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Clinical Characteristics, Risk Factors, and Outcomes of Acute Pulmonary Embolism in Thailand: 6-Year Retrospective Study

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Abstract

Background: Acute pulmonary embolism (APE) is a fatal disease with varying clinical characteristics and imaging. The aim of this study was to define the clinical characteristics, risk factors, and outcomes in patients with APE at a university hospital in Thailand.
Methods: Patients diagnosed with APE and admitted to our institute between January 1, 2017 and December 31, 2022 were retrospectively enrolled. The clinical characteristics, investigations, and outcomes were recorded.
Results: Over the 6-year study period, 369 patients were diagnosed with APE. The mean age was 65 years; 64.2% were female. The most common risk factor for APE was malignancy (46.1%). In-hospital mortality rate was 23.6%. The computed tomography pulmonary artery revealed the most proximal clots largely in segmental pulmonary artery (39.0%), followed by main pulmonary artery (36.3%). This distribution was consistent between survivors and non-survivors. Multivariate logistic regression analysis revealed that APE mortality was associated with active malignancy, higher serum creatinine, lower body mass index (BMI), and tachycardia with adjusted odds ratio (95% confidence interval [CI]) of 3.70 (1.59 to 8.58), 3.54 (1.35 to 9.25), 2.91 (1.26 to 6.75), and 2.54 (1.14 to 5.64), respectively. The prediction model was constructed with area under the curve of 0.77 (95% CI, 0.70 to 0.84).
Conclusion: The overall mortality rate among APE patients was 23.6%, with APE-related death accounting for 5.1%. APE mortality was associated with active malignancy, higher serum creatinine, lower BMI, and tachycardia.

Keywords: Acute Pulmonary Embolism; Computed Tomography Pulmonary Artery; Malignancy; Mortality; Risk Factors

Introduction

Venous thromboembolism (VTE) is a significant global burden, representing the third most frequent acute cardiovascular syndrome among the world population, following myocardial ischemia and stroke[1]. Acute pulmonary embolism (APE) is a form of VTE that refers to obstruction of the pulmonary artery or one of its branches by thrombus[1]. The incidence of APE is rising in Europe and America, with an incidence rate of approximately 39 to 115 cases per 100,000 population[2,3]. The mortality rate of APE is as high as 30% and can lead to long-term complication such as post-thrombotic syndrome, or chronic thromboembolic pulmonary hypertension (CTEPH). Prompt diagnosis and proper treatment in time will decrease the mortality rate by 2% to 8%[4,5].

There are three major mechanisms, known as Vir-

chow's triad, that cause VTE: (1) stasis of blood flow, (2) hypercoagulable stage, and (3) endothelial injury. The embolism in pulmonary circulation reduces the capability of gas exchange and could lead to hypoxemia. Moreover, a large APE increases pulmonary vascular resistance, resulting in high pressure in the right heart chambers and leftward shift of the interventricular septum, which could lead to hypotension, shock, or death[4].

APE is associated with both hereditary, such as thrombophilia, and acquired risk factors, such as major trauma, or immobilization. Among those, one of the major risk factors for APE is malignancy, particularly those affecting the lungs, pancreas, stomach, and hematologic system. These malignancies may lead to death either from the cancer itself or from APE[4].

The primary objective of our study was to determine the clinical characteristics, risk factors, and outcomes in patients with APE in our institute. The secondary objectives were to identify the mortality rate and the risks of death among those patients.

Materials and Methods

1. Study design and participants

This was a 6-year retrospective observational study between January 1, 2017 and December 31, 2022. Approval was obtained from the Human Research Ethics Committee of Thammasat University (Medical), Thailand (IRB No. MTU-EC-IM-0-045/66), in accordance with the Declaration of Helsinki, The Belmont Report, CIOMS Guidelines, and The International Practice. Written informed consent by the patients was waived due to a retrospective nature of our study.

