

# Clinical Characteristics and Outcome of Thai Patients with Acute Pulmonary Embolism

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**Background:** The incidence and clinical features of acute pulmonary embolism (APE) in Thailand are unknown. There was an unsubstantiated belief that APE in Thailand did not occur as frequently as reported in European countries. With new diagnostic tools and current advanced knowledge, APE was discovered to be much more frequent in Thailand than earlier believed. A subgroup of patients with massive APE who presented with systemic arterial hypotension tended to have poor prognosis. Clinical outcome predictor of massive APE had not been previously firmly identified.

**Study design:** Cross-sectional study.

**Objectives:** To evaluate common risk factors, symptoms, signs, commonly used investigations, treatment and outcome of Thai patients with APE. To compare the clinical characteristics of non-massive and massive APE patients. To identify the clinical characteristics and treatment that may predict the mortality of massive APE.

**Material and Method:** All patients with confirmed APE diagnoses who were admitted to Siriraj Hospital, Bangkok, Thailand between January 2001 and October 2005 were selected for analysis in the present study. All APE patients' data, including demographics, symptoms, signs, investigations, treatments, outcome and risk factors such as malignancy, surgery, immobilization, and congenital thrombophilia, were recorded.

**Statistical analysis:** Patients data is presented in the form of percent and mean. Fisher's exact test was used to compare the categorical data between massive and non-massive APE groups. T-test was used to compare continuous variable i.e. RVSP between subgroups.

**Results:** Seventy-one patients had a confirmed diagnosis of APE, 22 patients were male and 49 were female. Fourteen patients were diagnosed with massive APE. The ages of patients varied from 16 to 90 years old. The mean age was  $50 \pm 12.2$  years old. The most common presenting symptoms and signs were dyspnea (92%), followed by tachypnea (63%) and tachycardia (54%). Idiopathic APE was found in 42.2% of the patients. Malignancy, especially adenocarcinoma, was the most frequent risk factor (21%). The most frequent radiographic abnormalities noted in the present study were pulmonary parenchymal lesions (23.9%). Echocardiography findings were mostly elevated right ventricular systolic pressure (RVSP), ranging from 18.5 to 98 mmHg (mean RVSP of 54.4 mmHg). The most frequent diagnostic test used was ventilation-perfusion lung scan. Elevated serum troponin-T seemed to be more frequent in the massive APE group. In the non-survivor group, the author found hypotension and underlying malignancy statistically significant different from the survivor group.

**Conclusion:** APE was not infrequent. Characteristics of APE patients in Thailand were not different from previous reports in European countries. Malignancy seemed to be the most frequent risk factors of APE in the present study population. Troponin-T measurement may be useful to predict progression of APE.

**Keywords:** Acute, Pulmonary embolism, RVSP, Troponin, Risk factors

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More than 500,000 patients are diagnosed with acute pulmonary embolism (APE) in the United States annually, resulting in approximately 200,000 deaths. It is estimated that more than half of all patients with pulmonary emboli remain undiagnosed<sup>(1,2)</sup>.

The most common risk factors identified in the Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) study were immobilization, surgery within the last three months, stroke, history of venous thromboembolism and malignancy<sup>(4)</sup>.

Pulmonary embolism is known as “the great masquerader”. Clinical presentation of acute pulmonary embolism is variable. There were no specific symptoms and signs of this condition. Without treatment, pulmonary embolism is associated with a mortality rate of approximately 30 percent<sup>(3)</sup>. Quick and accurate diagnosis of this disorder is therefore essential.

The clinical setting, including risk factors, can help with the diagnosis of pulmonary embolism. The diagnosis is based on imaging modalities such as ventilation-perfusion lung scan (V/Q), computerized tomography of chest (CTA), magnetic resonance imaging of the chest (MRIC) or pulmonary angiography (PAgram), which is the gold standard.

