Prognostic Significance of Thrombocytopenia in Acute Pulmonary Embolism

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Abstract

A reduced platelet count has been reported in acute pulmonary embolism. This study investigated the prognostic role of thrombocytopenia in acute pulmonary embolism (APE). This study retrospectively reviewed 225 consecutive APE patients. Diagnosis of APE was confirmed by either spiral computed tomography or high probability ventilation and perfusion lung scans. On the day of admission, all enrolled patients underwent initial blood tests, including platelet count. Patient exclusion criteria included intermediate- or low-probability lung scan, clinical suspicion of septic emboli, recurrent APE, chronic lung disease, hematological malignancy, liver cirrhosis, gastrointestinal bleeding or stroke within the preceding 6 months and recent surgery with bleeding risk. Assessment of the prognostic value of initial thrombocytopenia was based on either 30-day death or 30-day composite event (death, cardiopulmonary resuscitation, mechanical ventilation, thrombolytic treatment and vasopressor therapy). The 30-day mortality rate was 21.8%, and the 30-day composite event rate was 34.2% in this study. Incidence of thrombocytopenia significantly differed between the 30-day death group and the 30-day survival group ($P < 0.001$) and between the 30-day composite endpoint group and the 30-day composite event-free survival group ($P < 0.001$). Multivariate Cox regression analysis revealed the hazard ratio for thrombocytopenia was 1.63 (95% CI = 0.92-2.90) for 30-day death and 1.76 (95% CI = 1.07-2.89) for 30-day composite event. The study revealed thrombocytopenia is a predictor of short-term composite event. The simple blood examination is a rapid, noninvasive and effective test for short-term risk stratification of APE. (J Intern Med Taiwan 2008; 19: 499-507)

Key Words ： Acute pulmonary embolism, Thrombocytopenia, Platelet

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Introduction

Acute pulmonary embolism (APE) can cause catastrophic cardiovascular collapse. The International Cooperative Embolism Registry (I-COPER) of 2454 APE patients reported a short-term cumulative mortality due to pulmonary embolism of 11.4 percent at 2 weeks and 17.4 percent at 3 months[1]. Platelet activation and aggregation are key events in both thrombus formation and vasoconstriction after acute pulmonary embolism[2]. Elevated fibrin degradation products, a consequence of disseminated intravascular coagulation, occurs after pulmonary embolism[3]. Both mechanical obstruction and platelet mediated release of humoral factors induce local pulmonary vasoconstriction and hypoxic effects. The resulting hemodynamic instability may be fatal.

A reduced platelet count has been reported in association with acute pulmonary embolism[1,5-6]. During the first 48 hours, Bruce et al. noted a significant relationship between low platelet count and high artery-alveolar oxygen difference (AaDO2) in patients with acute pulmonary embolism[6]. A high AaDO2 level is known to correlate strongly with perfusion defect and short-term mortality in acute pulmonary embolism[7-8].

This study examined the prognostic role of thrombocytopenia (platelet count < 150,000/ μ L) in APE.

Methods and Materials

Study sample

This study assessed 225 consecutive patients admitted to Chang Gung Memorial Hospital (CGMH), Taiwan, between March, 1999, and July, 2005, with APE confirmed by either computed tomography (CT) or high-probability ventilation and perfusion lung scan. During the first 24 hours of admission, all enrolled patients underwent initial blood tests including complete blood count, creatinine, electrolytes, troponin I and arterial blood gas analysis. Patients with one or more of the following characteristics were excluded from the study: intermediate- or low-probability lung scan, clinical suspicion of septic emboli, recurrent pulmonary embolism, history of chronic lung disease, history of hematological malignancy, liver cirrhosis, gastrointestinal bleeding within the preceding 6 months, stroke within the preceding 6 months, a known bleeding disorder or recent surgery with bleeding risk prohibiting anticoagulation treatment. This work originally collected 245 consecutive patients with acute pulmonary embolism. After excluding patients by above exclusion criteria, the final study group has 225 patients.

In all cases, APE was treated pharmacologically. Patients with acute PE received anticoagulant therapy with unfractionated heparin dosed according to activated partial thromboplastin time or weight-adjusted low molecular heparin administered subcutaneously. Seventeen patients underwent thrombolysis by a 2h intravenous infusion of 100 mg recombinant tissue plasmin activator (tPA) without concomitant heparin.

