Acute Pulmonary Embolism : Present Status and Therapeutic Strategy

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ABSTRACT Acute pulmonary embolism causes increasing right ventricular afterload and hypoxia by obstructing the pulmonary artery and triggering the release of vasoconstricting factors, especially in patients with massive or moderate-large pulmonary embolisms. Acute pulmonary embolism can cause sudden death in up to 10% of patients due to right ventricular dysfunction. We reviewed the records of 58 consecutive patients (mean age, 59.2 years) with pulmonary embolisms admitted at our institution from April 1994 to December 1999. Thirty-five patients were female. Predisposing factors for venous thromboembolism were postoperative state, immobilization, obesity, etc. Seven patients had caval thrombosis (free floating type in 4). The first choice clinical diagnostic methods were echocardiography and computed tomography for patients with massive or moderate-large PE. Echocardiography was a rapid, practical, and sensitive technique for the identification of right ventricular dysfunction due to pulmonary embolism. Treatments consisted of surgical embolectomy in 7, catheter-directed fragmentation in 3, heparin plus thrombolytic therapy alone in 20, anti-coagulation therapy alone in 22, and conservative therapy including inotropic agents in 6. Overall early mortality was 17.2%. Causes of death were shock in 2, right ventricular failure in 5, multiple organ failure with coagulopathy in 2, and postresuscitation syndrome in 1. Inferior vena cava filters were used in 9 patients (permanent type in 6, temporary type in 3). Neither recurrence of pulmonary embolism, nor complications caused by filters were clinically recognized. Multivariate analysis showed that modified Greenfield's classification and right ventricular dysfunction were significant incremental risk factors. If patients with massive pulmonary embolism can not improve severe right ventricular dysfunction only due to medical therapy, prompt catheter based therapy or surgical embolectomy are recommended. Moreover, combined surgical thrombectomy for free-floating type caval thrombosis and proximal massive pulmonary embolism was safe and effective. The use of IVC filter was effective to prevent the fatal recurrence of pulmonary embolism. If fatal pulmonary embolism could be predicted, first of all, the use of temporally IVC filter should be considered before treatments for pulmonary embolism or deep vein thrombosis.

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Introduction

Pulmonary embolism would be the third most frequent cause of death in the United States¹⁾. It used to be fewer in Japan compared with Europeans and Americans. Its fre-

quency in Japan was estimated to be about one third of that in USA in a report that studied autopsies in detail²⁾. Recently this disease was increased³⁾. About 10% of pulmonary embolism patients die suddenly¹⁾. If diagnosis was made and therapy was instituted promptly after onset, the mortality of pulmonary embolism was 8%, but if not, the mortality was 30%¹⁾. Precise and quick diagnosis improves prognosis. Acute pulmonary embolism suddenly increases right ventricular afterload and hypoxia by obstructing the pulmonary artery and triggering the release of vasoconstrictive factors, especially in patients with

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massive or moderate-large pulmonary embolism, leading to death⁴⁾. The purpose of this study was to assess the current status of pulmonary embolism incidence and investigate strategies for better outcomes.

Material and Methods

Patients

Between April 1992 and December 1999, 58 consecutive patients with clinical evidence of pulmonary embolism were studied. They were treated on the medical and surgical wards as emergency patients and inpatients in Oita Medical University Hospital, Oita, Japan. A final diagnosis of pulmonary embolism was made after ventilation-perfusion lung scan in 30 patients (51.7%), pulmonary angiography in 19 (32.8%), enhanced spiral CT in 6 (10.3%), and echocardiography in 3 (5.2%). Obesity was defined as over 25 of body mass index [BMI : body weight (kg) / height (m)²]⁵⁾. Slow onset was defined as onset with prodromic signs as momentary minor chest pain same days before onset.