The inclusion criteria were APE patients aged over 18 years with a confirmed diagnosis by computed tomography (CT) chest or computed tomography pulmonary angiography (CTPA) at our institute, Thammasat University Hospital, a 650-bed tertiary care university hospital in Thailand, located in Pathum Thani province near Bangkok. The participants whose medical records were unable to be obtained or were incomplete were excluded.

The clinical characteristics, including demographic data, comorbidities, type of malignancy, heart rate, laboratory test results (arterial blood gas [ABG], com-

Table 1. Baseline characteristics and comorbidities

Characteristic	Non-survival group (n=87)	Survival group (n=282)	Total (n=369)	p-value
Female sex	60 (69.0)	177 (62.8)	237 (64.2)	0.292
Age, yr	67.1±13.3	64.6±15.7	65.2±15.2	0.179
BMI, kg/m^2	22.1±3.9	25.4±5.8	24.8±5.6	<0.001
Comorbidities				
Hypertension	39 (44.8)	152 (53.9)	191 (51.8)	0.139
Dyslipidemia	29 (33.3)	120 (42.6)	149 (40.4)	0.125
Diabetes mellitus	24 (27.6)	74 (26.2)	98 (26.6)	0.804
Stroke	8 (9.2)	31 (11.0)	39 (10.6)	0.634
Chronic kidney disease	9 (10.3)	17 (6.0)	26 (7.1)	0.169
Coronary artery disease	6 (6.9)	11 (3.9)	17 (4.6)	0.244
COPD	4 (4.6)	6 (2.1)	10 (2.7)	0.215
Cirrhosis	1 (1.2)	6 (2.1)	7 (1.9)	0.559
Risk factors				
Malignancy	57 (65.5)	113 (40.1)	170 (46.1)	<0.001
Immobilization	20 (29.9)	94 (33.3)	114 (30.9)	0.068
DVT	13 (14.9)	57 (20.2)	70 (19.0)	0.768
OCP	1 (1.2)	11 (3.9)	12 (3.3)	0.206

Values are presented as number (%) or mean±standard deviation.
BMI: body mass index; COPD: chronic obstructive pulmonary disease; DVT: deep vein thrombosis; OCP: oral contraceptive pill.

plete blood count, coagulogram, renal function, albumin, and d-dimer), characteristics of the thrombus by chest imaging (CT chest or CTPA), treatment (including reperfusion, initial anticoagulant, and long-term anticoagulant), and outcomes (in-hospital mortality), were obtained from the electronic medical records. Active cancer status is defined by patients currently undergoing treatment for cancer.

2. Statistical analysis

Demographic data were reported as frequency and percentage for categorical variables, and as mean and standard deviation (SD) or median and interquartile range for continuous variables. Comparison of the continuous data between APE patients who survived and those who did not, using t-tests for normally distributed data and Mann-Whitney U tests for skewed distributed data. The proportion between the two groups was compared using Pearson's chi-square tests, or Fisher's exact test where appropriate.

The factors associated with mortality were identified using multivariate analysis, and the relationship of each factor was determined using logistic regression analysis. To construct the clinical prediction model of mortality with conventional method, the clinical significance, mean, median, or Youden index was applied for dichotomized the variable. The parsimonious model was constructed by a backward and forward elimination approach. The prediction score was constructed regarding unadjusted coefficient of the predictor, accordingly.

The clinical prediction model was constructed through conventional methods. For score performance

