Original Paper

ABO Blood Group and Clinical Outcomes in Acute Pulmonary Embolism: A Retrospective Cohort Study

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ABSTRACT: Background: ABO blood group is a well-established determinant of venous thromboembolism risk, largely attributed to differences in circulating von Willebrand factor and factor VIII levels. However, its influence on disease severity and short-term outcomes in acute pulmonary embolism (PE) remains uncertain. Methodology: In this retrospective single-center cohort study, 317 consecutive adult patients hospitalized with a first episode of acute PE were included. ABO blood group was categorized as O versus non-O for the primary analysis. The primary outcome was in-hospital mortality. Secondary outcomes included Pulmonary Embolism Severity Index (PESI) score, thrombus localization on computed tomographic pulmonary angiography (CTPA), systemic thrombolysis, respiratory support requirement, infectious complications, sepsis, and ischemic stroke. Multivariable logistic regression was used to evaluate the independent association between ABO blood group and mortality, adjusting for age, sex, cancer history, and heart failure history. Results: In-hospital mortality occurred in 35 patients (11.0%). Mortality was 14.5% in group O and 10.1% in non-O patients (crude OR 0.66, 95% CI 0.30-1.45; p=0.414). In adjusted analysis, non-O blood group was not independently associated with mortality (adjusted OR 0.67, 95% CI 0.30-1.49; p=0.324). Secondary analyses showed no consistent associations between ABO blood group and baseline severity markers, thrombus localization, or most in-hospital complications. Conclusions: In this cohort of patients with acute PE, ABO blood group was not independently associated with short-term mortality or clinically meaningful markers of disease severity, suggesting that ABO phenotype does not substantially influence prognostic stratification once pulmonary embolism is established.

KEYWORDS: *ABO blood group; pulmonary embolism; venous thromboembolism; thrombosis risk.*

Introduction

Pulmonary embolism (PE) is an acute obstruction of the pulmonary arterial circulation and a major clinical manifestation of venous thromboembolism (VTE), most commonly originating from lower-extremity deep vein thrombosis (DVT).

Its clinical presentation ranges from asymptomatic or mildly symptomatic hemodynamically stable cases to severe forms complicated by obstructive shock or cardiac arrest.

Acute pulmonary arterial obstruction leads to ventilation-perfusion mismatch, increased pulmonary vascular resistance, right ventricular (RV) overload, and impaired gas exchange.

These pathophysiological mechanisms may result in hypoxemia, hemodynamic instability, and death if untreated [1-5].

PE is a multifactorial disorder associated with both acquired and inherited risk factors, including prolonged immobilization, malignancy, recent surgery, trauma, hormonal therapy, chronic inflammatory diseases, and inherited thrombophilias.

Autoimmune conditions such as antiphospholipid syndrome are also strongly

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associated with recurrent and potentially severe thrombotic events [6].

The interaction between genetic predisposition and environmental triggers plays a central role in thrombus formation and clinical expression.

Risk stratification is fundamental to the management and prognosis of acute PE. Current European Society of Cardiology (ESC) guidelines recommend an integrated approach combining clinical assessment, imaging findings, and laboratory biomarkers to identify patients at risk of early mortality and guide therapeutic decisions [7].

High-risk PE is defined by hemodynamic instability (shock or sustained hypotension), whereas non-high-risk PE is further categorized based on the presence of RV dysfunction, elevated cardiac biomarkers, and validated clinical scores such as the Pulmonary Embolism Severity Index (PESI) and simplified PESI (sPESI) [8-12].

This multiparametric model represents the cornerstone of contemporary prognostic assessment in PE.

Despite advances in diagnostic strategies and anticoagulant therapy, PE remains a leading cause of cardiovascular mortality worldwide. Its annual incidence is estimated at 60-120 cases per 100,000 individuals and increases markedly with advancing age and comorbidity burden [13-15].

In-hospital mortality ranges from approximately 5-10% overall and exceeds 15-20% among hemodynamically unstable patients.

Moreover, long-term complications, including chronic thromboembolic pulmonary hypertension and post-PE syndrome, contribute to persistent morbidity and impaired quality of life [16,17,18].

