$.

To identify independent factors associated with mortality, univariate logistic regression analysis was performed, with the calculation of odds ratios (ORs) and 95 % confidence intervals (CIs). Variables demonstrating

statistical significance in the univariate analysis were subsequently entered into a multivariable binary logistic regression model using a stepwise selection approach. The performance of the resulting model was evaluated by assessing its discriminatory ability through ROC analysis, including calculation of AUC, sensitivity, specificity, and the optimal probability threshold (cut-off) determined according to the Youden index. Model calibration was assessed using the Hosmer-Lemeshow goodness-of-fit test and by graphical comparison of predicted and observed probabilities (calibration plot).

Internal validation of model stability was performed using bootstrap analysis with 1,000 resamples, allowing estimation of standard errors, confidence intervals, and regression coefficient bias.

Statistical analyses were conducted using IBM SPSS Statistics version 23.0 and R version 4.2.0, with the *pROC*, *rms*, *ggplot2* packages.

Results

During the follow-up period, 20 patients (13.3 %) died (Table 1). Recurrent PATE was the cause of death in 9 cases (45 % of all deaths), malignant neoplasms accounted for 8 deaths (40 %), and the cause of death remained unknown in 3 patients (15 %). In the overall cohort, men comprised 44.7 % of patients, with no significant differences between the groups (p = 1.000). Patients who died were significantly older than survivors, with a median age of 68 years vs. 63.5 years, respectively (p = 0.043). Body mass index tended to be lower among deceased patients (28.6 vs. 31.7 kg/m², p = 0.055).

No significant differences were observed between the groups with respect to the prevalence of major cardiovascular diseases, with the exception of lower-extremity deep vein thrombosis, which was less common among patients who died (25.0 % vs. 70.0 %, p < 0.001).

Table 1. *Demographic and clinical characteristics of the studied patients*

Characteristic	Total (n = 150)	Survival group (n = 130)	Mortality group (n=20)	p-value (between groups)
Male sex, n (%)	67 (44,7 %)	58 (44,6 %)	9 (45,0 %)	1,000
Age (years), Me [IQR]	64,00 [57,00; 71,00]	63,50 [56,00; 70,00]	68,00 [63,00; 75,50]	0,043*
Body mass index (kg/m²), M (SD)	31,27 (±6,83)	31,69 (±6,57)	28,55 (±7,96)	0,055
Coronary artery disease, n (%)	36 (24,0 %)	31 (23,8 %)	5 (25,0 %)	1,000
Arterial hypertension, n (%)	115 (76,7 %)	99 (76,2 %)	16 (80,0 %)	1,000
Lower extremity deep vein thrombosis, n (%)	96 (64,0 %)	91 (70,0 %)	5 (25,0 %)	<0,001*
Atrial fibrillation, n (%)	107 (71,3 %)	91 (70,0 %)	16 (80,0 %)	0,402
History of surgical intervention, n (%)	20 (13,3 %)	16 (12,3 %)	4 (20,0 %)	0,310
Active cancer, n (%)	43 (28,7 %)	31 (23,8 %)	12 (60,0 %)	0,002*
Chronic heart failure, n (%)	63 (42,0 %)	55 (42,3 %)	8 (40,0 %)	0,846
History of gastric and duodenal ulcer disease, n (%)	17 (11,3 %)	13 (10,0 %)	4 (20,0 %)	0,247
Pulmonary infarction, n (%)	21 (14,0 %)	18 (13,8 %)	3 (15,0 %)	1,000
Diabetes mellitus, n (%)	25 (16,7 %)	22 (16,9 %)	3 (15,0 %)	1,000
Lower extremity varicose veins, n (%)	34 (22,7 %)	32 (24,6 %)	2 (10,0 %)	0,249
History of stroke/transient ischemic attack, n (%)	13 (8,7 %)	11 (8,5 %)	2 (10,0 %)	0,685
Chronic non-inflammatory lung diseases, n (%)	25 (16,7 %)	21 (16,2 %)	4 (20,0 %)	0,812
Kidney disease, n (%)	71 (47,3 %)	60 (46,2 %)	11 (55,0 %)	0,481
Anemia, n (%)	64 (42,7 %)	45 (34,6 %)	19 (95,0 %)	<0,001*

Note: * — differences are statistically significant (p < 0.05)

In contrast, the prevalence of malignant neoplasms was significantly higher in this group (60.0 % vs. 23.8 %, p = 0.002).