Patients with massive acute pulmonary embolism (APE) present with systemic arterial hypotension. The cause of death of massive APE is right heart failure. Serum troponin I and troponin T are elevated in 30 to 50 percent of patients with a moderate to large pulmonary embolism. APE patients with elevated serum troponin T are at a higher risk for developing right ventricular dysfunction and cardiogenic shock. The presumed mechanism is acute right heart overload<sup>(5-10)</sup>. Most patients are currently treated with unfractionated heparin or low molecular weight heparin (LMWH) followed by oral warfarin. Thrombolytic therapy is indicated in massive APE. Inferior vena cava filter (IVC filter) placement is indicated when anticoagulation is contraindicated, when there is recurrence of APE despite adequate anticoagulation or in conjunction with the performance of pulmonary embolectomy, which is considered in massive APE when thrombolytic therapy is contraindicated. Failure to diagnose pulmonary embolism is associated with high mortality, and incorrect diagnosis of the condition unnecessarily exposes patients to the risk of anticoagulant therapy.

The incidence and clinical features of acute pulmonary embolism in Thailand are unknown. This may be due to the difficulties in diagnosis, poor disease recognition, non-specific presenting symptoms and

signs. There was no case series of APE in Thailand reported. With the new revolution in diagnostic tools and advanced knowledge about this condition, APE has been discovered much more frequently. The purpose of the present article was to review common risk factors, symptoms, signs, commonly used investigations, treatment and result of treatment in Thai patients with APE and the subgroup of massive APE. The secondary objective was to identify factors that may predict the progression of APE. The results of the present study may be useful as basic data for establishing future investigation and treatment guideline of patients with APE in Siriraj hospital.

## **Material and Method**

### **Subject Selection**

All adult patients (age of 15 years old and more) who were admitted to Siriraj Hospital which is a 2,500-bed university hospital in Bangkok, Thailand between January 2001 and December 2003 with ICD coding of I 26.0 (pulmonary embolism with acute cor pulmonale) and I 26.9 (pulmonary embolism without acute cor pulmonale); newly diagnosed APE patients from January 2004 to October 2005 were first recruited in the present study. The inpatient medical records were reviewed. Only the patients with confirmed APE diagnoses were selected for analysis in the present study. APE was diagnosed when one of the following criteria was met.

1. High probability V/Q by PIOPED criteria (confirmed diagnosis by radiologist)
2. Demonstration of blood clot in pulmonary artery and its branches by CTA or PAgram (confirmed diagnosis by radiologist)
3. Intermediate probability of V/Q plus demonstration of leg deep vein thrombosis (DVT) (confirmed diagnosis by radiologist)
4. Postmortem autopsy showed pulmonary embolism

The patients with acute pulmonary embolism who had hypotension of no specific causes or developed cardiac arrest were defined as having massive pulmonary embolism. The APE patients with large intra-cardiac or pulmonary arterial clot demonstrated by TTE or PAgram, right ventricular dysfunction (hypokinesia of right ventricle or paradoxical motion of interventricular septum) and stable vital signs were defined as submassive APE.

### **Method**

All APE patients' data included demographics,

symptoms (such as dyspnea, chest pain, palpitation, cough, hemoptysis, leg swelling, syncope), signs (observed in the first 24-hour of presentation such as fever defined as a temperature of over 38.3°C, tachypnea defined as a respiratory rate > 15 /minute, hypotension defined as a systolic blood pressure < 90 mmHg or a mean arterial pressure < 65 mmHg, tachycardia defined as a pulse rate > 100 beats/minute, rhonchi, crepitations, accentuated P<sub>2</sub> heart sound), investigations, treatments, outcome and risk factors, such as immobilization, trauma, surgery, obesity, hormonal treatment, previous DVT, malignancy and congenital thrombophilia, were recorded. The authors compared characteristics, risk factors, treatment and outcome between massive and non-massive APE patients in Siriraj Hospital. The parameters associated with in-hospital mortality were identified.

The present study protocol was approved by the Siriraj Hospital ethics committee.

#### Statistical analysis

The descriptive statistics were considered in this retrospective, cross-sectional study. Categorical data and continuous data were presented in terms of percent and mean with standard deviation, respectively.

Chi-square or Fisher's exact test where appropriate were used to compare the categorical data, i.e. signs, symptoms, laboratory findings, treatment and outcome between subgroups. T-test was used to

compare continuous variable, i.e. RVSP. P-value of 0.05 or less was considered significant. All statistical analyses were performed using computer software (SPSS version 13.0).