In recent years, increasing evidence has demonstrated that ABO blood group antigens influence the risk of VTE.

Individuals with non-O blood groups have an approximately 1.5-to 2-fold higher risk of VTE compared with those with blood group O [19-23].

This association is primarily attributed to higher circulating levels of von Willebrand factor (vWF) and factor VIII in non-O individuals, resulting in a prothrombotic phenotype.

However, while the relationship between ABO blood group and overall VTE risk is well established, its potential impact on PE severity, thrombus burden, clinical course, and short-term prognosis remains insufficiently explored and inconsistently reported.

The present retrospective cohort study aims to evaluate, in patients with acute PE, the association between ABO blood group and markers of clinical and imaging severity, as well as short-term outcomes, including in-hospital mortality, PESI score, need for systemic thrombolysis, thrombus localization on computed tomographic pulmonary angiography (CTPA), requirement for respiratory support, infectious complications, and ischemic stroke.

By analyzing a contemporary Eastern European cohort, this study seeks to determine whether ABO blood group may serve as a potential adjunct marker in PE risk stratification.

Objective

The present study aimed to evaluate the association between ABO blood group and clinical severity and short-term outcomes in patients with acute pulmonary embolism.

Specifically, we investigated whether ABO phenotype was associated with in-hospital mortality, Pulmonary Embolism Severity Index (PESI), thrombus localization on computed tomographic pulmonary angiography, need for systemic thrombolysis, requirement for respiratory support, infectious complications, sepsis and ischemic stroke.

Methods

Study Design and Setting: This retrospective, observational, single-center cohort study included consecutive adult patients admitted with a first episode of acute pulmonary embolism (PE) to the Municipal Hospital of Timișoara, Romania, between September 14, 2018, and June 3, 2023. Owing to the retrospective nature of the study and the use of anonymized data, the requirement for informed consent was waived.

Inclusion criteria: Patients were eligible if they met all of the following criteria:

Age ≥18 years;

Acute PE confirmed by computed tomography pulmonary angiography (CTPA);

Clinical presentation consistent with acute PE requiring hospitalization;

First documented episode of PE in the patient’s medical history.

Exclusion criteria: Patients were excluded if they had:

A history of recurrent PE;

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Chronic thromboembolic disease, including previously diagnosed chronic thromboembolic pulmonary hypertension (CTEPH);

Absence of documented ABO blood group in hospital medical records;

Missing data for the primary outcome (in-hospital mortality).

The final study cohort comprised 317 patients, representing all eligible consecutive cases during the study period. No patients met exclusion criteria after data verification.

Data Collection: Data were extracted from electronic and paper medical records, hospital discharge summaries, and the institutional laboratory database. ABO blood group was obtained from the hospital laboratory information system.

Data extraction was independently performed by two investigators, with discrepancies resolved by consensus.

No imputation of missing data was performed.

Collected variables included demographic characteristics (age, sex, and residence), pre-admission antiplatelet and anticoagulant therapy, and comorbidities documented prior to admission, including chronic venous insufficiency, chronic obstructive pulmonary disease, asthma, pulmonary hypertension, cancer, heart failure, hypertension, atrial fibrillation, prior myocardial infarction, prior stroke, dementia, hematologic diseases, diabetes mellitus, and obesity.

Hematologic diseases were defined as previously diagnosed disorders of the hematopoietic system, including chronic anemia, myeloproliferative or myelodysplastic syndromes, leukemia, lymphoma, inherited or acquired coagulopathies, and other chronic hematologic conditions documented in the medical record.

Obesity was defined as a documented diagnosis or body mass index $\geq 30 kg/m^2$. Place of residence was included as a sociodemographic covariate to account for potential differences in healthcare access and referral patterns rather than as a marker of disease severity.

Acute PE was confirmed by contrast-enhanced multidetector CTPA performed using standardized pulmonary embolism acquisition protocols on Siemens multidetector CT systems (Somatom Definition Edge and Somatom go platforms).

Image interpretation and thrombus localization were performed by board-certified radiologists as part of routine clinical practice.