Data Collection

Hospital records were reviewed retrospectively for patient demographic characteristics, status and comorbidity before onset, treatment, and outcome. Several variables included in the study were as follows : predisposing factors (Table 1), mode of onset, deep vein thrombosis, location of vein thrombi, symptoms, blood gas analyses, echocardiography, spiral CT, perfusion lung scan, ventilation lung scan, pulmonary angiography (PAG), location of thrombi of the pulmonary artery, treatments, and functions of coagulation and fibrinolysis.

The operative technique was similar in all patients who were had surgical embolectomy. After standard anesthesia, a median sternotomy was performed followed by routine aortic and bicaval cannulation. A cardiopulmonary bypass was carried out using membrane oxygenators, and mild systemic hypothermia (32°C). Thrombectomy for deep vein thrombosis (DVT) was performed from the femoral vein at the same time. If there was caval thrombosis, thrombectomy of the inferior vena cava was performed through the right atrium using a balloon under ventricular fibrillation without aortic cross clamping or cardioplegia. Both main pulmonary arteries were incised and an embolectomy was performed.

Greenfield's classification⁶⁾ of pulmonary thromboembolism was divided 4 categories as follows ; minor, major, massive, and chronic. To evaluate the severity of pul-

Table 1. Patient characteristics.

Variables	Data (n=58)	(%)
Age (yrs)	59.5 ± 16.3	
M / F	23/35	
Postoperative state	22	37.9
Immobilization >5 days	15	25.9
Obesity	13	22.4
Prior DVT/PE	10/4	17.2/6.9
Malignancy	9	15.5
Cardiac disease	8	13.8
Prethrombotic state	6	10.3
Catheterization	4	6.9
IVH from the femoral vein	3	5.2
Pregnancy and postpartum state	2	3.4
Hormonal therapy	2	3.4

Data show number of patients or mean ± SD.

DVT = deep vein thrombosis ; PE = pulmonary embolism ;

IVH = intravenous hyperalimentation

Obesity was defined as a body mass index over 25 [BMI : body weight (kg) / height (m)²].

monary embolism, the below minor, minor, moderate, major, and major with syncope of Greenfield's classification were coded as follows ; 0, 1, 2, 3, 4, respectively (Table 2).

Right ventricular dysfunction was assessed by echocardiography or measurement of right atrial pressure and defined as symptoms of pulmonary hypertension and right ventricular dysfunction (right ventricular dilatation, intraventricular septal flattening and paradoxical motion, or right ventricular wall hypokinesis)⁴⁾ or a mean right atrial pressure higher than 15 mmHg.

Statistical Analysis

Continuous variables were expressed as the mean ± 1 standard deviation and were compared using an unpaired two-tailed t test. Categorical variables were analyzed with a chi-square test or Fisher's exact test. Ordinal data was analyzed with the Mann and Whitney's U-test or the Kruskal-Wallis test. A p value of less than 0.05 was taken as significant. To evaluate independent risk factors for early mortality, significant variables due to univariate analyses before and after onset were examined by multivariate analyses by forward stepwise logistic regression. Coefficients were computed using the method of maximum likelihood.

Table 2. Modified greenfield's classification.

Score	Category	Signs and symptoms	Blood gases	PA occlusion (%)	Hemodynamics
0	below minor		PaO ₂ ≥ 80mmHg PaCO ₂ ≥ 35mmHg	0-20	
1	minor	anxiety, hyper-ventilation	PaO ₂ < 80mmHg PaCO ₂ < 35 mmHg	20-30	tachycardia
2	major	dyspnea, collapse	PaO ₂ < 65mmHg PaCO ₂ < 30mmHg	30-50	CVP elevated, PA > 20mmHg responds to resuscitation
3	massive	dyspnea, shock	PaO ₂ < 50 mmHg PaCO ₂ < 30 mmHg	> 50	CVP elevated, PA > 25mmHg Requires pressors, inotropes
4	massive +syncope	dyspnea, shock, syncope	PaO ₂ < 50 mmHg PaCO ₂ < 30 mmHg	> 50	CVP elevated, PA > 25mmHg Requires pressors, inotropes

Table 3. Univariate analysis of patients with PE occurring at out-hospital and patients with PE occurring in-hospital.