Other comorbid conditions, including atrial fibrillation, diabetes mellitus, peptic ulcer disease, chronic lung disease, and chronic kidney disease, did not differ significantly between the groups (p > 0.05). Notably, anaemia was markedly more prevalent among patients who died: this condition was present in 95 % of deceased patients compared with 34.6 % of survivors (p < 0.001).

Analysis of laboratory and instrumental test results obtained during hospitalisation before discharge revealed several significant differences between survivors and non-survivors (Table 2). Patients who died had

Table 2. *Comparison of clinical, laboratory, and instrumental parameters between the study groups.*

Characteristic	Total (n = 150)	Survival group (n = 130)	Mortality group (n=20)	p-value (between groups)
sBP (mmHg), Me [IQR]	130,00 [102,00; 157,00]	131,00 [110,00; 159,00]	110,00 [89,00; 146,25]	0,025*
dBP (mmHg), Me [IQR]	80,00 [65,00; 90,00]	80,00 [70,00; 90,00]	60,50 [51,50; 80,50]	0,007*
SPO2 (%), Me [IQR]	91,00 [87,00; 93,00]	92,00 [87,00; 94,00]	88,00 [84,75; 90,50]	0,005*
White blood cell (×10⁹/L), Me [IQR]	8,60 [6,30; 10,30]	8,50 [6,50; 10,10]	9,50 [4,65; 10,90]	0,643
Platelet (×10⁹/L), Me [IQR]	244,00 [180,00; 301,00]	250,00 [187,00; 323,75]	180,00 [133,00; 227,00]	<0,001*
Red blood cell (×10⁹/L), Me [IQR]	4,60 [4,10; 4,90]	4,60 [4,21; 4,90]	3,90 [3,58; 4,95]	0,053
Hemoglobin (g/L), Me [IQR]	123,00 [105,00; 135,50]	125,00 [112,00; 136,00]	102,00 [89,60; 109,50]	<0,001*
Urea (mg/dL), Me [IQR]	7,80 [6,72; 8,67]	7,55 [6,62; 8,50]	8,75 [8,18; 10,20]	0,001*
AST (U/L), Me [IQR]	25,91 [18,70; 41,30]	25,50 [18,30; 38,00]	48,35 [25,25; 98,22]	0,002*
ALT (U/L), Me [IQR]	29,65 [19,00; 53,10]	26,00 [18,52; 45,95]	57,65 [36,15; 104,45]	<0,001*
Total cholesterol (mmol/L), Me [IQR]	5,60 [4,90; 6,50]	5,37 [4,86; 6,40]	6,50 [5,55; 7,35]	0,018*
Glucose (mmol/L), Me [IQR]	6,00 [5,20; 6,84]	5,85 [5,18; 6,80]	6,40 [5,70; 8,85]	0,043*
BNP (pg/mL), Me [IQR]	375,70 [99,75; 884,00]	367,00 [85,59; 854,00]	501,00 [248,10; 1069,00]	0,193
Creatinine (μmol/L), Me [IQR]	102,00 [86,23; 118,73]	99,00 [85,80; 112,00]	124,00 [104,00; 137,50]	<0,001*
eGFR (mL/min/1.73 m²), M (SD)	52,14 (±14,21)	53,99 (±13,48)	40,08 (±13,22)	<0,001*
D-dimer (μg/L), Me [IQR]	2358,00 [1164,62; 3265,00]	2276,00 [1027,75; 3254,00]	3261,50 [1370,50; 3630,75]	0,149
INR (IU/mL), Me [IQR]	1,47 [1,085; 1,815]	1,37 [1,08; 1,67]	1,76 [1,58; 1,85]	0,003*
Fibrinogen (g/L), Me [IQR]	4,23 [3,87; 5,02]	4,23 [3,85; 4,89]	4,19 [3,99; 5,23]	0,648
aPTT (s), Me [IQR]	35,40 [30,12; 41,50]	35,70 [29,88; 41,50]	34,30 [30,40; 40,27]	0,951
Prothrombin time (s), Me [IQR]	14,80 [13,50; 15,80]	14,80 [13,50; 15,80]	14,80 [13,57; 15,20]	0,614
sPAP (mmHg), Me [IQR]	40,00 [28,75; 50,00]	38,00 [28,00; 48,00]	52,50 [40,00; 60,00]	<0,001*
LVEF (%), M (SD)	54,54 (±9,23)	55,79 (±9,02)	46,40 (±5,88)	<0,001*
Main pulmonary artery diameter (mm), Me [IQR]	31,00 [30,00; 34,00]	31,00 [30,00; 34,00]	34,50 [31,00; 35,75]	0,003*