All discharged patients were given oral warfarin with international normalized ratio 2.0-3.0 for at least 6 months . The Human Research Committee at CGMH approved this study. All participating survival patients gave informed consent. For the already expired patients, we tried to contact with their family by telephone and asked for their oral or written informed consent. Most family would give their oral consent.

Clinical features and biochemical data

Recorded clinical data included the following: age, gender, duration of symptoms, initial systolic blood pressure, underlying disease and possible risk factors. Baseline biochemical data such as blood urea nitrogen, serum creatinine, troponin I and platelet count were also examined before heparin treatment. Because the normal range of platelet count is between 150,000/uL and 400,000/uL in our hospital laboratory, we chose the cut-off value of <150,000/uL as the definition of thrombocytopenia in this study. Electrocardiography, chest X-ray and echocardio-

graphic findings were also reviewed.

Clinical Endpoints

Thirty-day all-cause death was the primary endpoint. The secondary endpoint was a composite endpoint of 30-day all-cause death and clinical deterioration requiring escalated treatment. Escalation therapy included cardiopulmonary resuscitation, mechanical ventilation, thrombolytic treatment and vasopressors to treat systemic arterial hypotension. Patients exhibiting coffee-ground material vomiting, tarry stool or bloody stool passage were treated as gastrointestinal bleeding cases. Cases of intracranial hemorrhage were confirmed by brain CT. All enrolled patients who survived the acute pulmonary embolism received follow-up (FU) treatment for at least 1 year. Additionally, any recurrence of pulmonary embolism over the one-year FU period was confirmed by high-probability lung scan and spiral CT.

Table 1.Clinical characteristics, platelet counts, echocardiographic parameters and cardiac troponin I in APE survivors and patients who expired at 30 days

Variable	30-day survival (n=176)	30-day death (n=49)	P value
Clinical characteristics			
Age	62.05 ± 15.98	63.78 ± 17.88	0.516
Duration of symptoms (day)	6.13 ± 8.15	6.53 ± 10.26	0.400
Women	93 (52.84%)	26 (53.06%)	1.000
Cancer	25 (14.20%)	20 (40.82%)	<0.001*
Systolic blood pressure (SBP)	131.47 ± 27.64	117.80 ± 28.56	0.003*
Shock (SBP <90 mmHg)	7 (3.98%)	12 (24.49%)	<0.001*
Diabetes mellitus	31 (17.61%)	10 (20.41%)	0.677
Hypertension	50 (28.41%)	10 (20.41%)	0.361
Congestive heart failure	12 (6.82%)	3 (6.12%)	1.000
Deep vein thrombosis	62 (35.23%)	14 (28.57%)	0.495
Coronary artery disease	7 (3.98%)	1 (2.04%)	1.000
Renal insufficiency	37 (21.02%)	14 (28.57%)	0.264
Recent surgery/immobilization	19 (10.79%)	6 (12.24%)	0.798
Platelet count (× 1000/ μ L)	200.38 ± 71.50	169.75 ± 91.94	0.014*
Thrombocytopenia (platelet count<150,000/ μ L)	38 (21.59%)	24 (48.98%)	<0.001*
Escalation therapy			
CPR	1 (0.57%)	17 (34.69%)	<0.001*
Mechanical ventilation	17 (9.66%)	31 (63.27%)	<0.001*
Inotropic agent	14 (7.95%)	34 (69.39%)	<0.001*
Tissue plasmin activator(tPA)	13 (7.38%)	4 (8.16%)	0.768
Other complications			
Recurrence	28(15.91%)	0 (0.00%)	0.001*
GI bleeding	3 (1.70%)	9 (18.37%)	<0.001*
ICH	0 (0.00%)	1 (2.04%)	0.218
Echocardiography and troponin I			
RVD(RV/LV ≧ 1)	42 (23.86%)	21 (42.86%)	0.012*
TnI ≧ 0.4 ng/ml	97 (55.11%)	38 (77.56%)	<0.001*

TnI: troponin I; RVD: right ventricular dilatation: RV: right ventricle; LV: left ventricle
*P<0.05

Table 2.Clinical characteristics, platelet count, echocardiographic parameters and cardiac troponin I in the 30-day composite-event-free group vs. the 30-day composite-event group