	Out-hospital		In-hospital		p value
	data	n	data	n	
Age (yrs)	58.0 ± 18.1	28	60.8 ± 14.5	30	p=0.51
Days from onset to diagnosis	12.6 ± 22.5	24	1.6 ± 3.8	25	p=0.026
Immobilization (days)	1.4 ± 4.0	24	7.4 ± 4.7	24	p<0.01
Postoperative state	4	28	17	30	p<0.01
PaO ₂ (mmHg)	60.4 ± 17.5	28	50.2 ± 17.2	29	p=0.030
m. Greenfield's classification	2.11 ± 1.13	28	2.79 ± 0.94	29	p=0.024
Fibrinogen (mg/dl)	298 ± 108	21	398 ± 197	22	p=0.044
No of pts with AT- III less than 60%	5	17	0	18	p=0.020
Slow onset	13	28	5	29	p=0.024

Data show mean ± SD or number of patients. m. Greenfield's classification = modified Greenfield's classification ; No of pts = number of patients ; AT- III = antithrombin III

Slow onset was defined as onset with prodromic signs as momentary minor chest pain some days before onset.

Results

Patient characteristics are shown in Table 1. The mean age was 59.5 ± 16.3 years old (range, 20-84 years). The location of onset was an out-hospital in 34 (58%), a former hospital in 4 (7%), our hospital in 20 (35%), and both out-hospital and recurrence in our hospital in 6 (10%). Acute pulmonary embolism occurred in-hospital in about half of the patients. **Table 3** shows a comparison between patients who had pulmonary embolism at an out-hospital and patients who had in-hospital. The patients who had pulmonary embolism at an out-hospital had short immobilization and slow onset.

The operative portion consisted of vertebrae and the spinal cord in 7 (12.1%), the head in 4 (6.9%), the digestive system in 4 (6.9%), the cardiovascular system in

3 (5.2%), and the bladder, kidney, genital organs, and lung in each 1. Thrombophilia consisted of antiphospholipid syndrome in 3 (5.2%), protein C deficiency in 2 (3.4%), anti-thrombin III deficiency in 1 (1.7%) as a prethrombotic state.

The symptoms at onset were unconsciousness in 6 (10.3%), shock in 6 (10.3%), dyspnea in 27 (46.6%), chest pain in 12 (20.7%), cough in 5 (8.6%), and no subjective symptoms in 5 (8.6%). The first methods for clinical diagnosis of acute pulmonary embolism are shown in **Fig.1**, distinguishing whether pulmonary thrombi were located proximal or distal. For patients with pulmonary embolism in the proximal pulmonary artery, in order, echocardiography, spiral CT, pulmonary arteriography, and ventilation-perfusion lung scans were used. For patients with pulmonary embolism in distal pulmonary artery, a ventilation-perfusion lung scan was used most fre-

Fig.1. First methods for clinical diagnosis.

sCT = spiral computed tomography ; PAG = Pulmonary angiography

These methods are showed to distinguish whether pulmonary thrombi were located proximal or distal.

■ = proximal PA ; □ = distal PA

quently.

Echocardiography detected right ventricular dysfunction (right ventricular dilatation, intraventricular septal flattening and paradoxical motion, or right ventricular wall hypokinesis in 25 patients (43.1%) and pulmonary hypertension estimated by tricuspid valve regurgitation in 11 (19%), in the acute phase⁴⁾. Enhanced spiral CT detected thrombi in the proximal pulmonary artery in 8 of 33 patients and delta-shaped consolidation of the peripheral lung field in 7 of 33 in the acute phase. Also, DVT was detected in 10 of 11 by enhanced spiral CT. DVT was detected in 9 of 10 patients by ultrasonography⁷⁾. Pulmonary angiography was not often used initially, but was used as a secondary survey in 23 patients (39.7%) and abnormalities were found in 19 in the acute phase.

