Prognostic Significance of Thrombocytopenia in Acute Pulmonary Embolism

Ju-Feng Hsiao¹, Chi-Ming Chu², Chang-Min Chung³, Shih-Tai Chang³, Chi-Tai Kuo¹, and Jen-Te Hsu³

1First Division, Cardiovascular Section, Lin-Kou Medical Center, Chang-Gung Memorial Hospital, Chang Gung University College of Medicine, Taiwan;
2Section of Health Informatics, Institute of Public Health, National Defense Medical Center and University;
3Division of Cardiology, Chiayi Chang Gung Memorial Hospital, Taiwan

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
Introduction

Acute pulmonary embolism (APE) can cause catastrophic cardiovascular collapse. The International Cooperative Embolism Registry (ICOPER) 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. Platelet activation and aggregation are key events in both thrombus formation and vasoconstriction after acute pulmonary embolism. Elevated fibrin degradation products, a consequence of disseminated intravascular coagulation, occurs after pulmonary embolism. 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. 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. A high AaDO2 level is known to correlate strongly with perfusion defect and short-term mortality in acute pulmonary embolism.

This study examined the prognostic role of thrombocytopenia (platelet count < 150,000/μL) 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/μL and 400,000/μL in our hospital laboratory, we chose the cut-off value of <150,000/μL as the definition of thrombocytopenia in this study. Electrocardiography, chest X-ray and echocardiography, chest CT scan, and ventilation-perfusion lung scan were performed to confirm pulmonary embolism.
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

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

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

### 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

<table>
<thead>
<tr>
<th>Variable</th>
<th>30-day free of composite event point (n=148)</th>
<th>30-day composite event point (n=77)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Clinical characteristics</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>62.36 ± 15.30</td>
<td>62.55 ± 18.42</td>
<td>0.938</td>
</tr>
<tr>
<td>Duration of symptoms (day)</td>
<td>6.07 ± 8.06</td>
<td>6.51 ± 9.68</td>
<td>0.718</td>
</tr>
<tr>
<td>Women</td>
<td>75 (50.68%)</td>
<td>44 (57.14%)</td>
<td>0.399</td>
</tr>
<tr>
<td>Cancer</td>
<td>21 (14.19%)</td>
<td>24 (31.17%)</td>
<td>0.005*</td>
</tr>
<tr>
<td>Systolic blood pressure (SBP)</td>
<td>133.43 ± 28.19</td>
<td>119.00 ± 26.30</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Shock (SBP&lt;90mmHg)</td>
<td>4 (2.70%)</td>
<td>15 (19.48%)</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>26 (17.57%)</td>
<td>15 (19.48%)</td>
<td>0.719</td>
</tr>
<tr>
<td>Hypertension</td>
<td>40 (27.03%)</td>
<td>20 (25.97%)</td>
<td>1.00</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>9 (6.08%)</td>
<td>6 (7.79%)</td>
<td>0.779</td>
</tr>
<tr>
<td>Deep vein thrombosis</td>
<td>56 (37.83%)</td>
<td>20 (25.97%)</td>
<td>0.077</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>6 (4.05%)</td>
<td>2 (2.60%)</td>
<td>0.719</td>
</tr>
<tr>
<td>Renal insufficiency</td>
<td>30 (20.27%)</td>
<td>21 (27.27%)</td>
<td>0.234</td>
</tr>
<tr>
<td>Recent surgery/immobilization</td>
<td>15 (10.14%)</td>
<td>10 (12.99%)</td>
<td>0.511</td>
</tr>
<tr>
<td>Platelet count (× 1,000/µ L)</td>
<td>200.40 ± 71.68</td>
<td>180.87 ± 85.97</td>
<td>0.072</td>
</tr>
<tr>
<td>Thrombocytopenia (platelet count&lt;150,000/µ L)</td>
<td>30 (20.27%)</td>
<td>32 (41.56%)</td>
<td>0.001*</td>
</tr>
<tr>
<td><strong>Other complications</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recurrence</td>
<td>22 (14.86%)</td>
<td>6 (7.79%)</td>
<td>0.142</td>
</tr>
<tr>
<td>GI bleeding</td>
<td>2 (1.35%)</td>
<td>10 (12.99%)</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>ICH</td>
<td>0 (0%)</td>
<td>1 (1.30%)</td>
<td>0.342</td>
</tr>
<tr>
<td><strong>Echocardiography and troponin I</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RVD(RV/LV ≥1)</td>
<td>32 (21.62%)</td>
<td>31 (40.25%)</td>
<td>0.005*</td>
</tr>
<tr>
<td>TnI ≥ 0.4 ng/ml</td>
<td>79 (53.38%)</td>
<td>56 (72.7%)</td>
<td>&lt;0.001*</td>
</tr>
</tbody>
</table>