Variable	30-day free of composite end point (n=148)	30-day composite end point (n=77)	P value
Clinical characteristics			
Age	62.36 ± 15.30	62.55 ± 18.42	0.938
Duration of symptoms (day)	6.07 ± 8.06	6.51 ± 9.68	0.718
Women	75 (50.68%)	44 (57.14%)	0.399
Cancer	21 (14.19%)	24 (31.17%)	0.005*
Systolic blood pressure (SBP)	133.43 ± 28.19	119.00 ± 26.30	<0.001*
Shock (SBP<90mmHg)	4 (2.70%)	15 (19.48%)	<0.001*
Diabetes mellitus	26 (17.57%)	15 (19.48%)	0.719
Hypertension	40 (27.03%)	20 (25.97%)	1.000
Congestive heart failure	9 (6.08%)	6 (7.79%)	0.779
Deep vein thrombosis	56 (37.83%)	20 (25.97%)	0.077
Coronary artery disease	6 (4.05%)	2 (2.60%)	0.719
Renal insufficiency	30 (20.27%)	21 (27.27%)	0.234
Recent surgery/immobilization	15 (10.14%)	10 (12.99%)	0.511
Platelet count (× 1,000/ μ L)	200.40 ± 71.68	180.87 ± 85.97	0.072
Thrombocytopenia (platelet count<150,000/ μ L)	30 (20.27%)	32 (41.56%)	0.001*
Other complications			
Recurrence	22 (14.86%)	6 (7.79%)	0.142
GI bleeding	2 (1.35%)	10 (12.99%)	<0.001*
ICH	0 (0%)	1 (1.30%)	0.342
Echocardiography and troponin I			
RVD(RV/LV ≧ 1)	32 (21.62%)	31 (40.25%)	0.005*
TnI ≧ 0.4 ng/ml	79 (53.38%)	56 (72.7%)	<0.001*

TnI: troponin I; RVD: right ventricular dilatation; RV: right ventricle; LV: left ventricle
*P<0.05

Statistical analysis

Continuous variables were compared by Student t test, and categorical variables between groups were compared by chi-square test according to primary and secondary end points.

Cumulative probability of the primary and composite end points in patients with and without thrombocytopenia were estimated by multivariate Cox regression analysis. After adjusting for age, gender, other significant parameters which were examined by univariate analysis, the hazard ratio of thrombocytopenia was calculated by the Cox proportional hazard model to predict primary and secondary points. As 30-day endpoint data were completed for all study patients, no patient was censored.

Finally, the 30-day and one year survival curves were constructed based on presence or absence of thrombocytopenia.

Results

Tables 1 and 2 show baseline characteristics of the study population of 225 patients according to primary and secondary endpoints, respectively.

In the 225 patients, PE was diagnosed by high-probability lung scan in 140 (62%) patients and positive CT scan in eighty-five (38%). The 30-day mortality rate was 21.8%, and the 30-day composite event rate was 34.2%. Clinical parameters significantly

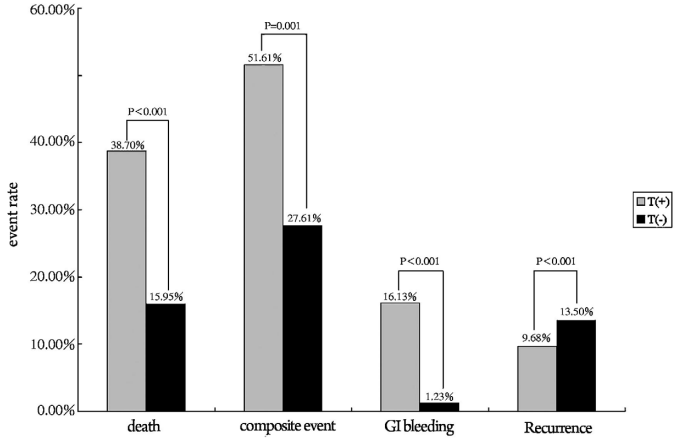

Fig.1.Comparisons of 30-day death, 30-day composite event, gasterointestinal bleeding and one-year recurrence in patients with and without thrombocytopenia.
T(+): with thrombocytopenia; T(-): without thrombocytopenia

