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Hospital Admission Rates, Determinants, and 30-day Outcomes in sPESI = 0 Patients With Acute Pulmonary Embolism: A Single-Center Retrospective Cohort Study

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Abstract

Aim

This study aimed to determine the proportion of sPESI = 0 patients with acute pulmonary embolism admitted to hospital at a single-center emergency department, to identify factors associated with the admission decision, and to compare 30-day adverse event rates between admitted and discharged patients as an exploratory objective.

Methods

This retrospective cohort study included adults with CTPA-confirmed acute symptomatic pulmonary embolism presenting to the emergency department of a tertiary care hospital in Istanbul, Turkey, between January 2015 and April 2026. Patients were classified by retrospective sPESI calculation. The guideline-discordant admission rate among sPESI = 0 patients was tested against a 30% reference value. Factors associated with admission were evaluated using a pre-specified logistic regression model with bootstrap internal validation. Thirty-day outcomes were compared using risk differences with Newcombe hybrid score 95% confidence intervals and Benjamini-Hochberg correction.

Results

Among 594 patients, 267 (44.9%) were classified as sPESI = 0. Of these, 102 (38.2%; 95% CI 32.6–44.2%) were formally admitted, significantly exceeding the 30% reference (p = 0.006). After excluding 33 patients who received high-acuity interventions, the admission rate fell to 32.1% (p = 0.501). The logistic regression model yielded an optimism-corrected AUROC of 0.555; no predictor reached statistical significance. Troponin was measured in only 39 of 267 patients (14.6%). The 30-day composite adverse event rate was 7.8% in admitted versus 3.6% in discharged patients (risk difference + 4.2 percentage points; 95% CI − 1.4 to + 11.4; FDR-adjusted p = 0.313). Five deaths occurred (1.9%): four in admitted patients and one in a discharged patient who was subsequently readmitted to the ICU.

Conclusion

The observed guideline-discordant admission rate among sPESI = 0 patients was partly attributable to clinically justified admissions for high-acuity patients in whom the score underestimated severity. The admission decision was not explained by guideline-recommended risk stratification variables, and troponin assessment was performed in a small minority. Implementation of a standardized biomarker-inclusive risk assessment protocol may warrant evaluation to align institutional practice with current guideline recommendations. Prospective multicenter validation is required before these findings can inform clinical pathways.

INTRODUCTION

Acute pulmonary embolism affects approximately 60 to 120 per 100,000 persons per year and causes an estimated 60,000 to 100,000 deaths annually in the United States alone.(1) The condition accounts for a substantial proportion of emergency department encounters, and short-term mortality varies widely depending on hemodynamic status and the extent of right ventricular compromise.(2, 3) Accurate risk stratification at the time of diagnosis is therefore central to determining the appropriate level of care.

The simplified Pulmonary Embolism Severity Index (sPESI), derived in 995 patients and externally validated in 7,106 patients from the RIETE registry, classifies patients with none of six adverse features (age > 80 years, active cancer, chronic cardiopulmonary disease, heart rate ≥ 110 beats per minute, systolic blood pressure < 100 mmHg, and arterial oxygen saturation < 90%) as low-risk, with a 30-day mortality below 2%.(4) On the basis of this and similar evidence, the 2019 European Society of Cardiology (ESC) guidelines recommend outpatient management or early discharge for hemodynamically stable patients classified as low-risk by the sPESI or the Hestia criteria, provided no other conditions necessitate hospitalization(2); the 2026 AHA/ACC multisociety guideline further reinforced this position by introducing a clinical category system in which patients with low clinical severity are considered appropriate for outpatient management or early discharge.(5) Randomized and observational data support the safety of this approach.(6, 7) However, sPESI alone does not incorporate biomarker or imaging markers of right ventricular dysfunction, and patients classified as low risk may still harbor elevated troponin levels or right ventricular dilation, prompting uncertainty about whether sPESI = 0 classification is sufficient for safe discharge without additional risk assessment.(8, 9) Implementation of outpatient management for these patients remains limited; system-level barriers including delayed imaging access, anticoagulant dispensing logistics, and clinician-level risk aversion continue to drive hospitalization rates that exceed guideline expectations, yet the patient-level and institutional determinants of admission in sPESI = 0 patients have not been formally characterized.(10, 11)

This study aimed to determine the proportion of sPESI = 0 patients with acute pulmonary embolism admitted to hospital in a single-center emergency department setting and to identify patient-level and institutional factors associated with hospital admission. As a secondary exploratory objective, 30-day adverse event rates were compared between admitted and discharged patients to generate safety signals for future prospective studies.

METHODS

2.1 Study Design and Setting

This single-center retrospective cohort study was conducted at the Emergency Medicine Department of Istanbul Medipol University Hospital, a tertiary care academic medical center with an annual emergency department volume of approximately [X] patient visits and round-the-clock computed tomography pulmonary angiography (CTPA) capability. The study analyzed data from patients presenting between

January 2015 and April 2026. Case identification was performed through systematic screening of the Picture Archiving and Communication System (PACS) radiology archive rather than ICD-10 discharge coding, in order to capture cases that may have been miscoded or uncoded at discharge. Each radiology report containing the terms ‘pulmonary embolism’, ‘pulmonary thromboembolism’, or equivalent descriptors was reviewed, classified as acute PE present or absent, and subsequently linked to the hospital information system (HBS) for retrieval of clinical records.

2.2 Research Questions and Study Objectives

The study was designed to address three sequentially related questions regarding the management of low-risk pulmonary embolism in routine emergency department practice.

Research Question 1 (Descriptive)

Among patients with CTPA-confirmed acute PE classified as low-risk by retrospective sPESI = 0, what proportion are formally admitted to the hospital (ward or intensive care unit) rather than managed as outpatients?

Research Question 2 (Analytic)

Which patient-level clinical features and institutional factors independently predict hospital admission in sPESI = 0 patients?

Research Question 3 (Evaluative)

Among sPESI = 0 patients, do 30-day adverse event rates differ between those admitted and those discharged?

Primary hypothesis: The proportion of sPESI = 0 patients admitted to the hospital differs from 30% (H_0: $p = 0.30$; H_1: $p \neq 0.30$). This reference value was selected a priori as the maximum admission rate consistent with majority outpatient management of low-risk pulmonary embolism, as recommended by the 2019 ESC (2) and 2026 AHA/ACC (5) guidelines; an admission rate at or below 30% would indicate that at least 70% of sPESI = 0 patients are managed without hospitalization.

2.3 Participants

Adults aged 18 years or older with a CTPA-confirmed diagnosis of acute symptomatic pulmonary embolism who presented to the emergency department during the study period were eligible. Exclusion criteria were: incidental PE detected on imaging performed for other indications without corresponding symptoms; massive PE presenting with cardiac arrest or refractory hypotension (systolic blood pressure < 90 mmHg despite fluid resuscitation, or vasopressor requirement at initial presentation); chronic thromboembolic disease (symptom duration exceeding four weeks or imaging features of chronicity); pregnancy; encounters with insufficient documentation to calculate all six sPESI components; and

duplicate encounters for the same thromboembolic episode (index encounter retained). Screening, exclusion, and final cohort numbers are reported in a flow diagram.

2.4 Primary Predictor: sPESI Classification

The simplified Pulmonary Embolism Severity Index (sPESI) was calculated retrospectively for each patient from medical records and the electronic health system by a single investigator using a standardized data collection form. The six binary components were operationally defined as follows: age greater than 80 years (identity document); active cancer (chemotherapy, radiotherapy, or targeted therapy within six months, or metastatic disease, confirmed from oncology records or active medication lists); chronic cardiopulmonary disease (previously documented congestive heart failure or chronic lung disease, verified by prior discharge summaries or ICD-10 codes); heart rate ≥ 110 beats per minute (first recorded measurement within 30 minutes of ED arrival); systolic blood pressure < 100 mmHg (same timepoint); and arterial oxygen saturation < 90% on room air (triage pulse oximetry). When SpO_2 was recorded only under supplemental oxygen, this component was treated as missing and handled according to the protocol described in Section 2.8.

sPESI classification was performed by a single investigator. Patients with sPESI = 0 were classified as low-risk; those with sPESI ≥ 1 as non-low-risk.

2.5 Variables

Demographics and presentation. Age (continuous, years), sex, symptom onset to presentation time (hours), admission mode (self-referral, ambulance, transfer), weekend or holiday presentation, and off-hours presentation (weekdays 17:00 to 08:00 and all weekend/holiday hours).

Comorbidities. Active cancer (with type), heart failure, coronary artery disease, COPD/asthma, chronic kidney disease, diabetes, hypertension, atrial fibrillation, prior stroke or TIA, and obesity (BMI ≥ 30 or documented diagnosis).

Vital signs. Systolic and diastolic blood pressure, heart rate, respiratory rate, SpO_2 (with documentation of room air versus supplemental oxygen), temperature, and Glasgow Coma Scale, all at initial ED assessment. Worst values during ED stay (lowest SBP, highest heart rate, lowest SpO_2) were extracted separately from monitoring records.