Radiologic data included thrombus localization on CTPA, categorized according to the most proximal level of embolic involvement.

Clinical variables included mortality (primary outcome), PESI score at admission, requirement for systemic thrombolysis, requirement for respiratory support, occurrence of infectious complications, and ischemic stroke during hospitalization. PESI was analyzed both as a continuous variable and according to established risk classes (I-V).

Respiratory support was categorized as conventional oxygen therapy, high-flow oxygen therapy (HFO), non-invasive ventilation (including CPAP or BiPAP), and invasive mechanical ventilation.

For analysis, respiratory support was recorded both as a binary variable (any advanced respiratory support beyond conventional oxygen therapy) and as a categorical variable reflecting escalation level.

Admission to the intensive care unit (ICU) was not used as a surrogate marker of severity, given that ICU admission may be influenced by institutional policies and resource availability rather than exclusively by clinical severity.

Infectious complications were defined as clinically documented infections occurring during hospitalization and requiring antimicrobial therapy.

Sepsis was defined according to the Sepsis-3 criteria as suspected or confirmed infection associated with an acute increase in Sequential Organ Failure Assessment (SOFA) score ≥ 2 points.

Laboratory parameters at admission included hematologic, renal, metabolic, hepatic, cardiac, inflammatory, lipid, and coagulation markers, including D-dimer and N-terminal pro-B-type natriuretic peptide (NT-proBNP).

Antithrombin activity was not routinely measured and was therefore not included in the analysis.

Laboratory analyses were performed in the hospital's central laboratory using standardized automated platforms, including hematology analyzers (Sysmex XN-1000; Nihon Kohden MEK-9100), coagulation analyzers (Sysmex CS-2500), and biochemistry analyzers (Cobas 6000 c501; Dimension EXL 200).

Urinalysis was performed using the Atellica 1500 system.

All assays were conducted according to the manufacturers' instructions and internal quality-control procedures in place during the study period.

Patients were followed from admission until hospital discharge, inter-hospital transfer, or death.

Statistical Analysis: All statistical analyses were performed using an integrated Python-based computational environment (matplotlib and statsmodels libraries). All tests were two-sided, and a p-value <0.05 was considered statistically significant.

Normality of continuous variables was assessed by visual inspection of histograms and formally tested using the Shapiro-Wilk test.

Continuous variables are presented as mean±standard deviation (SD) for approximately normally distributed data and as median (interquartile range, [IQR]) otherwise.

Categorical variables are presented as counts and percentages.

Baseline demographic and clinical characteristics were compared across ABO blood groups (A, B, AB, and O) using one-way analysis of variance (ANOVA) for normally distributed continuous variables and the Kruskal-Wallis test for non-normally distributed variables. Categorical variables were compared using the χ^2 test or Fisher's exact test when expected cell counts were <5.

For the primary analysis, patients with non-O blood groups (A, B, and AB) were compared with those with blood group O to evaluate the association between ABO blood group and in-hospital mortality in patients with pulmonary embolism.

Binary outcomes and categorical severity markers, including in-hospital mortality, systemic thrombolysis, respiratory support requirement, ischemic stroke, infectious complications, and CTPA thrombus localization, are presented as event rates (%).

Crude associations between ABO categories and binary outcomes were quantified using risk ratios (RRs) and odds ratios (ORs), with corresponding 95% confidence intervals (CIs).

Risk ratios are reported for descriptive interpretation of effect size, whereas odds ratios are presented to ensure consistency with logistic regression modeling.

Although the PESI score was not normally distributed, mean values are additionally reported for descriptive completeness and comparability with prior studies. In regression analyses, respiratory support was analyzed as a binary variable (any advanced respiratory support).

An exploratory multivariable model additionally including the Pulmonary Embolism Severity Index (PESI) score was constructed to evaluate whether the association between ABO blood group and in-hospital mortality remained independent of baseline clinical severity.

Multivariable logistic regression was performed to assess the independent association between ABO blood group (non-O vs O) and in-hospital mortality. Covariates were selected a priori based on established clinical relevance and included age (modeled per 10-year increase), sex, cancer history, and heart failure history. Adjusted odds ratios (aORs) with 95% confidence intervals (CIs) were reported.

