

Surgical Treatment of Pulmonary Embolism with Recent Intracranial Hemorrhage

Hidehito Endo, MD, Hiroshi Kubota, MD, Masaya Sato, MD, and Kenichi Sudo, MD

Massive pulmonary embolism (PE) with concomitant intracranial hemorrhage (ICH) has a high mortality rate. Although thrombolytic therapy is generally accepted as emergent treatment for massive PE, the risk of bleeding complications are significant. A 69-year-old woman presented with acute PE 11 days after onset of ICH. Thrombolysis was deferred, and emergent surgical embolectomy with cardiopulmonary bypass (CPB) was elected. Patient outcome was favorable without recurrence of ICH. (Ann Thorac Cardiovasc Surg 2005; 11: 256–9)

Key words: pulmonary embolism, intracranial hemorrhage, heparin, thrombolytic therapy, PCPS

Introduction

Despite recent advances in medical therapy, massive pulmonary embolism (PE) is still associated with a high mortality rate. Further, presence of massive PE with concomitant neurological disease is associated with even poorer outcomes. Although thrombolysis is generally accepted as first-line treatment for most patients with massive PE, the risk of bleeding complications are significant, and its use is contraindicated in a variety of situations, including pre-existing intracranial hemorrhage (ICH).

We describe a case of a patient with recent ICH who underwent successful embolectomy for acute massive PE.

Case Report

A 69-year-old woman presented with sudden onset of left hemiplegia and anarthria. Brain computed tomography (CT) scan revealed right thalamic and putaminal hemorrhage (Fig. 1A). The patient remained fully alert and oriented and was admitted for conservative therapy and continued observation. Her subsequent course was uneventful, and the patient commenced rehabilitation on post-

ICH day 4. However, on post-ICH day 11, the patient developed acute onset of extreme dyspnea after ambulation, and oxygen saturation was recorded by pulse oximetry as 65% on room air. Arterial blood gas analysis showed a PO₂ of 32.2 mmHg and a PCO₂ of 31.5 mmHg. Oxygen supplementation by face mask was initiated, and the oxygen saturation by pulse oximetry increased to 82%.

Hemodynamics were stable at the time of respiratory decompensation. A chest radiograph revealed a clear lung field without effusion or cardiomegaly, and an electrocardiogram revealed sinus tachycardia at 145 bpm without signs of right ventricular overload. Lung perfusion scintigraphy was interpreted as high probability for bilateral PE (Fig. 2A), and spiral contrast CT scan of the chest revealed a large saddle embolus at the bifurcation of the main pulmonary artery (PA), protruding into and nearly occluding the left and right PAs (Fig. 3).

After CT scan, arterial blood pressure suddenly decreased from 138/90 to 78/50 mmHg, and the patient became unresponsive. Endotracheal intubation was performed immediately, and dopamine hydrochloride, dobutamine hydrochloride and norepinephrine were initiated to augment cardiac output and blood pressure, but the patient's condition continued to worsen. Transthoracic echocardiography showed moderate tricuspid regurgitation, right ventricular dilatation and hypokinesis. Arterial blood gas analysis showed a PO₂ of 52.2 mmHg with 100% fraction of inspired oxygen. An intravenous bolus of heparin (3,000 units) was given followed by initiation of percutaneous cardiopulmonary bypass support (PCPS) to restore vital organ perfusion. Because of the patient's

From Department of Cardiovascular Surgery, University of Kyorin, Tokyo, Japan

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Address reprint requests to Hidehito Endo, MD: Department of Cardiovascular Surgery, University of Kyorin, 6-20-2 Shinkawa, Mitaka, Tokyo 181-8611, Japan.