The proximal portion of deep vein thrombosis was inferior vena cava (IVC) in 7 (12.1%), from the iliac vein to popliteal vein in 17 (29.3%), the calf vein in 8 (13.8%), and was unknown in 23 (39.7%), due to no symptoms or the unstable condition of the patients. Free-floating caval thrombosis accounted for 4 of 7 patients (**Fig.2**).

No significant relationship was found between a modified Greenfield's classification and proximal portion of venous thrombi (**Fig.3**).

Therapies for pulmonary embolism are shown in **Fig.4**, separated by the modified Greenfield's classification. The percentages of patients over class 3 were higher in the group with catheter-directed fragmentation and thrombolysis, at 100% and 63.2%, respectively. As for thrombolytic therapy, total urokinase was used in 15, and t-PA was used in 12 patients. Outcomes are shown in **Fig.5** separated by

treatment methods. All 3 patients with catheter-directed fragmentation showed improved circulatory condition, but one of them died due to cerebral damage after resuscitation. Patients receiving anti-coagulation therapy included 12 patients with minor pulmonary embolism or below minor pulmonary embolism, and 7 patients with postoperative pulmonary embolism. Seven patients receiving conservative therapy included one sudden death, one late diagnosis, and minor or below minor pulmonary embolism in 5.

Inferior vena cava filters were used in 9 patients. Six of these patients were treated with the permanent type, and 3 with temporary type. All patients who received permanent filters were over class 3, and 2 patients out of 6 had thrombophilia. Two patients with temporary filters had caval thrombosis and 1 had iliac vein thrombosis. The purpose of a temporary filter was to avoid fatal recurrence of pulmonary embolism for patients with vein thrombi during treatment. String-like thrombi twisted around the filter portion were pulled from the right atrium during surgical embolectomy (**Fig.6**). The durations for temporary IVC filter use were 4 days in 2 patients and 11 days in 1 patient. All were discharged without recurrence.

The cause of early death was shock in 2, respiratory and right ventricular failure in 5, disseminated intravascular coagulopathy (DIC) and multiple organ failure (MOF) in 2, and post-resuscitated cerebral damage in 1. Three patients had fatal recurrence some days after recovering from shock. Hospital death occurred in one patient with coronary bypass grafting for acute myocardial infarction because of sepsis.

Fig.2. Free-floating caval thrombosis causing pulmonary embolism.

Venogram (a) and CT (b) show filling defect in inferior vena cava due to free-floating caval thrombosis. Pulmonary angiogram (c) shows large thrombi in the left proximal pulmonary artery.

Fig.3. Modified Greenfield's classification and location of proximal venous thrombus.

IVC = inferior vena cava

Reasons of unknown were negative findings of Venogram or no examination for DVT because of no clinical sign of DVT.

4: massive with syncope; 3: massive; 2: major; 1: minor; 0: below minor

In univariate analyses, significant variables for early death were low PaO₂, low O₂ saturation, high pulmonary capillary wedge pressure, high right atrial pressure, high grade of modified Greenfield's classification, right ven-

tricular dysfunction detected by echocardiography, symptoms of syncope, and patients requiring a respirator (Table 4). In multivariate analysis, high grade of modified Greenfield's classification and right ventricular dysfunction were significant incremental risk factors (Table 5).

Discussion

Epidemiology

The acute mortality of pulmonary embolism is not known because the numerator (number of pulmonary embolism deaths per year) is uncertain, and the denominator (the total incidence of pulmonary embolism) is unknown in Japan. Because clinical diagnosis of pulmonary embolism is inaccurate and pulmonary embolism usually occurs as a complication of other potentially fatal disease. In reviewing the incidence of pulmonary embolism as detected at postmortem, there was large variance between reporters. Freiman et al⁸⁾ reported the incidence of pulmonary embolism was 64% with meticulous dissection after the lung had been inflated and fixed. The previously reported incidence of pulmonary embolism at postmortem at the same hospital had been 33%. Nakano et al²⁾ also reported the incidence of pulmonary embolism

Fig.4. Therapies and modified Greenfield's classification.
 4 : massive with syncope ; 3 : massive ; 2 : major ;
 1 : minor ; 0 : below minor