Table 2. Initial laboratory investigations

Characteristic	Non-survival group (n=87)	Survival group (n= 282)	Total (n=369)	p-value
Arterial blood gas				
pH	7.38±0.20	7.44±0.10	7.42±0.13	<0.001
PaO_2, mm Hg	75 (64–117)	63 (53–78)	66 (55–83)	<0.001
$PaCO_2$, mm Hg	35 (29–43)	35 (29–43)	35 (30–40)	0.681
HCO_3, mEq/L	24 (19–28)	25 (23–28)	25 (22–28)	0.059
Other labs				
Hb, g/dL	9.9±2.1	10.8±2.4	10.6±2.3	<0.001
Platelet, $10^3/\mu L$	247 (179–344)	253 (175–352)	252 (177–351)	0.524
Cr, mg/dL	0.93 (0.65–1.48)	0.90 (0.67–1.11)	0.90 (0.66–1.17)	0.174
Albumin, g/dL	2.51±0.65	3.03±0.67	2.90±0.70	<0.001
PT, sec	15.0±4.0	13.4±2.4	13.7±2.9	<0.001
PTT, sec	30.1±14.2	27.0±17.2	27.7±16.5	<0.001
INR	1.26±0.33	1.13±0.21	1.16±0.25	<0.001
EKG				0.010
Sinus tachycardia	46 (64.9)	111 (45.5)	157 (49.8)	
NSR	22 (31.0)	128 (52.4)	150 (47.6)	
AF/Aflutter	3 (4.2)	5 (2.1)	8 (2.5)	
The most proximal clots in CTPA				0.302
Main pulmonary artery	33 (37.9)	101 (35.8)	134 (36.3)	
Lobar pulmonary artery	15 (17.2)	56 (19.8)	71 (19.2)	
Segmental pulmonary artery	31 (35.6)	113 (40.0)	144 (39.0)	
Subsegmental pulmonary artery	8 (9.2)	12 (4.2)	20 (5.4)	

Values are presented as mean±standard deviation, median (interquartile range), or number (%).
Hb: hemoglobin; Cr: creatinine; PT: prothrombin time; PTT: partial thromboplastin time; INR: international normalized ratio; EKG: electrocardiogram; NSR: normal sinus rhythm; AF: atrial fibrillation; Aflutter: atrial flutter; CTPA: computed tomography pulmonary angiogram.

assessment, the discrimination of score was assessed using receiving operating characteristics (ROC) curve analysis, the Pearson's chi-square goodness of fit (GOF) Hosmer-Lemeshow GOF was applied to explore whether the observed matched against the expected number of observations.

In the internal validation process, we split the data with the ratio of 70 and 30 based on the different time periods. The score was created from the internal validated data and C-statistics were compared between derived and validation model. Statistical significance was defined as a two-sided p-value of <0.05, except for the univariate analysis. All statistical analyses were performed using STATA version 17.0 (StataCorp., College Station, TX, USA).

Results

Over a 6-year period, a total of 369 patients were included. The mean±SD age was 65.2±15.2 years. The majority of the patients were female (237/369; 64.2%). The most common underlying diseases were hypertension (191/369; 51.8%) and dyslipidemia (149/369; 40.4%). The most prevalent risk factor for APE was malignancy (170/369; 46.1%), followed by immobilization (114/369; 30.9%), deep vein thrombosis (70/369; 19.0%), and oral contraceptive pill use (12/369; 3.3%). The most common type of malignancy was lung (36/170; 21.2%) followed by colorectal (18/170; 10.6%) and breast (12/170; 7.1%) cancer. Other characteristics are described in Table 1.

The overall in-hospital mortality rate was 23.6% (87/369). Compared to the survival group, factors associated with mortality in the non-survival group were lower body mass index (BMI) (22.1 kg/m^2 vs. 25.4 kg/m^2, p<0.001) and malignancy (65.5% vs. 40.1%, p<0.001). The results of ABG analysis in the non-survival group showed a lower pH (7.38 vs. 7.44, p<0.001) and higher partial pressure of oxygen in arterial blood (PaO_2) (75 mm Hg vs. 63 mm Hg, p<0.001) compared to the survival group, respectively. Additionally, the non-survival group demonstrated greater severity of anemia (hemo-