Given the number of observed events, the model was restricted to a limited set of covariates to reduce the risk of overfitting.

Multicollinearity was assessed using variance inflation factors (VIFs). Model discrimination was evaluated using the area under the receiver operating characteristic curve (AUC). A pre-specified sensitivity analysis was conducted by replacing heart failure history with chronic lung disease in the multivariable model to evaluate the robustness of the association between ABO blood group and mortality. Calibration was assessed using the Hosmer-Lemeshow goodness-of-fit test.

NT-proBNP values were unavailable in 94 of 317 patients (29.7%), reflecting non-systematic measurement during routine clinical care. Analyses involving NT-proBNP were therefore conducted using available-case data.

NT-proBNP was not systematically measured and was therefore not incorporated into multivariable modeling. No imputation of missing data was performed.All other variables included in the primary analyses were complete.

Results

A total of 317 patients with acute pulmonary embolism were included in the current analysis (Tables 1, 2).

Table 1. Distribution of ABO Blood Groups (N=317).

A	B	AB	O	Non-O
158 (49.8%)	54 (17.0%)	36 (11.4%)	69 (21.8%)	248 (78.2%)

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Table 2. Demographic, comorbidities and chronic treatment prior to admission, stratified by ABO blood group.

Characteristic	A	B	AB	O	Non-O
Age (years), mean±SD	68.2±13.9	73.1±12.7	65.4±15.9	67.4±15.1	68.9±14.1
Female sex	79 (50.0%)	30 (55.6%)	16 (44.4%)	38 (55.1%)	125 (50.4%)
Urban residence	87 (55.1%)	32 (59.3%)	24 (66.7%)	46 (66.7%)	143 (57.7%)
Chronic lung disease (including COPD and asthma)	24 (15.2%)	9 (16.7%)	2 (5.6%)	5 (7.2%)	35 (14.1%)
COPD	16 (10.1%)	7 (13.0%)	2 (5.6%)	3 (4.3%)	25 (10.1%)
Asthma	8 (5.1%)	2 (3.7%)	0 (0.0%)	2 (2.9%)	10 (4.0%)
Hypertension	107 (67.7%)	35 (64.8%)	23 (63.9%)	49 (71.0%)	165 (66.5%)
Atrial fibrillation	27 (17.1%)	9 (16.7%)	4 (11.1%)	12 (17.4%)	40 (16.1%)
Chronic venous insufficiency	31 (19.6%)	8 (14.8%)	1 (2.8%)	17 (24.6%)	40 (16.1%)
Stroke history	21 (13.3%)	8 (14.8%)	8 (22.2%)	16 (23.2%)	37 (14.9%)
Chronic kidney disease	37 (23.4%)	19 (35.2%)	10 (27.8%)	23 (33.3%)	66 (26.6%)
Myocardial infarction history	8 (5.1%)	9 (16.7%)	2 (5.6%)	11 (15.9%)	19 (7.7%)
Dementia	13 (8.2%)	8 (14.8%)	4 (11.1%)	7 (10.1%)	25 (10.1%)
Hematologic disease	51 (32.3%)	14 (25.9%)	15 (41.7%)	25 (36.2%)	80 (32.3%)
Pulmonary hypertension	93 (58.9%)	27 (50.0%)	22 (61.1%)	28 (40.6%)	142 (57.3%)
Heart failure	111 (70.3%)	45 (83.3%)	21 (58.3%)	46 (66.7%)	177 (71.4%)
Diabetes	33 (20.9%)	6 (11.1%)	8 (22.2%)	8 (11.6%)	47 (19.0%)
Cancer history	38 (24.1%)	16 (29.6%)	3 (8.3%)	12 (17.4%)	57 (23.0%)
Obesity	46 (29.1%)	19 (35.2%)	14 (38.9%)	24 (34.8%)	79 (31.9%)
Antiplatelet therapy	49 (31.0%)	11 (20.4%)	10 (27.8%)	9 (13.0%)	70 (28.2%)
Anticoagulation therapy	54 (34.2%)	13 (24.1%)	7 (19.4%)	15 (21.7%)	74 (29.8%)

Among the 317 patients included in the study, 35 (11.0%) died during hospitalization.