Fig.5. Outcome and therapies.
 □ = alive ; ■ = early death ; ▨ = late death

Fig.6. Temporary vena cava filter with thrombi removed from the right atrium at embolectomy.
 String-like thrombi twisted around the filter portion.

was 24% about Japanese with meticulous dissection by the same method as Freiman et al⁸⁾. This suggested that Japanese incidence of pulmonary embolism was one third that of USA and that the incidence could be estimated to be higher than expectations. This is predicted to increase, because of the adoption of a Western life style and improvements in the diagnostic accuracy for pulmonary embolism in Japan. On the other hand, DVT occurs in 2 million people each year in USA¹⁾. Total incidence of pulmonary embolism was about 630,000. About 67,000 patients out of these cases die due to massive pulmonary embolism within 1 hour without time to diagnosis and in-

Table 4. Univariate analysis of risk factors for early death.

	Alive		Early death		p value
	data	n	data	n	
Age (yrs)	58.5 ± 16.6	48	64.2 ± 14.3	10	p=0.25
Male/Female	19/29	48	4/6	10	p=1.00
PaO ₂ (mmHg) (room air)	58.4 ± 15.7	46	40.2 ± 21.3	10	p<0.01
O ₂ SAT (%)	90.9 ± 6.9	38	77.1 ± 16.8	8	p<0.01
RV failure (echocardiography)	17	48	8	10	p=0.014
m. Greenfield's classification	2.27 ± 1.03	48	3.30 ± 0.95	10	p=0.038
PCWP (mmHg)	12.8 ± 8.5	12	27.3 ± 17.7	4	p=0.042
mRA (mmHg)	6.8 ± 7.0	13	15.4 ± 4.7	5	p=0.022
Syncope	7	46	5	12	p=0.024
No. of respirator use	4	48	6	10	p<0.01

Data show mean ± SD or number of patients. m. Greenfield's classification = modified Greenfield's classification ; PCWP = pulmonary capillary wedge pressure ; mRA = mean right atrial pressure

Table 5. Multivariate analysis of risk factors for early death.

Predictors	Odds ratio	95% CI	p value
m. Greenfield's classification	3.25	1.1-9.5	0.027
Right ventricular dysfunction	6.16	1.0-38.1	0.045

CI = confidence interval ; m. Greenfield's Classification = modified Greenfield's Classification. RV dysfunction was assessed by echocardiography or measurement of right atrial pressure and defined as symptoms of pulmonary hypertension and right ventricular dysfunction or a mean right atrial pressure higher than 15 mmHg.

stitute therapy, and 133,000 patients die after 1 hour.

Predisposing factors

Predisposing factors for venous thromboembolism in the present study were similar to the report of Society for Vascular Surgery / North American Chapter⁹⁾. These predisposing factors were often found in in-hospital patients (Table 2). As indicated by the American Heart Association (AHA)¹⁰⁾, the most effective way of reducing death from pulmonary embolism is to institute a comprehensive institutional policy of primary prophylaxis depending on the risks for venous thromboembolism¹¹⁾.

Diagnosis

It is relatively difficult to diagnose pulmonary embolism accurately. Among patients who were diagnosed as pulmonary embolism at autopsy, there were many under-diagnosed patients. The best accurate diagnosis rate in reports was 34%¹²⁾. According to the PIOPED¹³⁾ (Prospective Investigation of Pulmonary Embolism Diagnosis), 76 % of total patients were diagnosed with

intermediate or low probability scans. It is necessary to develop more accurate diagnostic method. In recent years, the usefulness of echocardiography and spiral CT from the chest to lower extremities was reported¹⁴⁾. Echocardiography is a rapid, practical, and sensitive technique for the identification of right ventricular overload after pulmonary embolism, and can be done at the patient's bedside⁴⁾. Spiral CT scan shows proximal pulmonary emboli and venous thrombi. For patients with chest pain or dyspnea, it is important to detect hypoxia, hypocapnia, or right ventricular dysfunction from blood gas analysis, electrocardiograms, or echocardiograms. D-dimer was reported to be effective as a screening test for vein thrombosis, especially in patients with massive or moderate-large pulmonary embolism¹⁴⁾. In this study, all 12 patients who had their D-dimer measured showed high values.