Note: * — differences are statistically significant (p < 0.05).
Abbreviations: *sBP* — systolic blood pressure; *dBP* — diastolic blood pressure; *SpO_2* — peripheral oxygen saturation; *AST* — aspartate aminotransferase; *ALT* — alanine aminotransferase; *BNP* — B-type natriuretic peptide; *eGFR* — estimated glomerular filtration rate; *INR* — international normalized ratio; *aPTT* — activated partial thromboplastin time; *sPAP* — systolic pulmonary artery pressure; *LVEF* — left ventricular ejection fraction; *Me* — median; *IQR* — interquartile range; *M* — mean; *SD* — standard deviation.

lower haemoglobin levels (102 vs. 125 g/L, p < 0.001) and lower platelet counts (median 180 × 10^9/L vs. 250 × 10^9/L in survivors; p < 0.001). No significant differences were observed between the groups with respect to other peripheral blood parameters.

Among haemostatic parameters, a significant difference was observed in the international normalised ratio (INR), which was higher in patients who died (p = 0.003), whereas no differences were found in fibrinogen levels, aPTT, or prothrombin time (p > 0.6). D-dimer levels tended to be higher in the mortality group; however, this difference did not reach statistical significance (p = 0.149).

Among biochemical parameters, the most pronounced differences were noted in markers of renal and hepatic function. Patients who died had a lower eGFR (40 vs. 54 mL/min/1.73 m^2, p < 0.001) and higher levels of urea (p = 0.001) and creatinine (p < 0.001). The activities of hepatic transaminases (AST and ALT) were also higher in the non-survivor group (p = 0.002 and p < 0.001, respectively), as were markers of lipid and carbohydrate metabolism, including total cholesterol and glucose levels (p = 0.018 and p = 0.043, respectively).

Among the haemodynamic parameters, lower blood pressure levels (both systolic and diastolic; p = 0.025 and p = 0.007, respectively) and lower oxygen saturation (p = 0.005) were observed in the mortality group.

Echocardiographic assessment demonstrated that patients who died had significantly higher systolic pulmonary artery pressure (sPAP; p < 0.001), a larger pulmonary trunk diameter (p = 0.003), and a lower left ventricular ejection fraction (46.4±5.9 % vs. 55.8±9.0 % in survivors; p < 0.001).

Overall, non-survivors were characterised by signs of multiorgan involvement, including reduced haemoglobin levels, impaired hepatic and renal function, hypoxemia, and pronounced haemodynamic disturbances.