In unadjusted analyses, in-hospital mortality occurred in 10 of 69 patients (14.5%) with blood group O and in 25 of 248 patients (10.1%) with non-O blood groups (A, B, or AB).

The crude risk ratio (RR) for mortality comparing non-O versus O blood groups was 0.70 (95% CI, 0.35-1.38), and the crude odds ratio (OR) was 0.66 (95% CI, 0.30-1.45; p=0.414), indicating no statistically significant association between ABO blood group and in-hospital mortality.

In the primary multivariable logistic regression model adjusted for age (per 10-year increase), sex, cancer history, and heart failure history, non-O blood group was not independently associated with in-hospital mortality (Table 3).

Table 3. Adjusted Associations Between ABO Blood Group and In-Hospital Mortality.

Variable	Adjusted OR	95% CI	p-value
Non-O vs O	0.67	0.30-1.49	0.324
Age (per 10 years)	1.09	0.81-1.47	0.553
Female sex	2.81	1.25-6.34	0.013
Cancer history	1.28	0.56-2.93	0.562
Heart failure history	1.15	0.46-2.88	0.767

Model discrimination was moderate, with an area under the receiver operating characteristic curve (AUC) of 0.675. No evidence of multicollinearity was observed.

Variance inflation factors were <2 for all predictors. In a pre-specified sensitivity analysis in which heart failure history was replaced with chronic lung disease, the association between ABO blood group and mortality remained materially unchanged (aOR, 0.67; 95% CI, 0.30-1.50; p=0.330), confirming the robustness of the findings (Table 4).

Table 4. Secondary In-Hospital Outcomes According to ABO Blood Group.

Outcome	Non-O events/total (%)	O events/total (%)	Crude RR (95% CI)	Crude OR (95% CI)	p-value
Systemic thrombolysis	25/248 (10.1%)	2/69 (2.9%)	3.48 (0.84-14.32)	3.76 (0.87-16.27)	0.0587
Any ventilatory support	75/248 (30.2%)	16/69 (23.2%)	1.30 (0.82-2.09)	1.44 (0.77-2.67)	0.2520
Invasive mechanical ventilation	41/248 (16.5%)	14/69 (20.3%)	0.81 (0.47-1.41)	0.78 (0.40-1.53)	0.4660
Non-invasive ventilation	46/248 (18.5%)	2/69 (2.9%)	6.40 (1.59-25.70)	7.63 (1.80-32.28)	0.0013
Infectious complications	114/248 (46.0%)	40/69 (58.0%)	0.79 (0.62-1.01)	0.62 (0.36-1.06)	0.0776
Sepsis	28/248 (11.3%)	10/69 (14.5%)	0.78 (0.40-1.52)	0.75 (0.35-1.63)	0.4688
Ischemic stroke	10/248 (4.0%)	4/69 (5.8%)	0.70 (0.23-2.15)	0.68 (0.21-2.25)	0.5140

Baseline clinical severity was further evaluated using the Pulmonary Embolism Severity Index, presented in Table 5.

Distribution of PESI scores differed modestly (Kruskal-Wallis p=0.047) across all four ABO groups, with higher median values observed in patients with blood group B and O compared with A and AB. However, when collapsed into non-O versus O categories, neither PESI score (p=0.710) nor PESI risk class distribution (p=0.311) differed significantly (Table 6).

Table 5. Distribution of Pulmonary Embolism Severity Index (PESI) Score by ABO Blood Group.

	A	B	AB	O	Non-O
n	158	54	36	69	248
Mean (SD)	108.6 (33.5)	123.4 (35.9)	109.2 (37.4)	113.0 (35.2)	111.9 (35.0)
Median (IQR)	102 (82-128)	122 (102-142)	103 (83-129)	118 (82-128)	104 (84-131)

Table 6. PESI Risk Class Distribution by ABO Blood Group.