Caval thrombosis is combined in about 15% of cases with venous thrombosis from the iliac to femoral vein¹⁵⁾. This can cause fatal pulmonary embolism, especially the free-floating type. Thrombectomy is effective against caval thrombosis, especially for the free-floating type. Anti-

coagulation therapy alone is not fully effective¹⁶⁾. Thrombectomy was performed on all 4 patients with the free-floating type.

Treatments

Therapy was selected according to the differences in symptoms. Goldhaber et al¹⁷⁾ defined 6 acute pulmonary embolism syndrome, 2 of which are associated with right ventricular dysfunction. Treatments for massive pulmonary embolism and moderate-large pulmonary embolism with right ventricular dysfunction involve heparin plus thrombolytic therapy or mechanical intervention. The treatments for patients without right ventricular dysfunction are heparin alone or combined heparin and NSAIDs (nonsteroidal anti-inflammatory drugs). Therapy depends on the right ventricular dysfunction and the presence or absence of contraindications for thrombolytic therapy. Patients with right ventricular dysfunction after pulmonary embolism have a worse prognosis⁴⁾. For right ventricular dysfunction after pulmonary embolism, there are three therapies; medical (thrombolytic therapy and inotropic agents), catheter based (suction embolectomy¹⁸⁾, thrombus pulverization, thrombus fragmentation, local mechanical thrombolysis, local pharmacologic thrombolysis), and surgical embolectomy^{19, 20)}. Urokinase and tissue-type plasminogen activator (t-PA) have been used for thrombolytic therapy. Two-hour 100mg alteplase (recombinant t-PA) infusion induced a faster hemodynamic improvement than the 12- and 24-h urokinase regimens, although the antithrombotic efficacy of alteplase and urokinase was similar at 12 to 24 h²¹⁾. Early improvement of hemodynamic disturbances is the primary objective of the treatment of massive pulmonary embolism, especially in the most critical patients. Bleeding was equally frequent in the two treatment groups²¹⁾. We should be take t-PA by choice for the most critical patients.

If patients with shock due to massive pulmonary embolism dose not improve after thrombolytic therapy alone, catheter-directed fragmentation may be an effective treatment. It could be done at patient's bed-side. Our all 3 cases recovered their circulatory condition. Transvenous catheter pulmonary embolectomy was also effective to lower pulmonary artery pressure and rapidly improve pulmonary flow compared with only fibrinolysis^{6, 18)}. However, not all patients can have their thrombi sucked; only those in parts of the thrombi. On the other hand, surgical embolectomy can remove the emboli in the proximal pulmonary artery and venous thrombi at the same time. Hypoxia with continuous hypotension is the indication for surgical em-

bolectomy of the ACCP Consensus Committee on Pulmonary Embolism Opinions²²⁾. We think pulmonary artery thrombi that obstruct the main pulmonary artery with right ventricular dysfunction should be considered as an indication for surgical embolectomy.

For patients with sudden cardiopulmonary arrest, cardiopulmonary resuscitation that can fragmentize the obstructive thrombi should be done. If this fails, there is a great need for percutaneous cardiopulmonary support (PCPS)²⁰⁾. However, PCPS alone is inadequate, and rapid surgical embolectomy is frequently necessary immediately after using PCPS.