Analysis of laboratory and instrumental test results obtained during hospitalisation before discharge revealed several significant differences between survivors and non-survivors (Table 2). Patients who died had lower haemoglobin levels (102 vs. 125 g/L, p < 0.001) and lower platelet counts (median 180 × 10^9/L vs. 250 × 10^9/L in survivors; p < 0.001). No significant differences were observed between the groups with respect to other peripheral blood parameters.

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Among the haemodynamic parameters, lower blood pressure levels (both systolic and diastolic; p = 0.025 and p = 0.007, respectively) and lower oxygen saturation (p = 0.005) were observed in the mortality group.

Echocardiographic assessment demonstrated that patients who died had significantly higher systolic pulmonary artery pressure (sPAP; p < 0.001), a larger pulmonary trunk diameter (p = 0.003), and a lower left ventricular ejection fraction (46.4±5.9 % vs. 55.8±9.0 % in survivors; p < 0.001).

Overall, non-survivors were characterised by signs of multiorgan involvement, including reduced haemoglobin levels, impaired hepatic and renal function, hypoxemia, and pronounced haemodynamic disturbances.

Table 3. *Results of univariate and multivariate logistic regression analysis of factors associated with 12-month mortality*

Predictors	Univariate analysis			Multivariate analysis		
	OR	95 % CI	p-value	OR	95 % CI	p-value
Presence of malignancy	4,790	[1,795–12,781]	0,002*	–—	–—	
eGFR (mL/min/1.73 m^2)	0,910	[0,867–0,956]	<0,001*	0,935	[0,884–0,989]	0,019*
Hemoglobin (g/L)	0,948;	[0,922–0,974]	<0,001*	0,956	[0,928–0,985]	0,003*
LVEF (%)	0,886	[0,832–0,942]	<0,001*	0,884	[0,819–0,953]	0,001*

Note: * — indicates a statistically significant effect of the predictor (p < 0.05)
Abbreviations: *eGFR* — estimated glomerular filtration rate; *LVEF* — left ventricular ejection fraction.

Lower haemoglobin levels were associated with an increased probability of death (OR 0.956; 95 % CI 0.928–0.985; p = 0.003). Similarly, reduced LVEF was associated with a higher risk of mortality (OR 0.884; 95 % CI 0.819–0.953; p = 0.001), whereas a decrease in eGFR was associated with a 6 % increase in mortality risk for each 1 mL/min/1.73 m² reduction (OR 0.935; 95 % CI 0.884–0.989; p = 0.019).

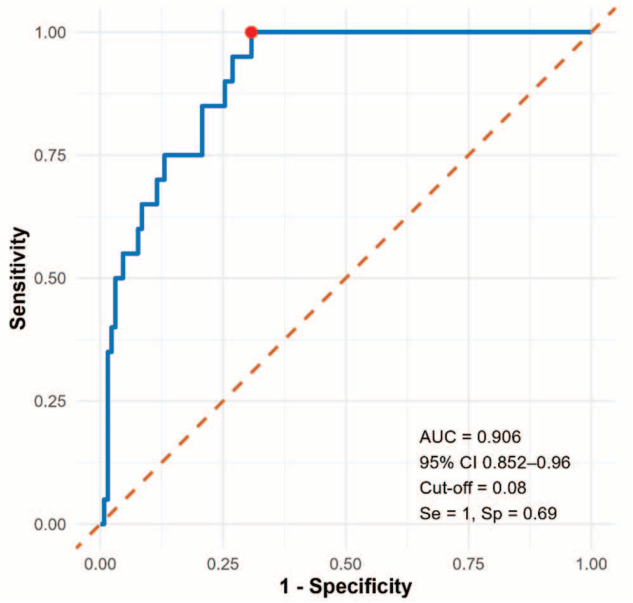

Figure 1. *ROC curve demonstrating the discriminative ability of the regression model in predicting mortality*