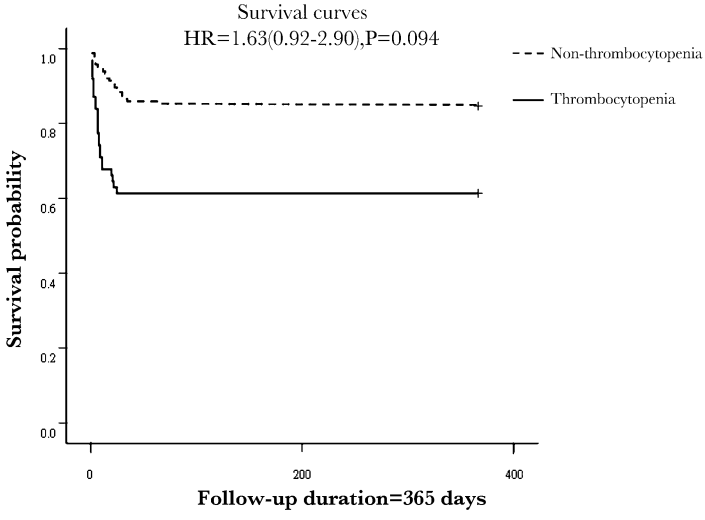

Fig.2.Survival curves at 1 year for thrombocytopenia vs. non-thrombocytopenia.

differing between the 30-day death group and the 30-day survival group included the following: cancer, systolic blood pressure, shock (systolic blood pressure < 90 mmHg), platelet count, thrombocytopenia, elevated troponin I, right ventricular dilatation and GI bleeding. The subsequent escalation therapy also significantly differed between the 30-day death group and the 30-day survival group. Between the 30-day composite event group and the 30-day composite event-free survival group, the above significant parameters count have the similar presentation except

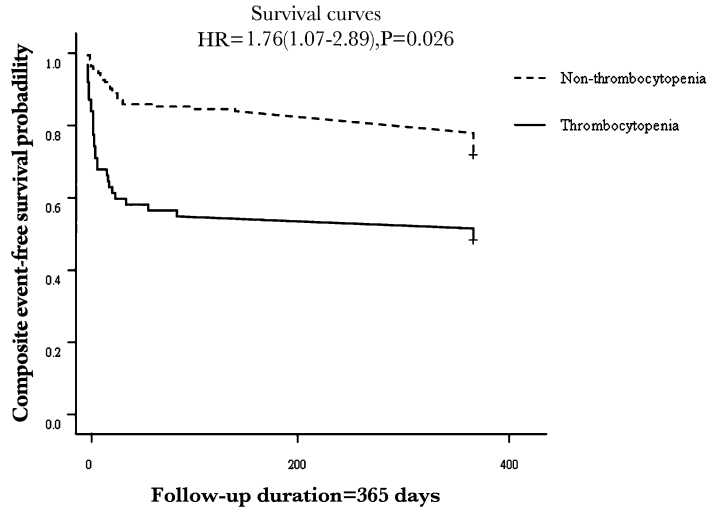

Fig.3.Composite event-free survival curves at 1 year for thrombocytopenia vs. non-thrombocytopenia.

platelet count. Difference was noted between platelet count in the 30-day composite event group and that of the 30-day event-free survival group (P=0.072) but did not reach statistical significance. However, incidence of thrombocytopenia significantly differed between the two groups (P=0.001).

The one year recurrence rate was 15.9% in the 30-day survival group (n=28) and did not statistically differ between the 30-day composite event group and the 30-day event-free survival group (P=0.142).

If we divided all patients into two groups according to the presence of thrombocytopenia, the presence of thrombocytopenia demonstrated remarkable prognostic value for short term mortality and morbidity (Fig. 1)The subsequent in-hospital GI bleeding rate was higher in patients with thrombocytopenia than in those without thrombocytopenia (Fig. 1; P<0.001). The tPA usage did not significantly correlate with the incidence of GI bleeding (P=0.256). Additionally, thrombocytopenia revealed no influence on recurrence rate after one year (Fig. 1; P=0.506).

Multivariate Cox regression was performed with parameters adjusted for age, gender and other significant interfering parameters shown in table 1 and 2. For 30-day death, the hazard ratio of thrombocytopenia was 1.63 (95% CI=0.92-2.90, P=0.094); for

30-day composite event, the hazard ratio of thrombocytopenia was 1.76 (95% CI=1.07-2.89, P=0.026). Figures 2 and 3 display the short-term and 1-year survival curves for mortality and composite events, respectively. Survival curves significantly differed between 30-day composite event versus event-free based on presence or absence of thrombocytopenia. The thrombocytopenia prior to treatment was demonstrated to have a trend between 30-day mortality versus survival by multivariate Cox-regression analysis (P=0.094).