PESI Class	A	B	AB	O	Non-O
n	158	54	36	69	248
Class I	19 (12.0%)	4 (7.4%)	7 (19.4%)	7 (10.1%)	30 (12.1%)
Class II	29 (18.4%)	4 (7.4%)	4 (11.1%)	14 (20.3%)	37 (14.9%)
Class III	43 (27.2%)	11 (20.4%)	9 (25.0%)	11 (15.9%)	63 (25.4%)
Class IV	26 (16.5%)	14 (25.9%)	6 (16.7%)	18 (26.1%)	46 (18.5%)
Class V	41 (25.9%)	21 (38.9%)	10 (27.8%)	19 (27.5%)	72 (29.0%)

Thrombus localization on computed tomographic pulmonary angiography also did not differ significantly across ABO groups (χ^2 p=0.055) or between non-O and O categories (Table 7).

Table 7. Thrombus Localization on CTPA According to ABO Blood Group.

Thrombus Localization	A (n=158)	B (n=54)	AB (n=36)	O (n=69)	Non-O (n=248)
Trunk	21 (13.3%)	3 (5.6%)	3 (8.3%)	0 (0.0%)	27 (10.9%)
Main pulmonary artery	60 (38.0%)	18 (33.3%)	12 (33.3%)	23 (33.3%)	90 (36.3%)
Lobar	44 (27.8%)	17 (31.5%)	16 (44.4%)	22 (31.9%)	77 (31.0%)
Segmental	32 (20.3%)	16 (29.6%)	5 (13.9%)	23 (33.3%)	53 (21.4%)
Subsegmental	1 (0.6%)	0 (0.0%)	0 (0.0%)	1 (1.4%)	1 (0.4%)

Discussion

In this retrospective cohort of patients with acute pulmonary embolism, ABO blood group was not independently associated with in-hospital mortality. Although crude analyses showed a numerically lower mortality rate among patients with non-O blood groups compared with those with blood group O, this difference was not statistically significant and remained non-significant after adjustment for age, sex, cancer history, and heart failure. The direction and magnitude of the association were stable across sensitivity analyses, including substituting chronic lung disease for heart failure, supporting the robustness of the null association across modeling approaches. These findings suggest that, although the ABO blood group is an established determinant of venous thromboembolism risk, its influence on short-term mortality after acute pulmonary embolism appears limited.

An independent association between female sex and an increase in in-hospital mortality was observed. This relationship persisted after adjustment for major clinical covariates and, in exploratory analyses, for baseline severity as reflected by the Pulmonary Embolism Severity Index. The mechanisms underlying this finding cannot be determined from the present dataset and may reflect residual confounding, differences in unmeasured comorbidity burden, or cohort-specific characteristics. Given the relatively small number of observed deaths, this association should be interpreted cautiously and requires confirmation in larger prospective studies.

Secondary analyses likewise did not demonstrate a consistent association between ABO phenotype and markers of disease severity or in-hospital complications. Although thrombolysis was numerically more frequent among non-O patients and non-invasive ventilation was significantly more common in this group, these findings were not accompanied by differences in overall ventilatory support, invasive mechanical ventilation, or mortality.

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Given the small number of events in certain strata, particularly in the O group, these isolated differences should be interpreted with caution.

No consistent pattern emerged across infectious complications, sepsis, ischemic stroke, or thrombus localization on computed tomographic pulmonary angiography. While modest differences in PESI distribution were observed across the four ABO groups, these were not maintained when ABO was analyzed as non-O versus O. Overall, the data do not support a biologically coherent relationship between ABO blood group and clinical severity in acute pulmonary embolism.

The absence of an association with mortality contrasts with the well-established link between non-O blood groups and increased risk of venous thromboembolism, largely attributed to higher circulating levels of von Willebrand factor (vWF) and factor VIII. ABO-dependent glycosylation influences vWF clearance, resulting in elevated plasma levels in non-O individuals and enhanced thrombin generation [24-29].

In addition, ABO polymorphisms may modulate endothelial activation and inflammatory signaling through adhesion molecules such as E-selectin, P-selectin, and intercellular adhesion molecule-1 [30].