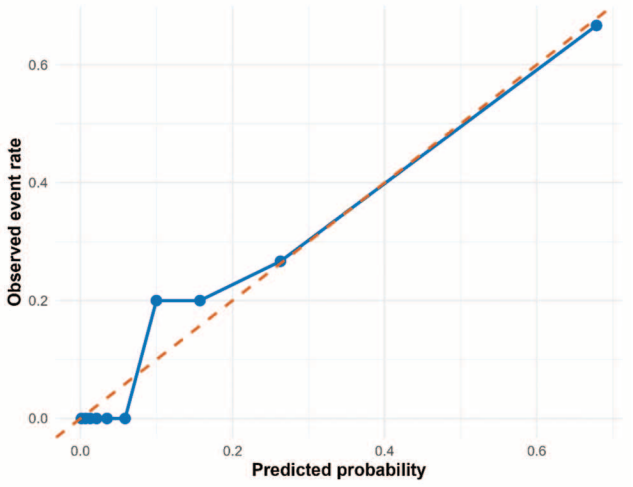

Figure 2. *Calibration plot of the mortality prediction model*

To quantify the predictive performance of the model, the area under ROC curve was calculated. The AUC was 0.906 (95 % CI 0.852–0.960; p < 0.001), indicating excellent discriminative ability for distinguishing between patients with favourable and unfavourable outcomes (Figure 1). The Hosmer-Lemeshow goodness-of-fit test (χ^2 = 4.009; p = 0.856) confirmed good agreement between predicted and observed probabilities, demonstrating adequate model calibration.

Visual inspection of the calibration plot (Figure 2) demonstrated some deviation of the empirical curve from the line of identity at low predicted probabilities, whereas good agreement was observed at intermediate and high probability values.

ROC curve analysis was used to evaluate the sensitivity and specificity characteristics of the model and to determine the optimal probability threshold (cut-off = 0.08). At this threshold, the developed model, designated the Mezo score, provided the best balance between true-positive and true-negative classifications (Table 4). The model demonstrated a sensitivity of 100 % and a specificity of 69.2 %, indicating its ability to reliably identify patients at high risk of mortality while maintaining a relatively low rate of false-positive predictions.

Internal validation of the model using bootstrap resampling (1,000 samples) demonstrated minimal coefficient bias (|bias| ≤ 0.02) for haemoglobin level, eGFR, and left ventricular ejection fraction, indicating a high degree of model stability and the absence of evidence of overfitting.

The logistic regression equation is as follows:

$$logit(P) = 12{,}648 - 0{,}045 \times Hb - 0{,}067 \times eGFR - \\ - 0{,}124 \times LVEF$$

where P denotes the probability of mortality, Hb is the haemoglobin level (g/L), eGFR is the estimated glomerular filtration rate (mL/min/1.73 m²), and LVEF is the left ventricular ejection fraction (%).

For practical application, the probability of mortality was calculated using the following formula:

$$P = \frac{1}{1 + e^{-(12{,}648 - 0{,}045 \times Hb - 0{,}067 \times \text{рСКФ} - 0{,}124 \times \text{ФВЛЖ})}}$$

Table 4. *Distribution of clinical outcomes according to risk group*

Risk group	Total number of patients	Number of deaths	χ^2	p-value
Low risk (<0.08)	90	0	34,615	<0,001*
High risk (≥0.08)	60	20		

Note: * — differences are statistically significant (p < 0.05)

The negative coefficients for haemoglobin, eGFR, and LVEF indicate that lower values of these parameters are associated with an increased probability of mortality.

Based on this equation, an online calculator was developed and is available at the following link: https://htmlpreview.github.io/?https://gist.githubusercontent.com/musa199692/40b282f3306bb9dee5cb05cb852eba4d/raw/93a1b570ecf7d7101b7e423e39a0e97257cd2930/mezo-calculator.html.

When compared with several established prognostic scores, including the sPESI [15], ICOPER [16], GPS [17], and Yamaki [18] scores, the Mezo score demonstrated superior discriminative performance and overall predictive accuracy (Figure 3).