Laboratory values. Troponin (institutional assay; upper reference limit recorded), BNP, NT-proBNP, hemoglobin, platelet count, white blood cell count, creatinine, eGFR (CKD-EPI), D-dimer (standardized to ng/mL FEU), INR, and lactate. Variables with > 60% missingness (D-dimer, lactate) were excluded from regression models and reported descriptively in the available subset only.

Imaging findings. PE anatomical location (central [main pulmonary artery or lobar] versus peripheral [segmental or subsegmental]), bilateral involvement, saddle embolus, right ventricular to left ventricular (RV/LV) diameter ratio measured on axial CTPA images at the level of maximum ventricular diameter by

a single investigator using the hospital PACS measurement tool (dichotomized as > 1 versus ≤ 1), RV dilation, septal bowing, pulmonary infarction, and pleural effusion. Echocardiographic data (RV dysfunction, TAPSE, estimated PASP) were recorded for patients with index-encounter echocardiography.

Institutional variables. Off-hours presentation, extracted from HBS records. Calendar year of presentation was included in the original protocol but was excluded from all analyses because patient-level date de-identification with random shifts prevented recovery of actual calendar years (Spearman ρ between subject identifier and shifted date = 0.018). Clinician position at the disposition decision (attending versus resident) was included in the original study protocol but could not be reliably extracted from archival records for the full study period and was therefore excluded from all analyses.

Three-category coding for variables with informative missingness. Two predictor variables were frequently absent because the test was ordered or the finding was reported selectively rather than routinely: RV/LV diameter ratio and PE anatomical location. For each, a three-category classification was applied: (0) measured or reported and normal/negative, (1) measured or reported and abnormal/positive, (2) not measured or not reported. This retains all patients, avoids misclassifying unmeasured patients as normal, and permits the unmeasured category to enter regression models as a separate indicator.

Troponin was handled separately due to extreme measurement sparsity: in the sPESI = 0 subgroup, troponin measurement was infrequent (see Results), with only 3 patients classified as normal. For the primary regression model, troponin was coded as a binary variable: (1) measured and elevated versus (0) all other patients (measured and normal or not measured). The three-category coding (normal / elevated / not measured) is retained as a pre-specified sensitivity analysis; however, the 3-patient ‘normal’ cell produces unstable coefficient estimates and is reported with appropriate caveats.

Risk marker assessment completeness. Three additional risk markers beyond sPESI were recorded for sPESI = 0 patients: troponin elevation, RV/LV diameter ratio > 1, and central PE location. Each was coded using the three-category system described above. Because troponin was measured in only 39 of 267 sPESI = 0 patients (14.6%), and no patient had all three markers measured and classified as normal, risk-marker-stratified outcome comparisons as originally planned were not feasible. Testing completeness rates are reported descriptively. Outcome comparisons for Research Question 3 are therefore conducted between admitted and discharged sPESI = 0 patients without risk marker stratification.

ED management. Anticoagulation initiated in ED (type), thrombolysis, catheter-directed therapy, surgical embolectomy, subspecialty consultation, IV fluid resuscitation, vasopressor administration, supplemental oxygen, and ED length of stay (hours). A high-acuity intervention flag was defined as receipt of any of: thrombolysis, catheter-directed therapy, surgical embolectomy, or vasopressors.

2.6 Outcomes

Primary outcome. ED disposition, defined as discharge from the emergency department versus formal hospital admission (ward or ICU). ED observation stays not exceeding 24 hours without formal admission were classified as discharge. Disposition data were extracted from HBS transfer codes.

The primary outcome was reported as the guideline-discordant admission rate: the proportion of all sPESI = 0 patients formally admitted, with Wilson score 95% CI. Risk-marker-stratified admission rates were not computed because no patient met the definition of fully assessed with no additional risk markers (see Section 2.5).

Secondary outcomes. Thirty-day all-cause mortality (HBS death records; national population registry linkage sought where feasible); 7-day all-cause mortality; 30-day same-institution readmission; ICU transfer during index hospitalization; major bleeding (ISTH criteria); imaging-confirmed recurrent VTE within 30 days; and a 30-day composite adverse event (death, VTE recurrence, major bleeding, or ICU transfer). All secondary outcomes are restricted to same-institution records. Events at outside institutions are not captured; this limitation applies disproportionately to the discharge group and is expected to produce systematic underestimation of adverse events in that subgroup. All secondary comparisons are explicitly underpowered and designated as exploratory. ICU transfer was defined as transfer from a general ward to the intensive care unit during index hospitalization for admitted patients, or as ICU admission during any same-institution readmission within 30 days for discharged patients.

2.7 Sample Size

The primary analysis estimates a proportion (guideline-discordant admission rate). Sample size was determined by the joint requirements of precision, hypothesis testing power, and regression model stability. With an expected proportion of 0.50 (maximum variance assumption), a target margin of ± 6 percentage points, and 95% confidence, the required number of sPESI = 0 patients was 267. Given a published sPESI = 0 prevalence of approximately 45% among CTPA-confirmed PE cohorts, the total cohort target was 594 patients (267/0.45). Accounting for exclusion attrition during screening, the target screening pool was approximately 650 CTPA reports.

For the logistic regression (Research Question 2), the model contains 8 predictors contributing 8 regression parameters. With a minimum events-per-variable threshold of 10, a minimum of 80 admission events among sPESI = 0 patients was required, corresponding to approximately 237 sPESI = 0 patients at the observed admission rate. Model coefficient stability was verified by bootstrap internal validation (200 resamples).

For the primary hypothesis test (observed admission rate versus 30% reference): assuming a true rate of 38%, two-sided alpha = 0.05, and power 0.80, the minimum required sample was 243 sPESI = 0 patients (approximately 540 total). The precision requirement of 267 sPESI = 0 patients governs the final study size.

Outcome comparisons (Research Question 3) are designated as exploratory and are not formally powered; observed event rates and confidence interval widths will inform sample size estimation for future prospective studies.

2.8 Statistical Analysis

All analyses were performed in Python (version 3.12) using scipy (version 1.14), statsmodels (version 0.14), and scikit-learn (version 1.5). Statistical significance was set at two-sided $\alpha = 0.05$. The analysis plan was finalized before data analysis; any deviation is labeled post hoc.

Descriptive statistics. Normality was assessed by Shapiro-Wilk test supplemented by skewness evaluation. Continuous variables are presented as mean ± SD if approximately normal or median [IQR] otherwise. Categorical variables are presented as n (%). Between-group comparisons (admitted versus discharged in the sPESI = 0 subgroup) used independent-samples t-tests or Mann-Whitney U for continuous and chi-square or Fisher exact tests for categorical variables. Standardized mean differences (SMD) were calculated for all baseline comparisons; SMD > 0.10 was considered clinically meaningful imbalance.

Research Question 1: Guideline-discordant admission rate. The proportion of sPESI = 0 patients admitted was calculated with a Wilson score 95% CI. The primary hypothesis was tested using a one-sample proportion z-test (two-sided) against the reference value of 0.30.

Research Question 2: Admission determinants. A pre-specified logistic regression model was constructed with admission (1) versus discharge (0) as the dependent variable, restricted to sPESI = 0 patients. Predictors were selected a priori on clinical grounds, not through stepwise or data-driven procedures: age (continuous), sex, PE location (central versus peripheral), RV/LV > 1 (> 1 versus ≤ 1), imaging assessment incomplete (PE location not reported or RV/LV ratio not measured versus both assessed), troponin elevation (binary: elevated versus other), off-hours presentation, and bilateral PE. The “not reported” category for PE location and the “not measured” category for RV/LV ratio were nearly identical in the sPESI = 0 subgroup (77 of 78 patients overlapping), producing quasi-separation and variance inflation factors exceeding 90 when entered as separate indicators. These were therefore collapsed into a single “imaging assessment incomplete” variable, reducing the parameter count from 10 to 8 and eliminating the collinearity.

sPESI components were excluded from the model because they define the inclusion criterion (sPESI = 0) and carry no within-group variance. Three additional variables from the original study protocol were excluded before analysis: calendar year of presentation could not be recovered from de-identified dates (see Section 2.5); clinician position (attending versus resident) could not be reliably extracted from archival records for the full study period; and symptom onset-to-presentation time had 79.9% missingness in the sPESI = 0 subgroup, precluding meaningful inclusion. Symptom duration is reported descriptively in the available subset only.

Model calibration was assessed with a calibration plot of observed versus predicted admission probabilities in deciles, calibration slope and intercept via offset logistic regression, observed-to-expected (O/E) ratio with Byar 95% CI, and Brier score. The Hosmer-Lemeshow test was computed but interpreted with caution given its sensitivity to sample size and grouping strategy. Model discrimination was quantified with AUROC and 95% CI (DeLong method). Multicollinearity was assessed with variance inflation factors (threshold > 5). Model coefficient stability was evaluated by bootstrap resampling (200 iterations); predictors selected in fewer than 60% of bootstrap samples were flagged as unstable. Optimism-corrected AUROC was computed from the bootstrap distribution. Results are reported as adjusted odds ratios with 95% CI and Nagelkerke R-squared.