However, once pulmonary embolism has occurred, short-term clinical outcomes are primarily driven by acute pathophysiological processes, including right ventricular dysfunction, hemodynamic compromise, hypoxemia, systemic inflammatory response, and comorbidity burden. These factors likely outweigh baseline prothrombotic tendencies associated with ABO phenotype, which may explain the lack of association with in-hospital mortality or disease severity observed in this cohort.

Some investigators have hypothesized that elevated vWF levels might predispose to greater thrombus burden or hemodynamic instability.

However, our findings, together with those of similar observational studies, do not support a clinically meaningful relationship between ABO phenotype and short-term mortality in acute pulmonary embolism [31,32].

Several limitations merit consideration. The retrospective single-center design introduces potential selection bias and limits generalizability. The modest number of deaths restricted the number of covariates included in multivariable modeling and limited statistical power for subgroup analyses. Certain laboratory markers were unavailable for all patients, and unmeasured confounders may have influenced the observed associations.

Secondary analyses were exploratory and not adjusted for multiple comparisons.

Additionally, hemodynamic parameters, biomarkers such as vWF and factor VIII levels, and systematic thrombophilia testing were not available. The analysis was restricted to in-hospital outcomes, and structured post-discharge follow-up was not available to assess long-term mortality, recurrent thromboembolism, or chronic thromboembolic pulmonary hypertension.

Despite these limitations, this study provides real-world data from a consecutively enrolled cohort with objectively confirmed pulmonary embolism and detailed clinical characterization. Importantly, it contributes data from an Eastern European population, which remains underrepresented in the literature.

From a clinical perspective, our findings do not support incorporating ABO blood group into risk stratification or prognostic assessment for patients with established pulmonary embolism. Although ABO typing is routinely available and inexpensive, it does not appear to provide incremental prognostic information beyond validated tools such as PESI, imaging markers of right ventricular dysfunction, and cardiac biomarkers. Clinical decision-making should therefore continue to rely on established multiparametric risk assessment models rather than blood group phenotype.

Future research should focus on large multicenter prospective cohorts integrating ABO genotyping with comprehensive hemostatic profiling, including vWF antigen levels, ADAMTS13 activity, and other thrombophilia markers. Longitudinal follow-up may clarify whether ABO blood group exerts delayed or long-term prognostic effects not apparent during the acute hospitalization phase.

Conclusions

In this retrospective cohort of patients with acute pulmonary embolism, ABO blood group was not independently associated with in-hospital mortality, baseline clinical severity, thrombus localization, or most short-term in-hospital complications.

Although isolated differences were observed in certain secondary endpoints, no consistent or clinically meaningful pattern emerged.

These findings suggest that ABO phenotype, while relevant to thrombotic risk, does not

appear to substantially influence short-term outcomes once pulmonary embolism is established.

Larger, prospective studies are warranted to further clarify the potential role of ABO blood group in the prognostic assessment of acute pulmonary embolism.

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None to declare.

Author Contributions

Conceptualization, A.J.E. and O.E.T.; methodology, S.I.S.; software, A.J.E.; validation, O.E.T., I.M.M. and S.I.S.; formal analysis, A.J.E. and A.D.D.; investigation, A.J.E.; resources, A.J.E., A.D.D. and B.P.M.; data curation, A.J.E.; writing-original draft preparation, A.J.E.; writing-review and editing, S.I.S., A.D.D., B.P.M., O.E.T. and I.M.M.; visualization, O.E.T., I.M.M. and S.I.S.; supervision, O.E.T., I.M.M. and S.I.S.; project administration, A.J.E. and S.I.S. All authors read and approved the final manuscript.

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Institutional Review Board

The study was conducted in accordance with the Declaration of Helsinki, and approved by the Ethics Committee of Victor Babeș University of Medicine and Pharmacy, Timișoara, Romania, Nr. 96/04.10.2021.

Consent Statement

The requirement for informed consent was waived due to the retrospective design and use of anonymized data.

Data availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request, subject to institutional and ethical restrictions.

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