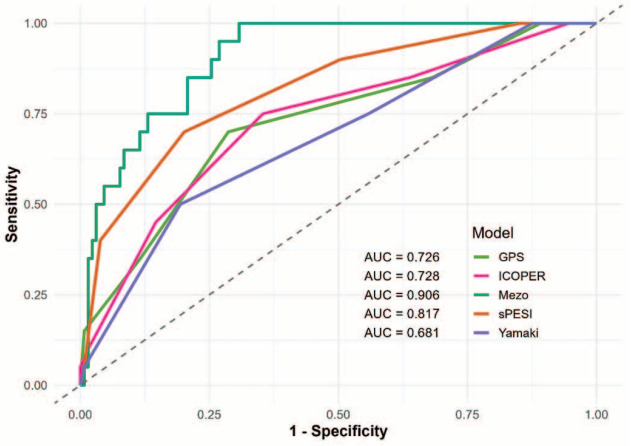

Figure 3. *Comparison of ROC curves of prognostic models*

Discussion

The prognostic model developed in the present study, based on haemoglobin level, estimated glomerular filtration rate, and left ventricular ejection fraction, demonstrated high accuracy in predicting mortality among patients discharged after an episode of PATE. The model exhibited excellent discriminative ability and satisfactory calibration, indicating its robustness and potential clinical utility for individualised risk assessment.

All patients after an acute episode of PATE should receive anticoagulant therapy for at least three months. The duration of further treatment depends on the balance between the risks of recurrence and bleeding [15, 16]. In patients with active malignancy and no increased bleeding risk, continuation of direct oral anticoagulant (DOAC) therapy is recommended for as long as the malignancy remains active and throughout the course of anticancer treatment [17]. The Mezo score may serve as a tool for early risk stratification and support decision-making regarding extended anticoagulant therapy in the early period after hospital discharge.

The lower prognostic performance of the sPESI, ICOPER, GPS, and Yamaki scores observed in our study is likely attributable to their original design, which focused primarily on predicting short-term outcomes or composite endpoints, as well as to their limited consideration of the extent of organ dysfunction. In contrast, the Mezo score incorporates quantitative parameters reflecting systemic impairment. This approach enables more accurate identification of patients at high risk of mortality and may explain the superior discriminative performance of the model.

Despite its high predictive accuracy and excellent discriminative performance, the proposed prognostic score has several limitations.

First, the model has not undergone formal external validation in an independent cohort, which limits the generalisability of its application to other patient populations.

Second, the analysis was performed on a relatively small sample size (n = 150), which increases the risk of overfitting and may reduce the stability of the model coefficients. In addition, the model was derived from retrospective data; therefore, the influence of unmeasured confounders and selection bias cannot be excluded.

Third, the model incorporated only a limited number of clinical and laboratory variables. The inclusion of additional parameters could potentially improve its predictive performance; however, this would require a larger sample size to maintain the statistical reliability and stability of the prognostic model.

Finally, the lack of statistical significance of malignancy in the multivariable analysis is likely attributable to the relatively small sample size and the limited duration of follow-up. Nevertheless, an indirect effect of the oncological process on prognosis, mediated through the systemic disturbances captured by the variables included in the final model, cannot be excluded.

The findings of the present study highlight several directions for future research. A stratified analysis of patients with cancer-associated pulmonary embolism and those without malignancy in a larger cohort appears particularly promising, as it may provide further insight into the contribution of the underlying malignancy and anticancer therapy to long-term outcomes. In addition, prospective multicentre studies aimed at validating the proposed prognostic model are warranted to assess its reproducibility and clinical utility across different patient populations.

Conclusion

The risk score developed in the present study for predicting long-term mortality in patients with a history of PATE is based on readily available clinical parameters and may be used for risk stratification and individualised

outcome prediction. However, despite its excellent discriminative performance and satisfactory calibration, the model requires further external validation in independent cohorts, as well as prospective studies to confirm its prognostic value and applicability in routine clinical practice.

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