#### Results

One-hundred and seventy-two patients were initially recruited from the ICD coding diagnoses; only seventy-one patients had confirmed diagnoses of APE. Fourteen out of 71 patients were diagnosed with massive APE by unstable vital signs and autopsy findings. One-hundred and one patients were excluded due to wrong or ambiguous diagnosis, wrong coding and diagnosis of chronic thromboembolic pulmonary hypertension (14/101).

Among the 71 APE patients, 22 patients were male and 49 were female. The age range varied from as young as 16 years to as high as 90 years. The mean age was 50 ± 12.2 years old. The symptoms and signs of patients with APE are demonstrated in Table 1. The most common presenting symptoms and signs were dyspnea (93%), followed by tachypnea (65%) and tachycardia (55%). All the symptoms were comparable between the patients with massive and non-massive APE. Syncope seemed to be more frequent in the massive APE group. Hypotension and cardiac arrest were more frequently observed signs in the massive APE group. All three non-massive APE patients with initial hypotension were reversed with volume resuscitation.

**Table 1.** Characteristics of patients with massive APE and non-massive APE

Symptoms and signs	Number (%)		Total (n = 71)	p-value
	Non-massive APE (n = 57)	Massive APE (n = 14)		
Dyspnea	54 (94.7)	12 (85.7)	66	0.237
Chest pain	19 (33.3)	5 (35.7)	24	1.000
Palpitation	2 (3.5)	1 (7.1)	3	0.488
Cough	20 (35.1)	0	20	0.007
Hemoptysis	4 (7.0)	1 (7.1)	5	1.000
Leg swelling	27 (47.4)	4 (28.6)	31	0.242
Syncope	6 (10.5)	3 (21.4)	9	0.366
Tachycardia	30 (52.6)	9 (64.3)	39	0.553
Tachypnea	38 (66.7)	8 (57.1)	46	0.543
Hypotension	3 (5.3)	13 (92.9)	16	<0.001
Cardiac arrest	0	3 (21.4)	3	0.006
Rhonchi	4 (7.0)	2 (14.3)	6	0.337
Crepitation	17 (29.8)	5 (35.7)	22	0.750
Fever	15 (26.3)	0	15	0.031
Accentuated P2	14 (24.6)	1 (7.1)	15	0.273

Cough and fever were observed only in non-massive APE, which is a statistically significant difference from massive APE patients.

Thirty out of 71 APE patients had no risk factors of APE. Among the known risk factor group (57.8%), malignancy, especially adenocarcinoma, was the most frequent risk factors found (21%), followed by a history of previous DVT (18.3%) and immobilization (15.5%). There were two patients with sub-massive APE developed after surgery. One had APE on the 4<sup>th</sup> day post total abdominal hysterectomy with bilateral oophorectomy for severe vaginal prolapse; the other one had APE on 11<sup>th</sup> day post radical prostatectomy for prostate cancer. The authors found two patients with protein C deficiency and another two patients with protein S deficiency. None of these four patients with protein C or protein S deficiency had massive APE. No patients had factor V Leiden or prothrombin gene mutation. The massive APE patients seemed to be significantly more obese compared to the non-massive APE group. Risk factors of patients with APE are shown in Table 2.

Initial investigations in suspected APE patients were CXR, EKG and ABG. Normal CXR was found in only 17.4% of all patients. In the present study, the majority of APE patients had radiographic abnormalities. The most common finding was pulmonary parenchymal lesions (23.9%). The other findings were cardiomegaly (19.6%), pleural effusion (15.2%) and prominent pulmonary trunk (15.2%). Specific CXR abnormalities such as Westermark's sign were found in one patient and Hampton's hump sign in another one. The most frequent abnormality of ABG in APE patients was respiratory alkalosis with hypoxemia (68.4%). Other abnormalities were only hypoxemia, only respiratory alkalosis and respiratory acidosis, found in 7%, 17.5% and 3.5%, respectively. EKG findings are shown in Table 3. Sinus tachycardia was demonstrated in 41% of patients. The pattern S1Q3T3 (an S wave in lead I, a Q wave in lead III, and inverted T wave in lead III) was found in about one-fifth of the APE patients. There was no significant difference of EKG findings in massive and non-massive APE patients. Positive serum D-dimer (> 500 IU/ml using