Table 3. Management of acute pulmonary embolism

Characteristic	Non-survival group (n=87)	Survival group (n= 282)	Total (n=369)	p-value
Initial treatment				
Reperfusion				0.485
rt-PA	4 (4.6)	7 (2.5)	11 (3.0)	
Surgical embolectomy	1 (1.1)	2 (0.7)	3 (0.8)	
Catheter embolectomy	0	1 (0.4)	1 (0.3)	
Initial anticoagulant				0.001
LMWH	48 (55.1)	204 (72.3)	252 (68.2)	
UFH	26 (29.9)	63 (22.3)	89 (24.1)	
Rivaroxaban	0	3 (1.1)	3 (0.8)	
Apixaban	0	0	0	
None	13 (14.9)	12 (4.6)	25 (6.8)	
Maintenance phase				<0.001
LMWH	33 (37.9)	126 (44.7)	159 (43.1)	
Warfarin	3 (3.4)	91 (32.3)	94 (25.5)	
DOACs	1 (1.1)	44 (15.6)	45 (12.2)	
Rivaroxaban	0	23 (8.2)	23 (6.2)	
Edoxaban	1 (1.1)	15 (5.3)	16 (4.3)	
Apixaban	0	6 (2.1)	6 (1.6)	
None	43 (49.4)	18 (6.4)	61 (16.5)	

Values are presented as number (%).
rt-PA: recombinant tissue plasminogen activator; LMWH: low molecular weight heparin; UFH: unfractionated heparin; DOAC: direct oral anticoagulant.

globin 9.9 g/dL vs. 10.8 g/dL, p<0.001), coagulopathy (prothrombin time 15.0 seconds vs. 13.4 seconds, p<0.001; partial thromboplastin time 30.1 seconds vs 27.0 seconds, p<0.001; international normalized ratio [INR] 1.26 vs. 1.13, p<0.001), and hypoalbuminemia (2.51 g/dL vs. 3.03 g/dL, p<0.001). The most proximal clots in CTPA were frequently found in the segmental pulmonary artery (144/369; 39.0%), followed by main pulmonary artery (134/369; 36.3%) as shown in Table 2.

A total of 15 patients (4%) received emergency reperfusion treatment, mostly using intravenous recombinant tissue plasminogen activator infusions. For initial anticoagulation therapy, low molecular weight heparin (LMWH) was administered to 252 patients (68.2%), followed by unfractionated heparin (UFH); 89 patients (24.1%). For maintenance anticoagulation therapy, 159 patients (43.1%) received LMWH, 94 patients (25.5%) received warfarin, and 45 patients (12.2%) received direct oral anticoagulant. There were 25 patients (6.8%) who did not receive initial treatment and 61 patients (16.5%) who did not receive maintenance treatment, due to the existence of contraindications to anticoagulant or thrombolytic therapy or the patients' decision to decline treatment. In non-survival group, 14.9% (13/87) of patients receiving no initial anticoagulation and 49.4% (43/87) lacking maintenance treatment. Among this subgroup, the mortality rate was 52% (13/25) for those without initial anticoagulation and 70% (43/61) for those without maintenance treatment. Other management details are shown in Table 3.

Pulmonary embolism-related mortality was 5.1% of the total cases (Figure 1). Among the cases of non-PE-related mortality, the leading cause of death was infection (31/68; 45.6%) with the majority of cases attributed to pneumonia (16/31; 51.6%) and intra-abdominal infection (7/31; 22.6%). Following infection, malignancy was the second leading cause of death (24/68; 27.6%). The incidence of CTEPH after APE was 3.3%. Details on outcome of patients with APE were summarized in Table 4.

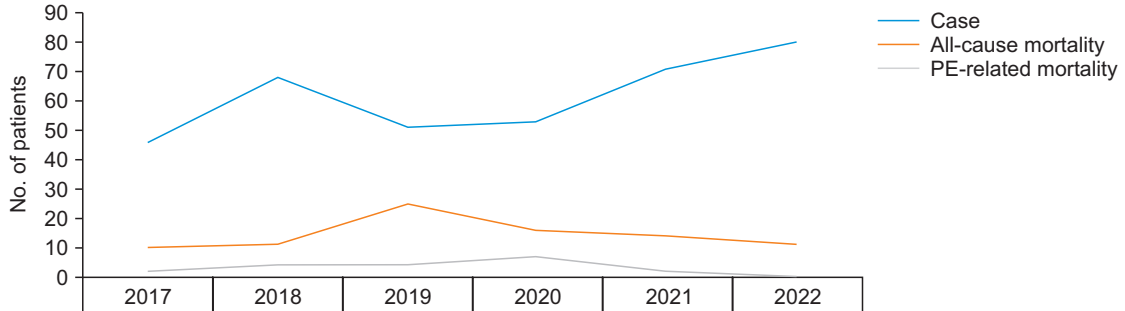
Multivariate logistic regression was performed in order to explore the clinically significant predictors of mortality. We found lower BMI, malignancy, higher se-