Research Question 3: Outcome comparison. Thirty-day adverse event rates were compared between admitted and discharged sPESI = 0 patients using chi-square or Fisher exact tests and reported as risk differences with Newcombe hybrid score 95% confidence intervals (Method 10; Newcombe RG, Stat Med 1998;17:873–890). (12) Proportions are reported with Clopper-Pearson exact 95% confidence intervals. Risk difference confidence intervals were calculated using the Newcombe hybrid score method, which avoids the non-feasible boundary values produced by the Wald method when one group contains zero events. Risk-marker-stratified comparisons were planned but could not be executed because no sPESI = 0 patient had all three additional risk markers measured and classified as normal (Section 2.5). Given the low expected event counts, all outcome comparisons are designated as exploratory; no causal inference is claimed, and results are interpreted as safety signals to inform sample size estimation for future prospective studies.

Multiple comparisons. The primary analysis (single proportion) requires no multiplicity correction. For secondary outcomes, Benjamini-Hochberg FDR correction was applied; both unadjusted and adjusted p-values are reported.

Missing data. Missing data proportions are reported per variable. For RV/LV ratio and PE anatomical location, three-category coding (Section 2.5) is the primary approach, with these two categories collapsed into a single imaging assessment indicator in the regression model (see above). For troponin, binary coding (elevated versus other) is used in the primary model; three-category coding is applied in sensitivity analysis. For variables with < 5% missingness, complete-case analysis was applied under MCAR assumption.

Pre-specified sensitivity analyses.

1. Troponin recoded as three-category (normal / elevated / not measured, reference = not measured) instead of binary, with documentation of coefficient stability and cell-specific confidence interval width for the 3-patient ‘normal’ category.
2. RV/LV and PE location three-category variables recoded as binary (unmeasured = normal, imaging incomplete indicator removed) to quantify the impact of the informative missingness assumption.
3. Complete-case restriction to patients with available troponin, RV/LV, and PE location data.

4. Stratification by ICU versus ward admission to separate high acuity from standard admissions.
5. Exclusion of patients who received thrombolysis, catheter-directed therapy, embolectomy, or vasopressors, whose admission was clinically indicated regardless of sPESI, to estimate the admission rate in the truly low-acuity subgroup.
6. Worst-case imputation for differential outcome ascertainment: assuming that 5% and 10% of discharged patients lost to external follow-up experienced an adverse event, the composite adverse event rate in the discharge group was recalculated and compared to the observed admitted-group rate.
7. Reclassification of the six discharged patients who received high-acuity interventions as admitted, to test the sensitivity of the primary admission rate and outcome comparison to the disposition coding rule (post hoc).

2.9 Reporting Compliance

This study was reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement. (13)

2.10 Ethical Considerations

This study was approved by the Istanbul Medipol University Ethics Committee (decision no: 34546, date: 04/2026). Given the retrospective design and access to patient data exclusively through anonymized archival records, the requirement for individual informed consent was waived. All data were processed using study-specific anonymous identification codes. The identity-code linkage table was used only during data collection and deleted before analysis. The analysis dataset contains no personally identifiable information.

RESULTS

Study Population and sPESI Classification

Systematic screening of the PACS radiology archive identified 731 CTPA reports with findings consistent with pulmonary embolism between January 2015 and April 2026. After applying exclusion criteria (incidental PE without corresponding symptoms, n = 32; massive PE with cardiac arrest or refractory hypotension, n = 47; chronic thromboembolic disease, n = 14; pregnancy, n = 7; insufficient documentation for sPESI calculation, n = 37), the final analytic cohort comprised 594 patients with CTPA-confirmed acute symptomatic pulmonary embolism (Fig. 1).

Systematic screening of the PACS radiology archive identified 731 CTPA reports with findings consistent with pulmonary embolism. After applying exclusion criteria, 594 patients formed the final analytic cohort. Retrospective sPESI classification identified 267 (44.9%) as low-risk (sPESI = 0) and 327 (55.1%) as non-low-risk (sPESI ≥ 1). Among sPESI = 0 patients, 102 (38.2%) were admitted to hospital (47 ward, 55 ICU) and 165 (61.8%) were discharged. Thirty-three sPESI = 0 patients received high-acuity interventions

(thrombolysis, catheter-directed therapy, or vasopressors); 27 of these were admitted and 6 were discharged. Thirty-day outcomes are shown for each disposition subgroup. CTPA, computed tomography pulmonary angiography; ICU, intensive care unit; PACS, Picture Archiving and Communication System; sPESI, simplified Pulmonary Embolism Severity Index; AE, adverse event.

Retrospective sPESI classification identified 267 patients (44.9%) as low-risk (sPESI = 0) and 327 (55.1%) as non-low-risk (sPESI ≥ 1). The median age was 62.0 years (IQR 50.0–72.0), and 277 (46.6%) were male. sPESI = 0 patients were younger (median 58.0 vs 64.0 years, $p < 0.001$), had higher systolic blood pressure (133.0 vs 129.0 mmHg, $p < 0.001$), and lower heart rates (88.0 vs 99.0 bpm, $p < 0.001$) than sPESI ≥ 1 patients (Table 1). Among sPESI = 0 patients, none had active cancer, heart failure, or chronic cardiopulmonary disease by definition of the score.

Table 1
Baseline characteristics of the full cohort by sPESI risk group.

Variable	Total (n = 594)	sPESI = 0 (n = 267)	sPESI ≥ 1 (n = 327)	p-value
Age, years	62.0 [50.0–72.0]	58.0 [48.0–69.0]	64.0 [53.0–76.0]	< 0.001
Male sex	277 (46.6)	129 (48.3)	148 (45.3)	0.458
sPESI components				
Age > 80	65 (10.9)	0 (0.0)	65 (19.9)	< 0.001
Active cancer	149 (25.1)	0 (0.0)	149 (45.6)	< 0.001
Chronic cardiopulmonary	139 (23.4)	0 (0.0)	139 (42.5)	< 0.001
Heart rate ≥ 110 bpm	111 (18.7)	0 (0.0)	111 (33.9)	< 0.001
SBP < 100 mmHg	24 (4.0)	0 (0.0)	24 (7.3)	< 0.001
SpO_2 <90%	7 (1.2)	0 (0.0)	7 (2.1)	0.018
Vital signs				
SBP, mmHg	131.0 [118.0–146.8]	133.0 [124.0–150.0]	129.0 [114.0–142.5]	< 0.001
Heart rate, bpm	92.0 [79.0–104.0]	88.0 [77.0–98.0]	99.0 [80.0–112.0]	< 0.001
SpO_2, %	98.0 [96.0–100.0]	98.0 [97.0–100.0]	97.0 [95.2–99.0]	< 0.001
Comorbidities				
CAD	89 (15.0)	26 (9.7)	63 (19.3)	0.001
CKD	70 (11.8)	26 (9.7)	44 (13.5)	0.162
Diabetes	119 (20.0)	45 (16.9)	74 (22.6)	0.080
Hypertension	311 (52.4)	125 (46.8)	186 (56.9)	0.015
Atrial fibrillation	33 (5.6)	8 (3.0)	25 (7.6)	0.014
Obesity	96 (16.2)	46 (17.2)	50 (15.3)	0.523
Imaging				
Bilateral PE	378 (63.6)	160 (59.9)	218 (66.7)	0.089
Pulmonary infarction	141 (23.7)	73 (27.3)	68 (20.8)	0.062
Pleural effusion	365 (61.4)	155 (58.1)	210 (64.2)	0.124
Disposition				

Variable	Total (n = 594)	sPESI = 0 (n = 267)	sPESI ≥ 1 (n = 327)	p-value
Hospital admission	298 (50.2)	102 (38.2)	196 (59.9)	< 0.001
30-day mortality	54 (9.1)	5 (1.9)	49 (15.0)	< 0.001
30-day composite AE	77 (13.0)	14 (5.2)	63 (19.3)	< 0.001

Continuous variables: median [IQR]; categorical variables: n (%). p-values: Mann-Whitney U test for continuous, chi-square or Fisher exact test for categorical variables.

Characteristics of the sPESI = 0 Subgroup

Among the 267 sPESI = 0 patients, 102 (38.2%) were formally admitted to hospital (47 to a ward and 55 to the intensive care unit) and 165 (61.8%) were discharged from the emergency department (Table 2). Admitted patients were older (median 61.0 vs 57.0 years, p = 0.032; SMD 0.26), more likely to have diabetes (23.5% vs 12.7%, p = 0.022; SMD 0.28), coronary artery disease (15.7% vs 6.1%, p = 0.010; SMD 0.31), and atrial fibrillation (6.9% vs 0.6%, p = 0.006; SMD 0.33). Admitted patients were also more likely to have received intravenous fluid resuscitation (53.9% vs 15.8%, p < 0.001; SMD 0.80) and high-acuity interventions including thrombolysis, catheter-directed therapy, or vasopressors (26.5% vs 3.6%, p < 0.001; SMD 0.64). Six of the 33 patients who received high-acuity interventions were classified as discharged under the study disposition coding rule (ED observation not exceeding 24 hours without formal hospital admission). Of these six, 3 received systemic thrombolysis and 3 received catheter-directed therapy; none received vasopressors. Their median ED length of stay was 6.4 hours (IQR 4.7–8.8). None experienced an adverse event within 30 days. These patients were retained in the discharge group for the primary analysis; a post hoc sensitivity analysis reclassifying them as admitted is reported in SA-vii. Troponin elevation was more common in admitted patients (19.6% vs 9.7%, p = 0.021; SMD 0.28), as was syncope at presentation (22.5% vs 12.7%, p = 0.036; SMD 0.26).