**Table 2.** Risk factors of patients with massive and non-massive APE

Risk factors	Number (%)		Total (n = 71)	p-value
	Non-Massive APE (n = 57)	Massive APE (n = 14)		
Immobilization	8 (14.0)	3 (21.4)	11	0.440
Trauma	1 (1.8)	0 (0.0)	1	1.000
Surgery	2 (3.5)	0 (0.0)	2	1.000
Obesity	0 (0.0)	2 (3.5)	2	0.037
Hormonal treatment	3 (5.3)	0 (0.0)	3	1.000
Previous DVT	11 (19.3)	2 (3.5)	13	1.000
Malignancy	12 (21.1)	3 (5.3)	15	1.000
Congenital thrombophilia	4 (7.0)	0 (0.0)	4	0.578
Idiopathic	25 (43.9)	5 (8.8)	30	0.764

**Table 3.** EKG findings in massive and non-massive APE patients

EKG findings	Number (%)		Total (n = 65)	p-value
	Non-massive APE (n = 52)	Massive APE (n = 13)		
Sinus tachycardia	19 (36.5)	9 (69.2)	28	0.058
Atrial fibrillation	2 (8.0)	2 (15.4)	4	0.176
S1Q3T3	12 (23.1)	2 (15.4)	14	0.717
Right axis deviation	9 (17.3)	1 (7.7)	10	0.672
Right bundle branch block	3 (5.7)	2 (15.4)	5	0.259
Inverted T wave in chest lead	21 (40.4)	23 (15.4)	23	0.115

latex agglutination test) was found in 97.6% of 71 APE patients.

Forty out of 71 patients had a transthoracic echocardiogram (TTE) to evaluate the existence and degree of pulmonary arterial hypertension and to identify a possible alternative diagnosis. Intra-cardiac mass or clot was found in five patients. The majority of the TTE findings was elevated right ventricular systolic pressure (RVSP), which ranged from 18.5 mmHg to 98 mmHg (mean RVSP was 54.4 mmHg). Mean RVSP of the non-massive APE group was  $54.24 \pm 19.87$  mmHg which was not significantly different from mean RVSP of the massive APE group of  $54.88 \pm 12.13$  mmHg ( $p = 0.932$ ). Right ventricular dysfunction, defined as hypokinesia of right ventricle or abnormal motion of interventricular septum, was observed in four out of 71 APE patients, three of these four patients were diagnosed with massive APE. Positive serum troponin-T (more than 0.1 mg/dl) was found in 12 of 39 patients (30.8%).

**Table 4.** Diagnostic tests for APE patients at Siriraj Hospital

Years	Total number of patients	V/Q (n)	CTA (n)	PAgram (n)
2001	8	6	1	0
2002	10	9	2	3
2003	17	9	2	3
2004	22	19	6	4
2005	14	9	7	1

n = total number of tests

Interestingly, a significantly higher number of patients with massive APE had positive serum troponin T than the group with non-massive APE (54.5% vs. 21.4%;  $p = 0.01$ ).

The most frequent diagnostic test used for diagnosis of APE was V/Q (52 out of 71 patients), followed by CTA (22 out of 71 patients) and PAgram (13 out of 71 patients). Twenty-two patients had more than one diagnostic test done such as V/Q plus CTA or V/Q plus PAgram. High probability of V/Q by PIOPED criteria was found in 48 patients, intermediate probability V/Q was found in three patients (2 out of these 3 patients subsequently had evidence of DVT by Duplex ultrasound of lower extremities and one demonstrated blood clot in pulmonary vasculature by CTA). There was only one patient with low probability of V/Q and high clinical suspicion for APE who ultimately had a positive PAgram to confirm the diagnosis of APE. All patients who underwent CTA or PAgram demonstrated a clot in the pulmonary artery or its branches. In the present study, 29 APE patients (40.8%) had concurrent DVT diagnosed by lower extremities duplex ultrasound. Four patients were diagnosed with APE from postmortem examination. The different tests used to diagnose APE at Siriraj Hospital from 2001-2005 are as shown in Table 4.