Using initial thrombocytopenia to predict short-term outcome of APE (n=225), the predictive value of thrombocytopenia for 30-day death had a sensitivity of 78.4% and a specificity of 49.0%. The negative predictive value was 84.7%, and the positive predictive value was 38.7%. For the 30-day composite end point, thrombocytopenia had a sensitivity of 79.7% and a specificity of 41.6%. The negative predictive value was 72.4%, and the positive predictive value was 51.6%.

When we subscribed shock patient (SBP <90 mmHg), the normotensive subgroup included submassive pulmonary embolism and small pulmonary embolism. Shock patients were classified as massive pulmonary embolism and excluded from normotensive subgroup analysis (n=19). Using the initial platelet count to analyze normotensive APE (n=206), thrombocytopenia in the 30-day death had a sensitivity of 81.6% and a specificity of 45.9%. The negative predictive value was 87.0%, and the positive predictive value 33.3%. For the 30-day composite end point, thrombocytopenia had a sensitivity of 80.6% and a specificity of 37.1%. The negative predictive value was 74.8%, and the positive predictive value was 45.1%.

Discussion

The relationship between platelet and pumonary embolism

The presentation of acute pulmonary embolism was directly related to mechanical obstruction in the pulmonary vasculature or indirectly related to humoral factors[4].

Activated platelets release vasoactive agents such as serotonin, adenosine diphosphate, platelet-derived growth factor, platelet-activating factor, prostglandins and thromboxane A2[9]. These humoral factors are powerful vasocontrictors. Adenosine diphosphate can also recruit additional platelets. Platelet-activating factor has other effects, including bronchocontriction, airway vascular leakage and procoagulation[9].

Platelet activation and mediated humoral factors may result in pulmonary hypertension, bronchoconstriction and right ventricular failure[10]. The subsequent effects, including increased total dead space, physiologic shunting and pulmonary arterial pressure as well as decreased cardiac index, may develop into severe cardiopulmonary dysfunction[4].

Prognostic value of thrombocytopenia in short-term outcome

Takayoshi et al. documented a decreased platelet count after pulmonary embolism in a dog model[4]. Subsequent observational studies noted similar findings in humans[5-6]. Modig et al. also suggested that the finding of reduced platelet count could be valuable for diagnosing pulmonary embolism[5]. In this observational study, initial platelet count had good predictive value for short-term mortality and morbidity. Initial post-embolism platelet count was unaffected by anticoagulation medications such as heparin or enoxaparine and was related to platelet consumption in thromboembolic events. Increased platelet consumption was associated with increased severity of pulmonary embolism. This relationship was demonstrated by the increased rates of thrombocytopenia in the 30-day death group and the 30-day composite group.

The analytical result raises the question of whether adjunctive antiplatelet therapy is effective for treating PE. In rabbit models with induced PE, as-

pirin can reduce mortality by inhibiting prostaglandin and serotonin release of activated platelets[12-13]. Klotz et al. observed that patients taking aspirin and other nonsteroidal anti-inflammatory drugs have low urinary thromboxane B2[11]. Antiplatelet therapy with aspirin is known to have a prophylactic effect against venous thrombosis and pulmonary embolism in surgical and high risk medical patients[14]. However, further prospective studies are needed to clarify the additional anti-thrombotic effects of aspirin in humans with acute pulmonary embolism.

Prognostic value of thrombocytopenia in normotensive pulmonary embolism.

The optimal treatment strategy for submassive pulmonary embolism remains controversial[15]. Aggressive thrombolysis was an uncertain choice for solve at this classification. To elucidate this problem, the relationship between thromobytopenia and short-term prognosis was analyzed in the normotensive subgroup. For both 30-day death and 30-day composite event, initial thrombocytopenia had a significant negative predictive value and a moderate positive predictive value in patients with normotensive pulmonary embolism.

The incidence of thrombocytopenia could give us more supplementary information about the short-term prognosis in the normotensive subgroup. Aggressive treatment may be considered in these patients. However, the GI bleeding rate was higher in thrombocytopenia patients (Fig. 1). The anticoagulation therapy or even aggressive thrombolysis has a conflicting role with the elevated bleeding rate in initial thrombocytopenia patients. Although tPA and GI bleeding rate were not significantly related (p=0.256), the findings of this retrospective study are inconclusive due to the limited number of patients who underwent thrombolysis.