PE anatomical location did not differ between groups (p = 0.969), nor did RV/LV ratio distribution (p = 0.212). ED length of stay was similar (median 6.5 vs 7.2 hours, p = 0.086). Among admitted patients, the median hospital length of stay was 4.9 days (IQR 1.9–8.8).

Table 2
Characteristics of sPESI = 0 patients by disposition.

Variable	Total (n = 267)	Admitted (n = 102)	Discharged (n = 165)	p	SMD
Age, years	58 [48–69]	61 [49–71]	57 [47–67]	0.032	0.26
Male sex	129 (48.3)	46 (45.1)	83 (50.3)	0.408	0.10
Off-hours	193 (72.3)	71 (69.6)	122 (73.9)	0.442	0.10
Troponin elevated	36 (13.5)	20 (19.6)	16 (9.7)	0.021	0.28
Echo performed	132 (49.4)	66 (64.7)	66 (40.0)	< 0.001	0.49
Echo RV dysfunction	30 (11.2)	15 (14.7)	15 (9.1)	0.158	0.17
Syncope	44 (16.5)	23 (22.5)	21 (12.7)	0.036	0.26
IV fluid resuscitation	81 (30.3)	55 (53.9)	26 (15.8)	< 0.001	0.80
High-acuity intervention	33 (12.4)	27 (26.5)	6 (3.6)	< 0.001	0.64
Subspecialty consult	258 (96.6)	95 (93.1)	163 (98.8)	0.029	0.29
Diabetes	45 (16.9)	24 (23.5)	21 (12.7)	0.022	0.28
CAD	26 (9.7)	16 (15.7)	10 (6.1)	0.010	0.31
Atrial fibrillation	8 (3.0)	7 (6.9)	1 (0.6)	0.006	0.33
Hypertension	125 (46.8)	55 (53.9)	70 (42.4)	0.067	0.23
Bilateral PE	160 (59.9)	64 (62.7)	96 (58.2)	0.460	0.09
Pleural effusion	155 (58.1)	65 (63.7)	90 (54.5)	0.140	0.19
PE location					
Central	133 (49.8)	51 (50.0)	82 (49.7)		
Peripheral	56 (21.0)	22 (21.6)	34 (20.6)		
Not reported	78 (29.2)	29 (28.4)	49 (29.7)	0.969	
RV/LV ratio					
≤1	68 (25.5)	32 (31.4)	36 (21.8)		
>1	122 (45.7)	42 (41.2)	80 (48.5)		
Not measured	77 (28.8)	28 (27.5)	49 (29.7)	0.212	
ED LOS, hours	7.1 [4.8–9.3]	6.5 [4.0–9.2]	7.2 [5.3–9.6]	0.086	0.02

Continuous variables: median [IQR]; categorical variables: n (%). SMD: standardized mean difference (> 0.10 considered clinically meaningful). p-values: Mann-Whitney U or Fisher exact/chi-square.

Research Question 1: Guideline-Discordant Admission Rate

Among 267 sPESI = 0 patients, 102 (38.2%; Wilson 95% CI 32.6–44.2%) were formally admitted to hospital. This proportion was significantly higher than the 30% reference threshold (z = 2.758, p = 0.006). Of the 102 admitted patients, 47 (46.1%) were admitted to a general ward and 55 (53.9%) to the intensive care unit.

Risk marker assessment was incomplete in the majority of patients. Troponin was measured in only 39 of 267 patients (14.6%), of whom 36 had elevated values and only 3 had normal results. RV/LV ratio was assessed in 190 patients (71.2%), and PE anatomical location was reported in 189 (70.8%). All three risk markers were assessed in only 25 patients (9.4%), and no patient had all three markers measured and classified as normal. (Supplementary Figure S1)

Research Question 2: Factors Associated with Hospital Admission

The pre-specified logistic regression model (Table 3) included eight predictors selected a priori (calendar year was excluded because dates were de-identified and could not be recovered). With 102 admission events and 8 predictors, the events-per-variable ratio was 12.8. All variance inflation factors were below 5 (range 1.01–2.63), indicating no problematic multicollinearity.

No predictor reached conventional statistical significance at $p < 0.05$. Troponin elevation showed the strongest association with admission (aOR 2.03, 95% CI 0.95–4.31, p = 0.066). RV/LV ratio > 1 showed a non-significant inverse association (aOR 0.62, 95% CI 0.34–1.15, p = 0.128). The apparent AUROC was 0.616 (95% CI 0.547–0.684). After bootstrap internal validation (200 resamples), the optimism-corrected AUROC was 0.555. No predictor was selected in more than 60% of bootstrap samples, confirming global coefficient instability.

The Brier score was 0.226, the calibration slope was 1.00, the calibration intercept was 0.00, and the observed-to-expected ratio was 1.00 (95% CI 0.82–1.21). The Hosmer-Lemeshow test was non-significant (χ^2=2.11, df = 8, p = 0.977). These calibration metrics represent apparent performance on the derivation sample. Figure 2 shows the forest plot of adjusted odds ratios; Fig. 3 shows the ROC curve; Fig. 4 shows the calibration plot.

Table 3
Logistic regression model: factors associated with hospital admission in sPESI = 0 patients.

Variable	aOR	95% CI	p	Bootstrap selection %
Age (per year)	1.01	1.00–1.03	0.116	31.5
Male sex	0.78	0.47–1.30	0.335	13.5
Central PE location	0.88	0.45–1.72	0.711	8.0
RV/LV ratio > 1	0.62	0.34–1.15	0.128	40.0
Imaging assessment incomplete	0.65	0.27–1.59	0.346	15.0
Troponin elevated	2.03	0.95–4.31	0.066	44.5
Off-hours presentation	0.83	0.47–1.45	0.508	10.5
Bilateral PE	1.13	0.58–2.19	0.722	6.0

aOR: adjusted odds ratio. Bootstrap selection %: proportion of 200 bootstrap resamples in which the variable was significant at p < 0.05. All variables selected in < 60% of bootstrap resamples. Nagelkerke R^2=0.059. AUROC = 0.616, optimism-corrected AUROC = 0.555.

Adjusted odds ratios (aOR) with 95% confidence intervals from the pre-specified logistic regression model (n = 267). The dashed vertical line indicates aOR = 1.0 (no association). No predictor reached statistical significance at p < 0.05. Bootstrap selection percentage indicates the proportion of 200 bootstrap resamples in which each variable achieved p < 0.05; all values were below 60%, confirming global coefficient instability. Model apparent AUROC = 0.616; optimism-corrected AUROC = 0.555. CI, confidence interval; PE, pulmonary embolism; RV/LV, right ventricular to left ventricular diameter ratio.

The solid line represents the apparent ROC curve (AUROC = 0.616, 95% CI 0.547–0.684). The dashed diagonal line represents the reference line of no discrimination (AUROC = 0.500). After bootstrap internal validation (200 resamples), the optimism-corrected AUROC was 0.555, indicating that virtually all apparent discriminative ability reflects overfitting. AUROC, area under the receiver operating characteristic curve; CI, confidence interval.

Observed admission proportions (y-axis) versus mean predicted probabilities (x-axis) across deciles of predicted risk. The dashed diagonal line represents perfect calibration. Error bars indicate 95% confidence intervals for observed proportions within each decile. Calibration slope = 1.00, calibration intercept = 0.00, Brier score = 0.226, observed-to-expected ratio = 1.00 (95% CI 0.82–1.21). These metrics reflect apparent performance on the derivation sample.

Research Question 3: 30-Day Outcomes

Thirty-day outcomes are presented in Table 4. Among sPESI = 0 patients, 5 deaths occurred within 30 days (1.9%, 95% CI 0.6–4.3%): 4 in admitted and 1 in discharged patients. The 30-day composite adverse

event rate was 7.8% (95% CI 3.4–14.9%) in admitted and 3.6% (95% CI 1.3–7.7%) in discharged patients (risk difference + 4.2 percentage points, 95% CI − 1.4 to + 11.4, FDR-adjusted p = 0.313). No outcome comparison reached statistical significance after Benjamini-Hochberg correction.

All five 30-day deaths occurred in patients who were in an intensive care unit at the time of death; four were in patients whose index disposition was hospital admission, and one was in a patient originally discharged from the ED who was subsequently readmitted to the ICU within 30 days. Three of the five had received high-acuity interventions (thrombolysis, catheter-directed therapy, or vasopressors). Death causes included sepsis-related complications (n = 2), defibrination syndrome (n = 1), pulmonary fibrosis (n = 1), and a trochanteric fracture complication (n = 1, discharged patient). These outcome comparisons are designated as exploratory (see Section 2.8). Adverse event ascertainment is restricted to same-institution records.

Table 4
30-day outcomes in sPESI = 0 patients by disposition.