Various treatments of APE patients at Siriraj Hospital are shown in Table 5. Forty-two patients (59.2%) received unfractionated heparin, adjusted for partial thromboplastin time (APTT) ratio of 1.5-2, while 26 patients (36.6%) received LMWH. Ten patients

**Table 5.** Treatments and treatment outcome of patients with acute pulmonary embolism

Treatments	Number (%)			Total (n = 71)
	Small to moderate APE (n = 50)	Submassive APE (n = 7)	Massive APE (n = 14)	
Treatments				
Unfractionated heparin	28 (56.0)	7 (100.0)	7 (50.0)	42
LMWH	22 (44.0)	0 (0)	4 (28.6)	26
Thrombolytic therapy				
Systemic	0 (0)	4 (57.1)	6 (42.9)	10
Local	0 (0)	2 (28.6)	0 (0)	2
IVC filter	4 (8.0)	2 (28.6)	2 (14.3)	8
Pulmonary embolectomy	0 (0)	1 (14.2)	1 (7.1)	2
No treatment	1 (2.0)	0 (0)	4 (28.6)	5
Treatment outcome				
Improve	46 (92.0)	7 (100.0)	7 (50.0)	60
Death	4 (8.0)	0 (0)	7 (50.0)	11

received systemic thrombolytic therapy. Six of these 10 patients had massive APE; four patients had sub-massive APE, by evidence of right ventricular dysfunction or large pulmonary emboli shown from PAgam, or a large intra-cardiac clot demonstrated by TTE. Two patients received local intrapulmonary arterial thrombolytic therapy immediately after PAgam due to suspected submassive APE indicated by PAgam findings. There was no serious bleeding complication after thrombolytic therapy in the present study. Two patients underwent emergency pulmonary embolectomy. One patient with squamous cell carcinoma of cervix had suspected massive APE from tumor emboli. In this case, thrombolytic therapy was not considered because of its uncertain benefit in tumor emboli. Another single patient with a large intra-cardiac clot, extending from right atrium through patent foramen ovale (PFO) demonstrated from TTE, underwent pulmonary embolectomy to remove a large right atrial clot and closure of PFO to prevent paradoxical emboli. Eight patients had IVC filter placed by different indication (Four patients had absolute contraindication for using anticoagulant, 1 patient had recurrence of APE despite adequate anticoagulation and 3 patients had a large clot from PAgam findings).

Outcomes of treatment are shown in Table 5. The mortality rate of massive APE was 50% (7 out of 14 patients). Among these seven patients, three received no specific treatment such as anticoagulant or thrombolytic therapy because the diagnoses of APE were made by postmortem examination. Another three

massive APE patients received anticoagulants without thrombolytic therapy due to high risk of bleeding complication. The other one died on the first day following pulmonary embolectomy. The mortality rate of non-massive APE was 7% (4 out of 57 patients). Two of these four patients had in-hospital recurrent pulmonary emboli despite adequate anticoagulants (one of these two patients had positive serum troponin-T) and the other two patients died from other causes such as septic shock. Among seven survivors from massive APE, six patients received systemic thrombolytic therapy. Only one surviving massive APE patient presented with cardiac arrest and received only heparin therapy due to the risk of serious bleeding complication after resuscitation.

Factors that may affect treatment outcome of APE patients are shown in Table 6. Comparing between the survivors and non-survivors, massive APE was significantly more prevalent in non-survivors as expected. Hypotension is the only sign that was significantly more prevalent in non-survivors as well. Cardiac arrest tends to be an unfavorable parameter ( $p = 0.06$ ). No other symptoms and signs were different between survivors and non-survivors group. Significantly higher number of non-surviving patients had underlying malignancy compared to survivors. Conversely, there was a significantly higher number of idiopathic APE in the survivors group. The number of patients with elevated serum troponin-T and the mean level of RVSP were not significantly different between survivors and non-survivors. The LMWH use was