The low rate of thrombolytic therapy in this study

The poor prognosis of massive PE with RV dysfunction and systemic hypotension has been well documented[1,15-16]. In this study, mortality in patients with initial shock was 63.2%(n=12). Thrombolytic agents are strongly suggested for managing acute massive pulmonary embolism with hypotension and right ventricular dysfunction[15-16]. The rate of aggressive thrombolytic therapy was low in high risk patients with hypotension in this retrospective analysis. This study revealed that 24.49% of patients in the 30-day death group had initial hypotension (SBP <90 mmHg). However, thrombolytic therapy was performed in only 8.16% of cases (Table 1). This important finding may help improve treatment strategy in the future.

Limitations

First, this was a retrospective observational study. Not all patients consented to a complete survey for congenital and acquired predisposing factors. Follow-up platelet count was not performed in every patient; this analysis was performed according to clinical condition and managing physician.

Second, two diagnostic tools were used to select patients for this study. The initial selection criteria were either positive CT scan (n=85) or high-probability lung perfusion scan (n=140). The use of two different diagnostic methods resulted in inconsistent presentation in the extent of pulmonary embolism. Correlating reduced platelet count with extent of pulmonary embolism was difficult at initial evaluation and during recovery phase.

Third, some selection bias is noted. The initial selection criteria excluded patients with intermediate- and low-probability lung perfusion scan. In previous study, patients with APE had a 57% probability of presenting with low- or intermediate-probability lung perfusion scan. The inclusion criteria allowed enrollment of patients with more severe PE than general acute PE.

Fourth, the sample size was limited. The predictive significance of initial thrombocytopenia only showed a trend for primary end point by multivariate analysis.

Conclusion

This study was the first report to reveal the predictive value of thrombocytopenia for in-hospital composite events.

Post-embolism thrombocytopenia had a significant negative predictive value and a moderate positive predictive value in all patients and in patients with normotensive APE, respectively. In addition to the well-known prognostic power of both hemodynamic instability and right ventricular dysfunction, thrombocytopenia provides additional information about the severity of acute pulmonary embolism. A simple blood examination is not only an objective measure of platelet consumption but also a rapid, noninvasive and useful test for short-term risk stratification of APE.

Although the risk of GI bleeding is elevated in patients with low platelet count, this high risk group requires more thorough evaluation for aggressive treatment strategies.

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血小板低下用於預測急性肺栓塞預後的重要性

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摘 要

已經有文獻報告過在急性肺栓塞的患者有血小板減少的情形；這個研究主要是在探討血小板過低和急性肺栓塞的預後的關係。這個研究回朔性地收集分析225 個急性肺栓塞的病患，確定診斷的篩選方式有二：經過電腦斷層證實或是高度可能性的肺部核子醫學換氣灌注掃瞄結果。在病患住院的當天，所有的病患都必須接受包含血小板在內的血液初步檢查。病患篩選的排除標準包括：中度可能或是低度可能性的肺部核子醫學換氣灌注掃瞄結果、臨床懷疑敗血性栓塞、再發性的急性肺栓塞、慢性肺疾病、血癌、肝硬化、六個月內有腸胃道出血、腦中風或是大手術而有出血的危險患者均加以排除。評估病患短期預後的指標爲30 天內的死亡率以及30 天內的綜合併發症發生率(包括：死亡、實行心肺復甦術、氣道插管、病程治療中需給予血栓溶解劑者、需給予昇壓劑者)。所有的病患30 天內的總死亡率爲21.8% ，30 天內的全部綜合併發症發生率爲34.2% 。初步的單一因子分析比較入院時有血小板過低的病患和入院時沒有血小板過低的病患，無論是在30 天內的死亡率(P ＜ 0.001)或是30 天內的綜合併發症發生率(P ＜0.001)兩個預後指標均有顯著的差異。進一步的克式(Cox) 多變異數分析顯示入院時若有血小板過低的患者其30 天內的死亡危險分率爲1.63 (95% 信賴區間爲0.92-2.90)，另外30 天內的綜合併發症危險分率爲1.76 (95% 信賴區間爲1.07-2.89)。這個研究結果顯示出在急性肺栓塞病患中，若於剛入院時檢查出有血小板過低的情形，針對在30 天內發生綜合併發症的危險性而言是一個有意義的預測因子。對於急性肺栓塞的短期危險度分類，這個簡單的檢查是一個快速、非侵入性而有效率的判斷方式。