Outcome	Admitted (n = 102)	Discharged (n = 165)	RD (95% CI)	FDR p
7-day mortality	1 (1.0%)	0 (0.0%)	+ 1.0 (− 1.4 to + 5.3)	0.669
30-day mortality	4 (3.9%)	1 (0.6%)	+ 3.3 (− 0.3 to + 9.1)	0.313
30-day readmission	15 (14.7%)	14 (8.5%)	+ 6.2 (− 1.4 to + 15.0)	0.313
Recurrent VTE	1 (1.0%)	3 (1.8%)	−0.8 (− 4.3 to + 3.7)	1.000
Major bleeding	1 (1.0%)	1 (0.6%)	+ 0.4 (− 2.5 to + 4.8)	1.000
ICU transfer	2 (2.0%)	2 (1.2%)	+ 0.7 (− 2.7 to + 5.7)	0.893
Composite adverse event	8 (7.8%)	6 (3.6%)	+ 4.2 (− 1.4 to + 11.4)	0.313

RD: risk difference (admitted minus discharged). 95% CI: Newcombe hybrid score method. FDR p: Benjamini-Hochberg false discovery rate adjusted p-value. All comparisons are exploratory and underpowered. The composite counts unique patients; one discharged patient experienced two qualifying events.

Sensitivity Analyses

Exclusion of high-acuity intervention patients (SA-v). After excluding 33 patients who received thrombolysis, catheter-directed therapy, or vasopressors, 234 patients remained. The admission rate

decreased to 32.1% (75/234; Wilson 95% CI 26.4–38.3%) and was no longer significantly different from 30% (z = 0.672, p = 0.501). Among these 75 admitted patients, 43 were admitted to a ward and 32 to the ICU.

Troponin three-category model (SA-i). Replacing binary troponin coding with three categories (normal, elevated, not measured; reference = not measured) did not materially change model performance (AUROC = 0.618). The normal troponin category (n = 3) produced an unstable coefficient (OR 3.42, 95% CI 0.29–40.47, p = 0.329). Troponin elevated OR remained similar (2.09, 95% CI 0.98–4.45, p = 0.056).

Binary imaging coding (SA-ii). Recoding unmeasured RV/LV and unreported PE location as normal (removing the imaging incomplete indicator) yielded similar results (AUROC = 0.612). No predictor reached significance.

Complete case analysis (SA-iii). Only 25 patients (9.4%) had all three risk markers assessed. Among these, 15 (60.0%) were admitted. The sample was too small for meaningful regression analysis.

ICU versus ward stratification (SA-iv). Of 55 ICU admissions, 23 (41.8%) received high-acuity interventions. Of 47 ward admissions, 4 (8.5%) received high-acuity interventions. The 30-day mortality rate was 7.3% (4/55) for ICU, 0.0% (0/47) for ward, and 0.6% (1/165) for discharged patients. The 30-day composite adverse event rate was 10.9% (6/55) for ICU, 4.3% (2/47) for ward, and 3.6% (6/165) for discharged patients.

Worst-case imputation (SA-vi). Imputing adverse events for 5% of discharged patients lost to external follow-up raised the discharge composite adverse event rate to 9.1% (15/165), eliminating the observed difference (p = 0.724). At 10% imputation, the discharge rate (13.9%) exceeded the admitted rate (7.8%, p = 0.131), reversing the apparent direction of effect.

Reclassification of discharged high-acuity patients (SA-vii, post hoc). Six discharged patients who received thrombolysis, catheter-directed therapy, or vasopressors were reclassified as admitted. This increased the admitted group to 108 and reduced the discharged group to 159. The guideline-discordant admission rate increased to 40.4% (108/267; Wilson 95% CI 34.7–46.4%). None of the six reclassified patients experienced an adverse event within 30 days. The 30-day composite adverse event rate was 7.4% in the reclassified admitted group versus 3.8% in the reclassified discharged group (risk difference + 3.6 percentage points; 95% CI -1.9 to + 10.5; Fisher exact p = 0.263).

DISCUSSION

This study examined the disposition of sPESI = 0 patients with CTPA-confirmed acute pulmonary embolism at a single-center tertiary emergency department and produced three main findings. The proportion of sPESI = 0 patients formally admitted to hospital was 38.2% (95% CI 32.6–44.2%), which exceeded the 30% reference value representing the maximum admission rate consistent with majority outpatient management. A pre-specified logistic regression model incorporating clinical and imaging

variables yielded an optimism-corrected AUROC of 0.555, with no predictor reaching statistical significance and no variable selected in more than 60% of bootstrap resamples, indicating that the admission decision was not systematically explained by the measured variables. Thirty-day adverse event rates did not differ between admitted and discharged patients after multiple comparison correction, though these comparisons were underpowered and subject to ascertainment bias.

The 38.2% admission rate falls toward the lower end of published international estimates for sPESI = 0 patients. In the original sPESI derivation cohort, Jimenez et al. did not report disposition patterns, but subsequent studies have documented that the majority of sPESI-classified low-risk patients are hospitalized regardless of score result.(4) In a US Veterans Health Administration inpatient cohort, Wells et al. reported that 28.4% of hospitalized PE patients were classified as low-risk by sPESI, with lower complication rates and shorter lengths of stay than high-risk patients.(14) Salazar et al. documented that early discharge within 24 hours occurred in only 20.9% of fully risk-stratified low-risk PE patients at a Colombian tertiary center, with barriers including echocardiography delays, anticoagulant access, and pain management.(11) Aujesky et al. have noted that both the sPESI and outpatient management remain underutilized despite guideline recommendations dating to 2014.(15) Recent US data reinforce this pattern: Watson et al. reported that only 33.1% of sPESI low-risk patients were discharged from emergency departments in a nationally representative sample (2012–2020),(16) and Kabrhel et al. found that even after implementation of a structured outpatient protocol at two academic centers, 72% of low-risk PE patients remained hospitalized.(17) In this context, the present study's admission rate of 38.2% falls substantially below these US benchmarks.

The present study extends this literature by decomposing the observed admission rate. After excluding 33 patients who received high-acuity interventions (thrombolysis, catheter-directed therapy, or vasopressors), the admission rate fell to 32.1% ($p = 0.501$ versus 30%), indicating that a substantial portion of the excess admissions involved patients in whom sPESI failed to capture clinical severity and clinicians appropriately overrode the score. The remaining admission rate among the truly low-acuity subgroup did not differ from international benchmarks, suggesting that the degree of guideline discordance at this institution is less pronounced than the headline figure implies.

The null regression result is the most analytically informative finding of this study. The admission decision for sPESI = 0 patients was not systematically driven by the clinical and imaging variables that current guidelines recommend for further risk stratification. (2, 5) Two non-exclusive explanations merit consideration. The decision may have been driven by unmeasured factors including clinical gestalt, patient preference, bed availability, medicolegal environment, or institutional culture, none of which are captured in retrospective chart data. Westafer et al. identified clinician beliefs about consequences, fear of adverse outcomes, and institutional inertia as dominant determinants of PE disposition in a qualitative study of 26 emergency physicians across 11 hospital systems, with external barriers such as follow-up availability and medicolegal climate serving as universal but individually moderated determinants. (18) Alternatively, the decision may have been non-systematic. The 85.4% troponin non-measurement rate among sPESI = 0 patients at this institution means that the biomarker step of the

ESC-recommended risk stratification algorithm was not executed for the vast majority of patients. Without standardized assessment, disposition decisions may have varied inconsistently across clinicians and shifts. That off-hours presentation, which characterized 72.3% of sPESI = 0 encounters, showed no association with admission (aOR 0.83, p = 0.508) is consistent with this interpretation, as it indicates that staffing context did not systematically influence the decision. The inverse association between RV/LV ratio > 1 and admission (aOR 0.62, 95% CI 0.34–1.15) is counterintuitive given the prognostic significance of right ventricular dilation in pulmonary embolism. Cross-tabulation of the RV/LV subgroups revealed that patients with RV/LV ≤ 1 had higher rates of diabetes, hypertension, intravenous fluid resuscitation, and high-acuity interventions than patients with RV/LV > 1, indicating that this subgroup carried a greater non-PE-specific illness burden. The apparent inverse association therefore reflects confounding by comorbidity and acute clinical status rather than a clinically meaningful relationship between right ventricular dilation and discharge. This pattern provides additional evidence that the admission decision at this institution was driven by the overall clinical picture rather than by PE-specific risk markers as recommended by current guidelines.

The 14.6% troponin measurement rate among sPESI = 0 patients warrants separate consideration as a quality-of-care finding. The 2019 ESC guidelines explicitly recommend cardiac troponin measurement as part of the risk stratification algorithm for all hemodynamically stable PE patients, and the 2026 AHA/ACC multisociety guideline incorporates biomarker assessment into its clinical severity categorization.(2, 5) Among the 39 sPESI = 0 patients who did undergo troponin testing, 36 (92.3%) had elevated values, suggesting that the test was ordered selectively when clinical suspicion was already high rather than routinely as a screening tool. This pattern of selective rather than systematic biomarker assessment has been identified as a barrier to implementing guideline-concordant PE management at other institutions.(18, 19) Karolak et al. demonstrated in a prospective cohort of 409 sPESI = 0 patients that troponin retained independent prognostic value for clinical deterioration, with patients whose troponin did not exceed 1.7 times the upper limit of normal constituting a very low-risk subgroup suitable for outpatient treatment.(8) Because systematic troponin measurement was not performed at the present institution, this very low-risk subgroup could not be identified, and the planned risk-marker-stratified outcome analyses could not be executed. The most immediate clinical implication of this study may therefore concern standardized biomarker-inclusive assessment rather than disposition decision-making itself.