**Table 6.** Comparison of survivors and non-survivors with APE\*

Parameter	Survivors (n = 60)	Non-survivors (n = 11)	p-value
Age, years	55.1±11.1	56.8±17.6	0.760
Syncope	8 (13.3)	1 (9.1)	1
Hypotension	10 (16.7)	6 (54.5)	0.013
Cardiac arrest	1 (1.6)	2 (18.1)	0.061
Malignancy	9 (15.0)	6 (54.5)	0.008
Idiopathic APE	29 (48.3)	1 (9.1)	0.019
Massive APE	7 (11.7)	7 (63.6)	0.001
Positive serum troponin T	10 (16.7)	2 (18.1)	0.810
RVSP, mmHg	53.7±18.4	61.3±17	0.497
RV dysfunction	3 (5.0)	1 (9.1)	0.427
Heparin therapy	36 (60.0)	6 (54.5)	0.750
LMWH therapy	25 (41.7)	1 (9.1)	0.046
Thrombolytic therapy	12 (20.0)	0 (0.0)	0.191
Pulmonary embolectomy	1 (1.6)	1 (9.1)	0.288

\* Values given as no (%) or mean ± SD

observed more in the survivors group.

## Discussion

In the past, APE was believed to be a rare condition in Thailand. With the new revolution of diagnostic tools and current advanced knowledge about this condition, APE was discovered to be much more prevalent than earlier believed. In the present study, the authors initially recruited 172 patients using the ICD diagnosis coding system. After thorough review using standard diagnostic criteria, only 71 patients had definite APE diagnoses in a 5-year period. This suggests that the hospital ICD diagnosis coding needs to be improved. Nevertheless, the true prevalence of APE is underestimated, since a few massive APE cases were found only at postmortem. In Siriraj Hospital, the prevalence of APE diagnosed in the recent years has increased which suggests an increased awareness and availability of the modern diagnostic techniques.

As described in previous studies, mostly from European countries, there were no specific symptoms and signs for APE<sup>(14)</sup>. The most common presenting symptom described was dyspnea, which was the most common presenting symptom observed in the present study population as well (93%). The two most common signs were tachycardia and tachypnea. Massive APE was found in about one fifth of all APE patients in the present study.

In previous studies, at least one risk factor of venous thromboembolism was found in 78-96 percent of APE patients<sup>(11-12)</sup>. In the present study, 57.8 percent of patients had at least one risk factor of thrombosis, which is less than previous studies. Forty-two percent of the patients had no risk factors. The authors also found much less APE in post-operative patients despite no heparin prophylaxis for deep venous thrombosis in the present patients<sup>(15)</sup>. The authors found 4 out of 71 patients with either protein C or protein S deficiency but none of these patients had massive APE. Congenital thrombophilia may increase the risk of venous thromboembolism but may not relate to the severity of thromboembolism.

The majority of patients had initial investigations such as CXR, EKG, ABG, which are non-specific to this condition; however, they are useful to identify alternative diagnosis such as myocardial infarction, heart failure, etc. Lung parenchyma lesion was the most abnormal CXR finding. ABG usually revealed hypoxemia and respiratory alkalosis, as described in a previous study<sup>(13)</sup>.

TTE was available and could be performed at bedside so it was used as an initial screening test in majority of patients (56%). TTE is useful to identify pulmonary hypertension, intra-cardiac clot, right ventricular dysfunction and other cardiac abnormalities such as atrial septal defect, ventricular septal defect that may lead to paradoxical embolism. Besides TTE, V/Q was the most frequently used test to aid in the diagnosis of APE patients in the present study, but it is not available in case of emergency in Siriraj Hospital and also had limitation for diagnosing APE in patients with preexisting lung parenchymal or airway diseases. The authors found that CTA was more frequently used to diagnose APE in the recent years in Siriraj Hospital. The advantage of CTA is that the thrombus was directly visualized so that mediastinum and lung parenchyma could be evaluated to identify other alternative diagnoses; it was available even in case of emergency. Limitation of CTA was accurate diagnosis of small emboli<sup>(16)</sup>. PAgams were done in patients with high clinical probability but cannot be diagnosed by other imaging modalities or with clinical suspected massive APE patients. Thirty-two percent of patients required more than one imaging modality to diagnose APE in Siriraj Hospital. Some patients were already diagnosed with APE by CTA or PAgam but underwent V/Q as a baseline procedure for follow up purposes.