Table 4. The outcome of patients with acute pulmonary embolism (n=369)

Outcomes	No. (%)
Overall in-hospital mortality rate	87 (23.6)
PE-related mortality	19 (5.1)
Non-PE-related mortality	68 (18.4)
Non-PE-related mortality (n=68)	
Infection (n=31)	31 (45.6)
Pneumonia	16 (51.6)
Intra-abdominal infection	7 (22.6)
Urinary tract infection	5 (16.1)
Others*	3 (9.7)
Cancer	24 (35.3)
Major bleeding	4 (5.9)
Others†	9 (13.2)
Turn to CTEPH	12 (3.3)

*Central nervous system infection, osteomyelitis. †Volume overload, acute myocardial infarction, pulmonary hypertension type I, arrhythmia, and hyperkalemia.
PE: pulmonary embolism; CTEPH: chronic thromboembolic pulmonary hypertension.

Figure 1. Line graphs display the number of acute pulmonary embolism (PE) cases, all-cause mortality, and acute PE-related mortality by year, during 2017–2022.



	2017	2018	2019	2020	2021	2022
Case	46	68	51	53	71	80
All-cause mortality	10	11	25	16	14	11
PE-related mortality	2	4	4	7	2	0

Year

rum creatinine and tachycardia were associated with mortality with adjusted odds ratio (95% confidence interval [CI]) of 2.91 (1.26 to 6.75), 3.70 (1.59 to 8.58), 3.54 (1.35 to 9.25), and 2.54 (1.14 to 5.64), respectively (Supplementary Table S1). The clinical predictive score was constructed based on the variables' coefficient; the accuracy was shown on Supplementary Table S2, and the unadjusted predictive score was shown on Supplementary Table S3. The cut-off of −1.57 or higher associated with high mortality with sensitivity of 82.05% and specificity of 62.63%.

Score performance assessment was conducted, with the initial ROC curve showing the area of 0.77 (95% CI, 0.70 to 0.84), which is close to excellent discrimination with 83.84% correctly classified (Figure 2). The results of the Pearson's chi-square GOF test and regrouping the data for fitted model showed the model fitted reasonably well with p-value of 0.14 and 0.39, respectively. The estimated observed/expected ratio was 1.05 (95% CI, 0.92 to 1.19), which was close to one, meant that the predicted values were closer to the observed values. The calibration plot was shown in Figure 3.

The internal validation was performed by splitting the data of 66 and 163 samples. The score was generated according to the coefficient of the validation model, then compared with the derived model. The area under the ROC curve (AUC) of derivation model and validation model shown 0.74 (95% CI, 0.63 to 0.87), and 0.77 (95% CI, 0.69 to 0.87), respectively with p-value of 0.67.

The bootstrap validation was applied by sampling the subjects with replacement using 1,000 replications from the original dataset, the estimated prediction score form derived model with C-statistics of 0.77 (95% CI, 0.70 to 0.84), with bootstrap performance with optimism adjusted the C-statistics showed 0.75 (95% CI, 0.68 to 0.83). The heuristic shrinkage factor and bootstrap shrinkage factor showed 0.880 and 0.885. The model's performance of origin was close to bootstrap, the small differences of calibration coefficient showed lower bias of the model.

Discussion

Our study's findings align with a prior cross-sectional analysis of 696 Thai patients with APE[6]. We noted a higher prevalence of females and identified hypertension as the most frequent comorbidities. The predominant risk factors for APE in our study were active malignancy (55.7%), and immobilization (30.4%), similar to their observation. The clinically significant predictors for mortality were BMI, malignancies, renal function, and tachycardia.