The existence of 33 sPESI = 0 patients (12.4%) who received thrombolysis, catheter-directed therapy, or vasopressors exposes a sensitivity limitation of the sPESI score that has been described in other contexts.(3, 9) These patients had none of the six sPESI adverse features yet required ICU-level care; their severity was driven by factors outside the sPESI construct, including imaging findings, biomarker elevation, syncope, or clinical trajectory during emergency department evaluation. Barco et al. demonstrated in a systematic review and meta-analysis of 3,295 low-risk PE patients that the presence of right ventricular dysfunction on imaging or elevated troponin was associated with significantly higher early mortality (OR 4.19 and 6.25, respectively) even among patients classified as low risk by clinical scores.(20) O'Hare et al. reported that concerning CT findings (RV enlargement, bilateral central emboli,

septal abnormality) in PESI low-risk patients were associated with increased hospitalization and resource utilization but not short-term adverse outcomes, indicating that clinicians respond to imaging signals not captured by clinical scores even when those signals may not translate into measurable harm. (21) In the present study, clinicians appeared to have recognized high-acuity patients despite the sPESI = 0 classification, as 27 of the 33 were admitted (23 to the ICU). This represents appropriate clinical override of a score rather than guideline non-adherence; the 2019 ESC and 2026 AHA/ACC guidelines both specify that sPESI alone is insufficient for disposition and that additional assessment including cardiac biomarkers and right ventricular evaluation is required before discharge can be considered. (2, 5)

The 30-day outcome comparisons in this study are subject to three interpretive threats that preclude causal inference. Confounding by indication is the primary concern: admitted patients were systematically sicker across multiple dimensions (IV fluid resuscitation SMD 0.80, high-acuity interventions SMD 0.64, troponin elevation SMD 0.28), and the higher adverse event rate in admitted patients reflects baseline severity rather than a harmful effect of hospitalization. The ICU-versus-ward stratification supports this interpretation; all five 30-day deaths occurred in ICU patients, whereas ward patient outcomes (0% mortality, 4.3% composite adverse event rate) were similar to discharge outcomes (0.6% mortality, 3.6% composite adverse event rate). Ascertainment bias is the second threat: adverse events occurring at outside institutions after discharge were not captured, and the worst-case sensitivity analysis demonstrated that imputing adverse events for only 5% of discharged patients lost to follow-up reversed the direction of the observed difference (discharge composite adverse event rate 9.1% versus admitted 7.8%). At 10% imputation, the discharge rate substantially exceeded the admitted rate (13.9% versus 7.8%), indicating that the apparently favorable outcome profile in the discharge group may be an artifact of incomplete follow-up. The third threat is insufficient statistical power: the study was powered to detect a risk difference of approximately 12 percentage points, whereas the observed differences ranged from 1 to 6 percentage points. The non-significant comparisons do not constitute evidence of equivalent outcomes.

The single-center retrospective design introduces several limitations that constrain the interpretation and generalizability of these findings. Confounding by indication, as discussed, prevents any causal inference from the outcome comparisons; admitted and discharged patients differed on unmeasured severity characteristics that cannot be adjusted for in a retrospective analysis. Ascertainment bias for the discharge group means that adverse events at outside institutions were not captured, and the sensitivity of the composite adverse event comparison to even modest imputation rates (5%) suggests that the observed between-group difference may not reflect the true difference. The retrospective sPESI calculation means that clinicians did not use the score for decision-making during the study period; applying the label "guideline-discordant" to their admission decisions therefore assumes that sPESI = 0 patients should have been considered for outpatient management, when the clinical context at presentation may have included information not captured in the dataset. Six patients who received thrombolysis, catheter-directed therapy, or vasopressors during their ED stay were classified as discharged because they did not undergo formal hospital admission within the study's coding framework. The inclusion of these high-acuity patients in the discharge group may attenuate the

observed difference in adverse event rates between groups and may underestimate the true guideline-discordant admission rate. Post hoc reclassification of these six patients as admitted (SA-vii) did not materially change the primary or secondary outcomes, as none experienced an adverse event within 30 days. Date de-identification prevented recovery of calendar years, precluding assessment of temporal trends across the 2015–2026 study period; changes in PE management including the adoption of Hestia criteria, effects of the COVID-19 pandemic on hospital capacity, and the publication of the 2026 AHA/ACC guideline could not be evaluated. Consequently, the pooled admission rate of 38.2% represents an 11-year average that may mask substantial temporal variation; if admission rates declined over the study period as outpatient management gained acceptance, the current institutional practice rate may already be at or below the 30% reference value. The 14.6% troponin measurement rate limited the ability to identify fully risk-stratified subgroups, and the regression model estimated associations using variables that were themselves often unavailable to the clinicians making the disposition decision. Finally, the planned risk-marker-stratified outcome comparison, pre-specified in the protocol, could not be executed because no sPESI = 0 patient had all three additional risk markers (troponin, RV/LV ratio, PE location) measured and classified as normal, a finding that is itself informative about the completeness of risk assessment at this institution.

These findings apply most directly to tertiary emergency departments with similar patient selection, consultation patterns, and diagnostic workflows. The most actionable recommendation from this study concerns the implementation of a standardized troponin-inclusive risk assessment protocol for all hemodynamically stable PE patients, which would enable identification of the very low-risk subgroup described by Karolak et al. and align institutional practice with ESC and AHA/ACC recommendations. A prospective multicenter study targeting the ward-admitted sPESI = 0 subgroup, whose outcomes were indistinguishable from those of discharged patients in the present analysis, could determine whether a structured assessment protocol safely redirects these patients to outpatient management. The observed event rates (composite adverse event 3.6–7.8%) and confidence interval widths from this study can inform sample size estimation for such a trial.

CONCLUSION

In this single-center retrospective cohort of 267 sPESI = 0 patients with CTPA-confirmed acute pulmonary embolism, the observed guideline-discordant admission rate of 38.2% was partly attributable to patients who received high-acuity interventions despite a low sPESI classification; after their exclusion, the admission rate did not differ from published international benchmarks. The admission decision was not systematically explained by the clinical and imaging variables recommended by current guidelines for further risk stratification, and troponin was measured in only 14.6% of this population, precluding identification of a fully assessed very low-risk subgroup. These findings suggest that implementation of a standardized troponin-inclusive risk assessment protocol for hemodynamically stable PE patients may warrant evaluation as an initial step toward guideline-concordant disposition at this institution. Prospective multicenter validation targeting the ward-admitted sPESI = 0 subgroup,

whose outcomes were similar to those of discharged patients, is required to determine whether structured risk assessment can safely reduce hospitalization rates.

Abbreviations

AE
adverse event
aOR
adjusted odds ratio
AUROC
area under the receiver operating characteristic curve
BMI
body mass index
BNP
brain natriuretic peptide
CAD
coronary artery disease
CDT
catheter-directed therapy
CI
confidence interval
CKD
chronic kidney disease
CKD-EPI
Chronic Kidney Disease Epidemiology Collaboration
COPD
chronic obstructive pulmonary disease
CTPA
computed tomography pulmonary angiography
ED
emergency department
eGFR
estimated glomerular filtration rate
ESC
European Society of Cardiology
FDR
false discovery rate
FEU
fibrinogen equivalent units
GCS

Glasgow Coma Scale
HBS
hospital information system
HR
heart rate
ICD-10
International Classification of Diseases,10th Revision
ICU
intensive care unit
INR
international normalized ratio
IQR
interquartile range
ISTH
International Society on Thrombosis and Haemostasis
IV
intravenous
LV
left ventricular
NT-proBNP
N-terminal pro-brain natriuretic peptide
O/E
observed-to-expected
PACS
Picture Archiving and Communication System
PASP
pulmonary artery systolic pressure
PE
pulmonary embolism
RD
risk difference
ROC
receiver operating characteristic
RV
right ventricular
RV/LV
right ventricular to left ventricular diameter ratio
SA
sensitivity analysis
SBP

systolic blood pressure
SD
standard deviation
SMD
standardized mean difference
SpO2
arterial oxygen saturation by pulse oximetry
sPESI
simplified Pulmonary Embolism Severity Index
STROBE
Strengthening the Reporting of Observational Studies in Epidemiology
TAPSE
tricuspid annular plane systolic excursion
TIA
transient ischemic attack
VIF
variance inflation factor
VTE
venous thromboembolism
WBC
white blood cell count.

Declarations

Ethics Approval and Consent to Participate

The study was approved by the Istanbul Medipol University Non-Interventional Clinical Research Ethics Committee (approval number: E-10840098-202.3.02-473; decision number: 108). Individual patient consent was waived in accordance with the retrospective design.

Competing Interests:

The authors declare that they have no competing interests.

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Data Availability

The data that support the findings of this study are not publicly available due to patient privacy constraints and institutional ethics committee restrictions. De-identified aggregate data and the analysis

code are available from the corresponding author upon reasonable request, subject to approval by the Istanbul Medipol University Non-Interventional Clinical Research Ethics Committee.