TnI: troponin I; RVD: right ventricular dilatation; RV: right ventricle; LV: left ventricle

*P<0.05
Fig. 1: Comparisons of 30-day death, 30-day composite event, gastrointestinal 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.

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

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 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).
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 was 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 pulmonary embolism

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

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

Platelet activation and mediated humoral factors may result in pulmonary hypertension, bronchoconstriction and right ventricular failure. 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.

Prognostic value of thrombocytopenia in short-term outcome

Takayoshi et al. documented a decreased platelet count after pulmonary embolism in a dog model. Subsequent observational studies noted similar findings in humans. Modig et al. also suggested that the finding of reduced platelet count could be valuable for diagnosing pulmonary embolism. 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. Klotz
et al. observed that patients taking aspirin and other
nonsteroidal anti-inflammatory drugs have low uri-
nary thromboxane B2. Antiplatelet therapy with as-
pirin is known to have a prophylactic effect against
venous thrombosis and pulmonary embolism in sur-
gical and high risk medical patients. However, fur-
ther prospective studies are needed to clarify the ad-
tional anti-thrombotic effects of aspirin in humans
with acute pulmonary embolism.

Prognostic value of thrombocytopenia in normo-
tensive pulmonary embolism.

The optimal treatment strategy for submassive
pulmonary embolism remains controversial. Aggres-
sive thrombolysis was an uncertain choice for
solve at this classification. To elucidate this problem,
the relationship between thrombocytopenia and short-
term prognosis was analyzed in the normotensive
subgroup. For both 30-day death and 30-day com-
posite event, initial thrombocytopenia had a signifi-
cant negative predictive value and a moderate posi-
tive 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 pa-
tients. However, the GI bleeding rate was higher in
thrombocytopenia patients (Fig. 1). The anticoagula-
tion therapy or even aggressive thrombolysis has a
conflicting role with the elevated bleeding rate in ini-
tial 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 dys-
function and systemic hypotension has been well doc-
umented. 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 ven-
tricular dysfunction. The rate of aggressive throm-
bolytic therapy was low in high risk patients with hy-
potension in this retrospective analysis. This study re-
vealed 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 sur-
vey for congenital and acquired predisposing factors.
Follow-up platelet count was not performed in every
patient; this analysis was performed according to clin-
ical 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-proba-
bility 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 pul-
monary embolism was difficult at initial evaluation
and during recovery phase.

Third, some selection bias is noted. The initial
selection criteria excluded patients with interme-
diate- and low-probability lung perfusion scan. In pre-
vious study, patients with APE had a 57% probabili-
ty of presenting with low- or intermediate-probabi-
dity lung perfusion scan. The inclusion criteria al-
lowed enrollment of patients with more severe PE
than general acute PE.

Fourth, the sample size was limited. The pre-
dictive 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, non-invasive 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.

References

血小板低下用於預測急性肺栓塞預後的重要性

蕭如豐 朱基銘 鍾昌珉 張士奎 郭啓泰 徐仁德

1. 長庚紀念醫院林口醫學中心 第一心臟內科 長庚大學
2. 國防醫學院 公共衛生系
3. 長庚紀念醫院嘉義分院 心臟內科

摘 要

已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。這個研究回顧性地收集分析225個急性肺栓塞的病患，確定診斷的篩選方式有三：經過電腦斷層證實或是高度可能性的肺部核磁共振掃描結果。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磁共振掃描結果、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、肝硬化以及六個月內有腸胃道出血、腸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為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、需給予昇壓劑者)。所有的病患30天內的綜合併發症發生率為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Cox多變異數分析顯示入院時若有血小板過低的病患其30天內的死亡危險分數為1.63 (95% 信賴區間為0.92-2.90)，另外30天內的綜合併發症危險分數為1.76 (95% 信賴區間為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。