In this study, APE-related mortality was 5.1%, which aligns with previous studies conducted among Thai and European populations[5,6]. The leading causes of death observed in our cohort were infection, followed by malignancy, consistent with a previous study[7]. Our study intentionally included untreated patients to mirror real-world practices, with 14.9% of non-surviving patients receiving no initial anticoagulation and 49.4% lacking maintenance treatment. The significantly elevated mortality rates within these subgroups (52% and 70%, respectively) highlight a notable concern. This information empowers clinicians to make well-informed treatment decisions, underscoring the critical, life-saving role of initiating and maintaining prompt and continuous anticoagulation therapy.

Our study observed a greater incidence of active

Figure 2. Receiving operating characteristics (ROC) curve analysis.

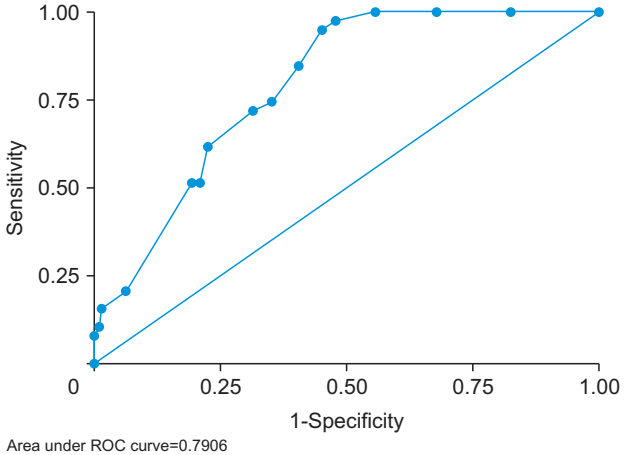
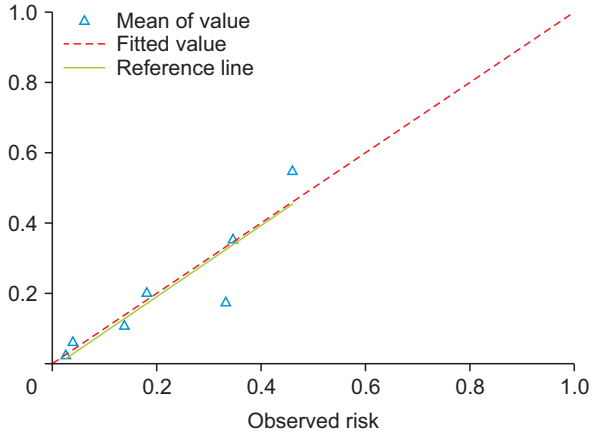


Figure 3. Calibration plot. Calibration plot showed the fitted line was close to the reference line. Y-axis show the expected risk, X-axis showed the observed risk.



malignancy in the non-survival group compared to the survival group. This finding is consistent with the results of an earlier multicenter prospective cohort study that explored clinical predictors for fatal pulmonary embolism among 15,520 patients diagnosed with VTE[8]. This connection emphasizes the detrimental impact of active malignancy on APE outcomes. Therefore, raising awareness of APE in cancer patients, may lead to proper investigation and early management. A retrospective study previously conducted in Thailand observed that the non-survival group exhibited a higher prevalence of anemia, and a higher INR. However, there were no significant differences between the two groups in terms of active malignancy and BMI[9]. In our study, the non-survival group demonstrated significantly higher rates of low BMI, hypoalbuminemia, acidosis, anemia, and coagulopathy, which are commonly indicative of chronic illness, malignancy, and/or infection. These findings suggest that these indicators may serve as a vulnerable marker for identifying patients at a higher risk of poor outcome in the context of APE. Notably, the non-survival group exhibited elevated PaO_2 levels, a phenomenon that may be attributed to their initial ABG analysis being performed after oxygen supplementation or mechanical ventilation support, leading to higher PaO_2 levels than anticipated. However, our study found no significant association between prolonged immobilization and fatal pulmonary embolism, which contrasts with previous studies suggesting that immobilization lasting more than four days increases the risk of this outcome for patients with neurological disease, cancer and advanced age[8].