Author Contributions

S.Z.E.K.: Conceptualization, Methodology, Formal analysis, Investigation, Resources, Writing – original draft, Writing – review & editing, Validation, Project administration.

U.D.: Methodology, Software, Investigation, Writing – review & editing, Validation, Visualization

M.S.I.: Data curation, Resources

S.B.: Review & editing, Supervision.

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Figures

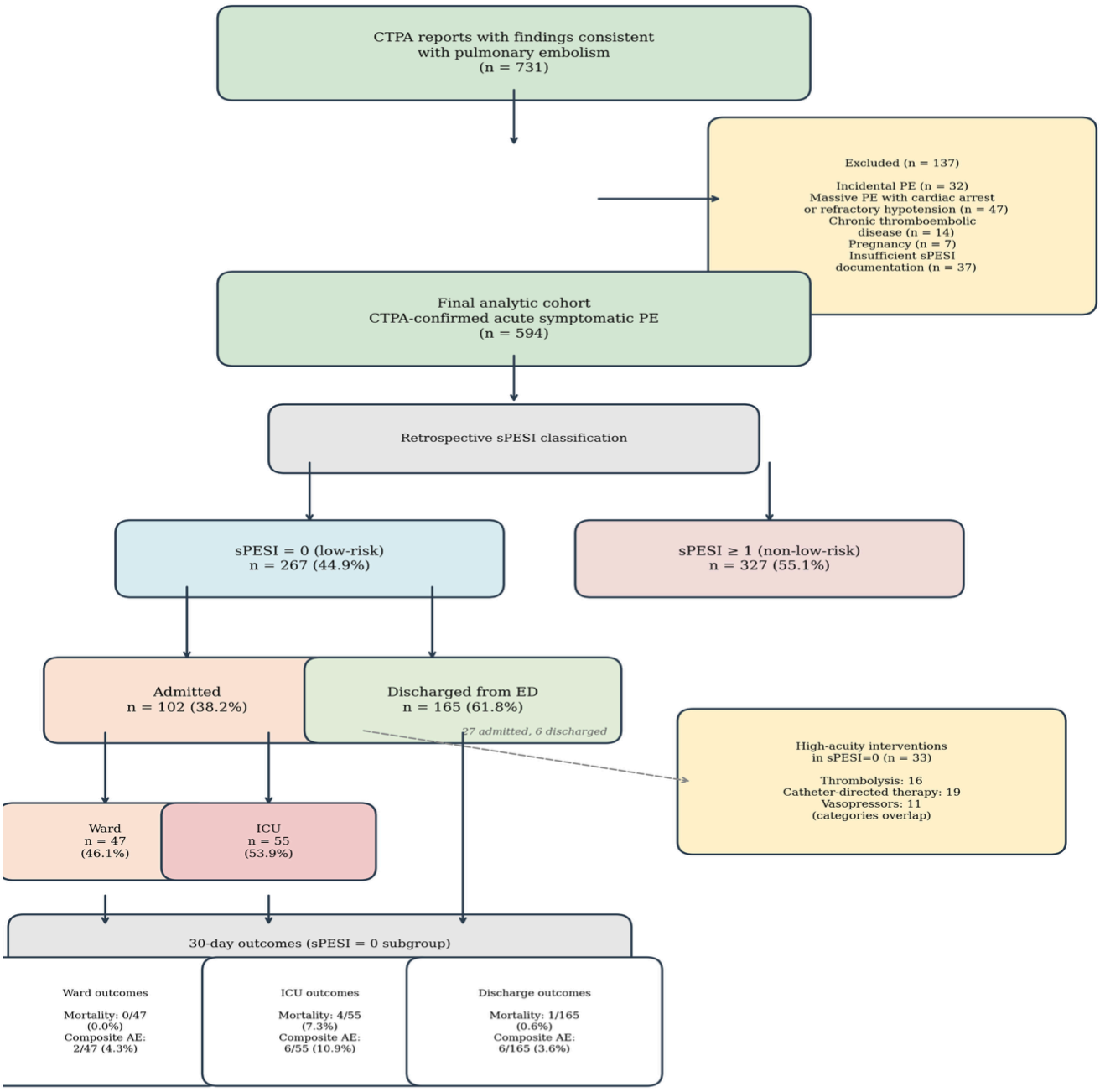


Figure 1

Patient flow diagram.

Systematic screening of the PACS radiology archive identified 731 CTPA reports with findings consistent with pulmonary embolism. After applying exclusion criteria, 594 patients formed the final analytic cohort. Retrospective sPESI classification identified 267 (44.9%) as low-risk (sPESI = 0) and 327 (55.1%) as non-

low-risk (sPESI ≥ 1). Among sPESI = 0 patients, 102 (38.2%) were admitted to hospital (47 ward, 55 ICU) and 165 (61.8%) were discharged. Thirty-three sPESI = 0 patients received high-acuity interventions (thrombolysis, catheter-directed therapy, or vasopressors); 27 of these were admitted and 6 were discharged. Thirty-day outcomes are shown for each disposition subgroup. CTPA, computed tomography pulmonary angiography; ICU, intensive care unit; PACS, Picture Archiving and Communication System; sPESI, simplified Pulmonary Embolism Severity Index; AE, adverse event.

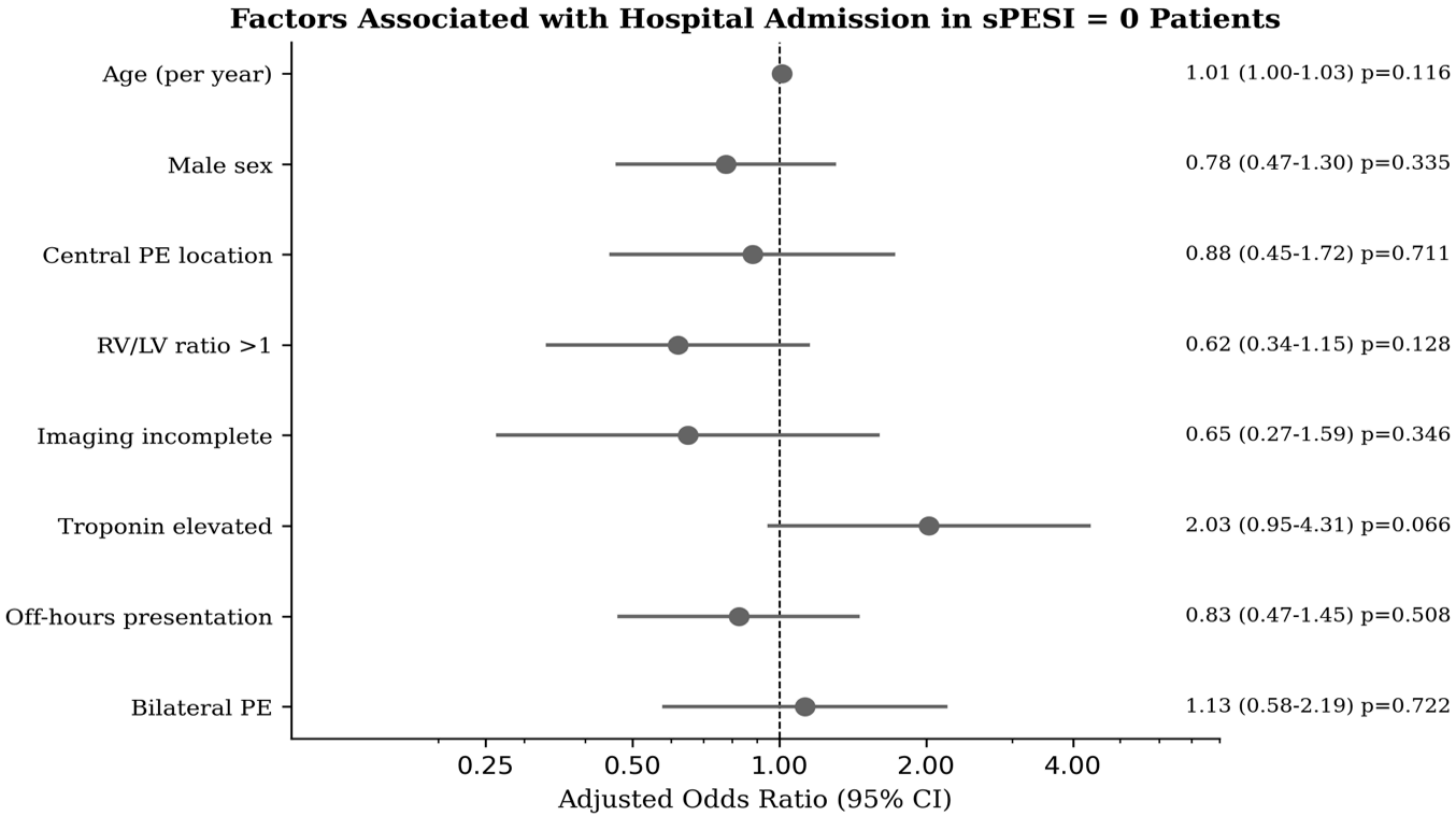


Figure 2

Forest plot of adjusted odds ratios for factors associated with hospital admission in sPESI = 0 patients.