Three patients had fatal pulmonary embolism recurrence after recovery from shock. It is important to attempt prophylaxis in patients at risk of pulmonary embolism, especially those with massive or moderate-large pulmonary embolism. Caval thrombosis or DVT, even without combined severe pulmonary embolism, may require a IVC filter. Recently, temporary IVC filters have been used in addition to the permanent type²³⁾. In the two patients whose temporally filters were taken out at embolectomy, thrombi were trapped by IVC filters and recurrence of pulmonary embolism was not clinically detected. However, temporary filters can generally be used for 10 to 14 days to avoid difficult removal. When temporary filters are taken out, adhered thrombi need to have fibrinolysis. Taking out the thrombi trapped by a filter at embolectomy is safe. Permanent IVC filters have improved recently; complications have decreased with filters such as the Greenfield Titanium^{6, 24)}. Greenfield Titanium differs in the length and orientation of the attachment hooks and in the over-the-wire delivery from the percutaneous steel Greenfield filter. Greenfield Titanium was a low incidence of recurrent pulmonary embolism (3.5%) and a high caval patency rate (99%) after 1 year²⁴⁾. High-risk patients for recurrence should be actively considered for this type of filter.

Multivariate analysis showed that high grade of modified Greenfield's classification and right ventricular dysfunction were significant incremental risk factors. Immediately after onset of uncertain massive or moderate-large pulmonary embolism, right ventricular dysfunction should be evaluated by echocardiography and other clinical examinations. If right ventricular dysfunction is recognized, it should be treated as soon as possible. Patient prognosis can be improved with this therapy.

Conclusion

High grade of modified Greenfield's classification and right ventricular dysfunction were significant incremental risk factors for early death due to acute pulmonary embolism. Echocardiography was a rapid, practical, and sensitive technique for the identification of right ventricular dysfunction due to pulmonary embolism. If patients with massive pulmonary embolism can not improve severe right ventricular dysfunction only due to medical therapy, prompt catheter based therapy or surgical embolectomy are recommended. Moreover, combined surgical thrombectomy for free-floating type caval thrombosis and proximal massive pulmonary embolism was safe and effective. The use of IVC filter was effective to prevent the fatal recurrence of pulmonary embolism. If fatal pulmonary embolism could be predicted, first of all, the use of temporally IVC filter should be considered before treatments for pulmonary embolism or DVT.

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原著論文

急性肺塞栓症：現状と治療戦略

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要旨 重症急性肺塞栓症では、肺動脈の狭窄あるいは閉塞と血管収縮因子の放出により急性の右心室の後負荷増大と低酸素血症を引き起こし、10%の患者に突然死が起こるとされている。対象は1994年4月より1999年12月の期間に、当施設に急性肺塞栓症にて入院した58人（平均年齢59.2歳）で、うち35人は女性であった。下肢の静脈血栓症の促進因子としては、術後状態、長期安静、肥満等があった。7名は下大静脈血栓症を伴い、うち4例は浮遊型であった。重症例での初期診断は、超音波検査やCT検査が有用であった。超音波検査は迅速で、ベッドサイドにて実施可能であり、急性肺塞栓による右心不全や深部静脈血栓の診断に有用であった。治療法は7例に外科的血栓摘除術、3例にカテーテルによる肺塞栓破砕術、20例にヘパリンを併用した血栓溶解療法、22例には抗凝固療法のみを行い、6例では昇圧剤等などの一般的治療のみ施行した。発症後28日以内の早期死亡は全体の17.2%であった。死亡原因は2例で高度ショック、5例で右心不全、2例でDICを伴った多臓器不全、1例は蘇生後症候群であった。下大静脈フィルターは計9例に使用し、3例は一時的、6例は永久的フィルターを使用した。静脈フィルター使用例では観察期間中、臨床的再発は見られず、合併症も認めなかった。致死肺塞栓の可能性のある場合には、下肢深部静脈血栓症や肺塞栓症に対する治療以前にまず一時的な下大静脈フィルターを使用すべきであると思われた。多変量解析にて、修正Greenfield分類と右心不全が早期死の有意な危険因子であった。広範型肺塞栓症例で薬物療法のみで重症右心不全が改善しない場合には、迅速なカテーテル治療あるいは外科的血栓摘除術が推奨される。また、浮遊型の下大静脈血栓症と肺動脈中枢の肺塞栓の合併例では、同時に二ヶ所の血栓・塞栓の外科的摘除が安全で有効であった。

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