We found that BMI, malignancies, renal function, and tachycardia were associated with the mortality of APE, thus, the prediction equation was constructed accordingly. The AUC was 0.77 (95% CI, 0.70 to 0.84). These results were in concordance with a previous study[10], which had AUC of 0.72 (95% CI, 0.63 to 0.81). The model construction methods and report followed the Transparent Reporting of a multivariable prediction model for Individual Prognosis Or Diagnosis (TRIPOD) statement[11] including development and validation using separate data and non-random split sample. Our model achieved an impressive accuracy with an AUC of 0.77 (95% CI, 0.70 to 0.84) comparable to the original simplified Pulmonary Embolism Severity Index (PESI) score[12] with an AUC of 0.75 (95% CI, 0.69 to 0.80). Notably, we achieved this level of accuracy with fewer prognostic factors, demonstrating the effectiveness of our approach.

The therapeutic measures of our study were consistent with a previous study in Asian population and Western countries[5,6]. LMWH was used more often than UFH in initial coagulation. For maintenance anticoagulant, LMWH and warfarin were predominant choices. In our study, we observed an incidence rate of CTEPH at 3.3%, aligning with a previous meta-analysis reporting a 2.7% incidence in pulmonary embolism survivors[13]. However, there is slightly lower incidence rate of CTEPH (3.3%) compared to a previous study at our institute (5.1%)[14]. Despite this lower rate, it is anticipated that additional patients may eventually be diagnosed with CTEPH within 2 years. Therefore, clinical and echocardiography follow-up in selected patients are warranted, as a strong correlation between maximal tricuspid regurgitation velocity and mean pulmonary arterial pressure was found[15].

The strength of our study is a relatively large sample size from a 6-year period. There are some limitations in this study. First, our study faced the limitation of not being able to definitively determine the exact cause of death in every case. Therefore, there is a possibility that some of the recorded deaths may not have been solely attributable to APE. This limitation may have influenced the accuracy of our model in predicting mortality specifically related to APE. Second, we did not collect baseline O_2 saturation, or level of oxygen supplementation, which could have affected the interpretation of ABG analysis. Third, some data could not be obtained, a common limitation of retrospective design. Lastly, our study's findings may not immediately influence the management of APE in Asian countries with comparable incidence rates. However, the study's specific focus still holds implications for APE management in these regions. The demonstrated ability of our model to achieve an AUC of 0.77 with fewer prognostic factors demonstrates the potential for its use as a tool for predicting APE risk in these regions. Further regional adaptation and validation including larger, geographically diverse datasets will be necessary to ensure its accuracy and applicability in diverse Asian contexts.

In conclusion, the overall mortality rate among APE patients in our study was 23.6%, with APE-related death accounting for 5.1%. The significant predictors for APE mortality were active malignancy, higher serum creatinine, lower BMI, and tachycardia. Our study highlights the importance of early recognition and appropriate management of APE patients with these risk factors to improve outcomes. Further studies with larger sample sizes and longer follow-up periods are needed to validate and strengthen these findings and provide more robust evidence for clinical practice.

Authors' Contributions

Conceptualization: Pirompanich P. Methodology: Pirompanich P. Formal analysis: Sapankaew T. Data curation: Suppakomonnun T. Software: Sapankaew T. Validation: Pirompanich P. Investigation: Suppakomonnun T. Writing - original draft preparation: Suppakomonnun T. Writing - review and editing: Pirompanich P, Sathitakorn O. Approval of final manuscript: all authors.

Conflicts of Interest

No potential conflict of interest relevant to this article was reported.

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Supplementary Material

Supplementary material can be found in the journal homepage (http://www.e-trd.org).

Supplementary Table S1. Multivariate logistic regression.

Supplementary Table S2. Detailed report of sensitivity and specificity.

Supplementary Table S3. Clinical predictive score derived from coefficient.

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