Adjusted odds ratios (aOR) with 95% confidence intervals from the pre-specified logistic regression model (n = 267). The dashed vertical line indicates aOR = 1.0 (no association). No predictor reached statistical significance at p < 0.05. Bootstrap selection percentage indicates the proportion of 200 bootstrap resamples in which each variable achieved p < 0.05; all values were below 60%, confirming global coefficient instability. Model apparent AUROC = 0.616; optimism-corrected AUROC = 0.555. CI, confidence interval; PE, pulmonary embolism; RV/LV, right ventricular to left ventricular diameter ratio.

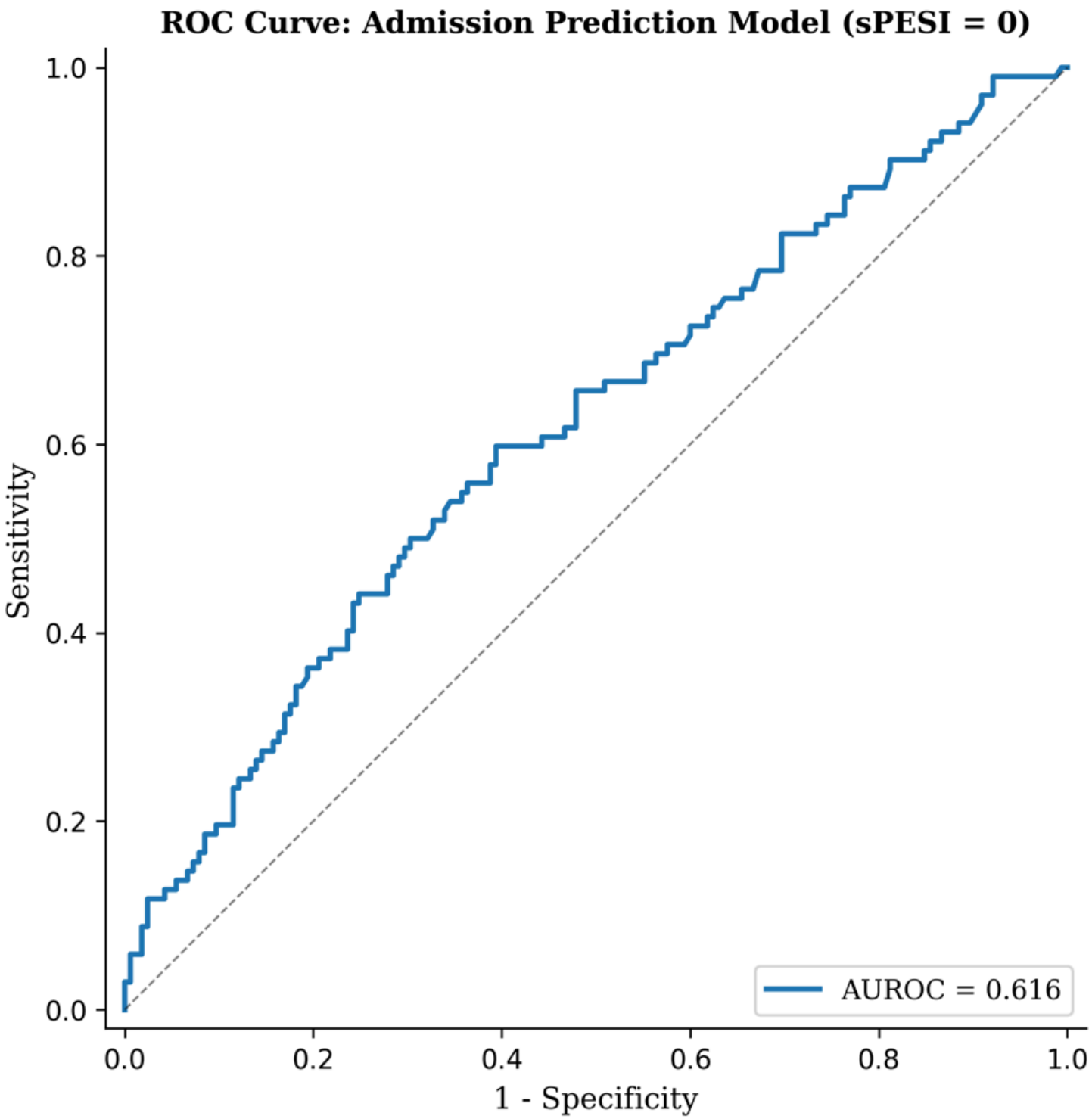


Figure 3

Receiver operating characteristic curve for the logistic regression model predicting hospital admission in sPESI = 0 patients.

The solid line represents the apparent ROC curve (AUROC = 0.616, 95% CI 0.547-0.684). The dashed diagonal line represents the reference line of no discrimination (AUROC = 0.500). After bootstrap internal validation (200 resamples), the optimism-corrected AUROC was 0.555, indicating that virtually all apparent

discriminative ability reflects overfitting. AUROC, area under the receiver operating characteristic curve; CI, confidence interval.

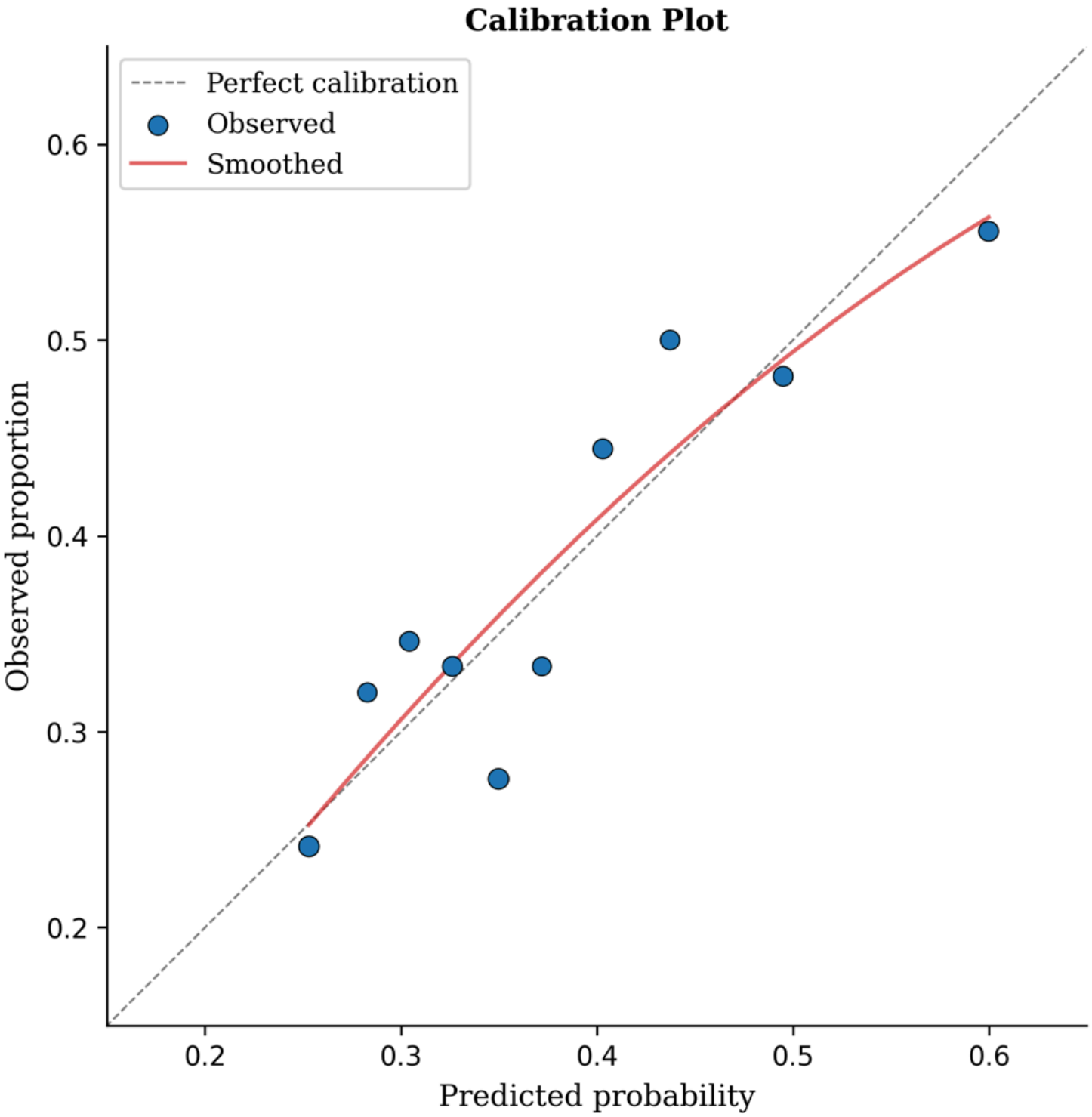


Figure 4

Calibration plot of the logistic regression model for hospital admission in sPESI = 0 patients.

Observed admission proportions (y-axis) versus mean predicted probabilities (x-axis) across deciles of predicted risk. The dashed diagonal line represents perfect calibration. Error bars indicate 95% confidence intervals for observed proportions within each decile. Calibration slope = 1.00, calibration intercept = 0.00, Brier score = 0.226, observed-to-expected ratio = 1.00 (95% CI 0.82-1.21). These metrics reflect apparent performance on the derivation sample.