Besides hypotension or cardiac arrest, syncope seemed to be more frequent in massive APE patients (no statistical significance). In a previous study, elevated serum troponin correlates with higher in-hospital mortality in APE patients<sup>(7)</sup>. Elevated serum troponin T was found more frequently in massive APE patients but it was not significantly different statistically between the survivors and non-survivors groups in our study. A limitation of the present study was that it was a partly retrospective data collection. Troponin T was not done in all APE patients. However, the authors felt that serum troponin T might be useful to predict clinical progression and outcome. The APE patients who have elevated serum troponin should be closely monitored.

The majority of the presented patients received anticoagulants immediately, either unfractionated heparin or LMWH. All massive APE patients who received thrombolytic treatment improved. Some submassive APE patients received thrombolytic drugs because of huge clots found in PAgam or demonstration of right ventricular dysfunction by TTE. Thrombolytics use for submassive APE is still controversial. The indications of IVC filter placement

in the presented patients were contraindication to anticoagulant, and recurrence of pulmonary emboli despite adequate anticoagulation. IVC filters were also placed in some submassive APE patients. In the present study, various treatments were used in APE patients. The authors found that the majority of patients were improved following proper treatment, such as prompt and adequate anticoagulation, with thrombolytic therapy in truly indicated patients. The highest mortality was found in massive APE and patients with underlying malignancy. The present study suggested that early diagnosis as well as prompt and proper treatments is essential to decrease mortality of APE patients.

The present study is the first study in Thailand that reviewed characteristics and outcomes of APE in Thai patients. The limitation of the present study is that it was partly retrospective. Future prospective studies are needed to accurately evaluate the prevalence of APE in Thailand and to identify the clinical predictors of progression, massive APE and mortality.

### Conclusion

Characteristics of APE patients in Siriraj Hospital including clinical presentations, risk factors, investigations that are commonly used included common findings; treatment and outcome are not different from other previous studies in Western countries. Massive APE had a few different characteristics such as hypotension, syncope and worse outcome than non-massive APE. Troponin T may be useful as one of the predictors of disease severity. The results of the present study may be useful as basic data for establishing future investigation and treatment guidelines of patients with APE in Siriraj Hospital.

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## ลักษณะทางคลินิกและผลการรักษาภาวะลิ้มเลือดอุดตันหลอดเลือดแดงปอดเฉียบพลัน ในผู้ป่วยไทย

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**ภูมิหลัง:** มีความเชื่อเดิมว่าอัตราการเกิดภาวะลิ้มเลือดอุดตันหลอดเลือดแดงปอดเฉียบพลันในประเทศไทยนั้นเกิดได้น้อยกว่าในต่างประเทศ โดยเฉพาะแถบยุโรป ในปัจจุบันมีวิทยาการและความรู้ใหม่ๆ เกี่ยวกับการวินิจฉัยภาวะนี้มากขึ้น อัตราการเกิดภาวะลิ้มเลือดอุดตันหลอดเลือดแดงปอดเฉียบพลันในไทย ดูจะมีมากขึ้นตามแต่ยังไม่มีการศึกษาอัตราการเกิด ลักษณะทางคลินิกของผู้ป่วยที่มีภาวะนี้ในประเทศไทย นอกจากนี้ผู้ป่วยภาวะลิ้มเลือดอุดตันหลอดเลือดแดงปอดเฉียบพลันบางรายมีอาการรุนแรงและมีอัตราตายสูง ลักษณะทางคลินิกหรือปัจจัยเสี่ยงต่อการเกิดอาการรุนแรงนั้นยังไม่เป็นที่ทราบกันดี

**ลักษณะการศึกษา:** Cross-sectional study

**วัตถุประสงค์:** ศึกษาลักษณะทางคลินิก อาการ อาการแสดง ปัจจัยเสี่ยง วิธีการวินิจฉัย การรักษาและผลการรักษาของผู้ป่วยภาวะลิ้มเลือดอุดตันหลอดเลือดแดงปอดเฉียบพลันในไทย เปรียบเทียบลักษณะทางคลินิกของผู้ป่วยภาวะลิ้มเลือดอุดตันหลอดเลือดแดงปอดเฉียบพลัน ประเภทรุนแรงและไม่รุนแรง ศึกษาลักษณะทางคลินิกหรือการรักษาที่อาจสัมพันธ์หรือบ่งชี้ถึงอัตราการตายในผู้ป่วยภาวะนี้

**วัสดุและวิธีการ:** คัดเลือกผู้ป่วยที่ผ่านเกณฑ์การวินิจฉัยภาวะลิ้มเลือดอุดตันหลอดเลือดแดงปอดเฉียบพลันซึ่งเข้ารับการรักษาเป็นผู้ป่วยในของโรงพยาบาลศิริราช กรุงเทพฯ ระหว่างเดือนมกราคม พ.ศ.2543 ถึง เดือนตุลาคม พ.ศ.2548 เป็นผู้ป่วยที่เข้าเกณฑ์การศึกษา ข้อมูลในด้านต่างๆ เช่น อาการ อาการแสดง วิธีการตรวจวินิจฉัย การรักษา ผลการรักษา ปัจจัยเสี่ยงต่างๆ เช่น โรคมะเร็ง ภาวะผิดปกติทางพันธุกรรมที่ก่อให้เกิดลิ้มเลือดอุดตัน การผ่าตัด ฯลฯ ของผู้ป่วย เหล่านี้จะถูกรวบรวมและวิเคราะห์

**การวิเคราะห์ทางสถิติ :** Fisher's exact test และ student t-test

**ผลการศึกษา :** ผู้ป่วยที่ได้รับการวินิจฉัยภาวะลิ้มเลือดอุดตันหลอดเลือดแดงปอดเฉียบพลันทั้งหมด 71 คน (ชาย 22 ราย หญิง 49 ราย) เป็นกลุ่มที่มีอาการรุนแรง 14 ราย อายุเฉลี่ยคือ  $55 \pm 12.2$  ปี อาการแสดงที่พบบ่อยที่สุดคืออาการเหนื่อยหอบ (92%) ตามด้วย การหายใจเร็ว (63%) อัตราการเต้นหัวใจเร็ว (54%) กลุ่มที่มีภาวะนี้โดยไม่ทราบสาเหตุพบถึง 42.2% ส่วนสาเหตุที่พบบ่อยที่สุดคือ โรคมะเร็ง (21%) ลักษณะภาพรังสีทรวงอกที่พบบ่อยที่สุด คือมีฝ้าขาวในเนื้อปอด (23.5%) การตรวจอัลตราซาวด์หัวใจพบมีความดันหัวใจด้านขวาสูงเป็นส่วนใหญ่ เฉลี่ยประมาณ 54.4 มิลลิเมตรปรอท การตรวจเพื่อยืนยันการวินิจฉัยที่ใช้บ่อยที่สุดคือ ventilation-perfusion lung scan ในผู้ป่วยภาวะลิ้มเลือดอุดตันหลอดเลือดแดงปอดเฉียบพลันชนิดรุนแรงพบมีค่า troponin-t ในเลือดที่มากกว่า กลุ่มที่เสียชีวิตพบมีความดันโลหิตต่ำ เป็นกลุ่มที่มีโรคมะเร็งหรือมีสาเหตุร่วมของการเกิดภาวะลิ้มเลือดอุดตันหลอดเลือดแดงปอดเฉียบพลันมากกว่าอย่างมีนัยสำคัญทางสถิติ

**สรุป :** ภาวะลิ้มเลือดอุดตันหลอดเลือดแดงปอดเฉียบพลันพบได้ไม่น้อยในประเทศไทย ลักษณะทางคลินิกที่พบในผู้ป่วยไทยไม่แตกต่างจากที่พบในต่างประเทศ โรคมะเร็งเป็นสาเหตุร่วมที่พบบ่อยในผู้ป่วยไทย การตรวจค่า troponin-t ในเลือดอาจมีประโยชน์ในการพยากรณ์ความรุนแรงของภาวะนี้ได้

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