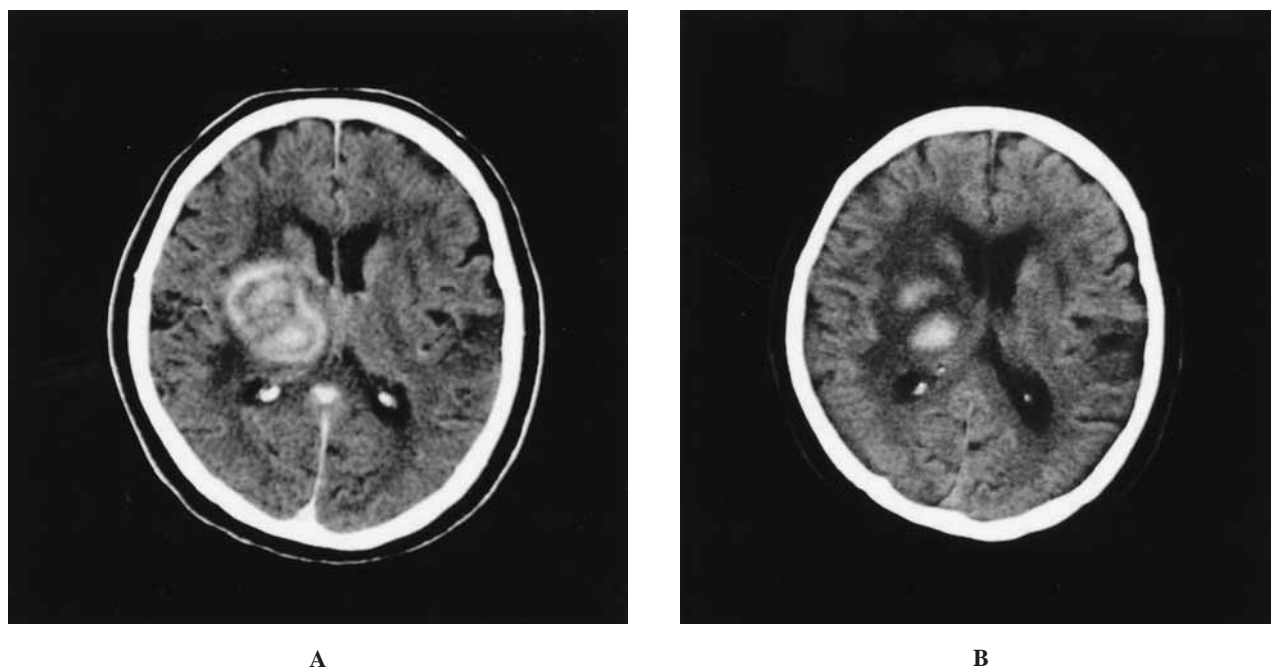


Fig. 1. CT scan of brain showing right thalamic and putaminal hemorrhage.
A: Preoperative CT scan.
B: Postoperative CT scan.

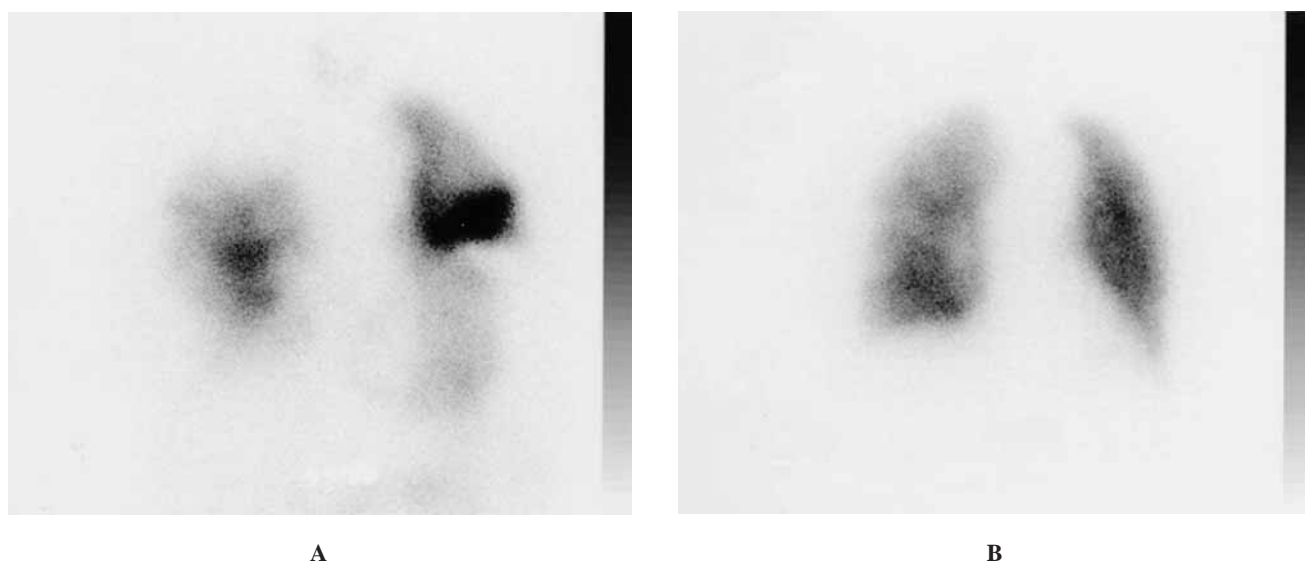


Fig. 2. Lung perfusion scintigraphy.
A: Preoperative scintigraphy.
B: Postoperative scintigraphy.

history of recent ICH, thrombolytic therapy was not performed.

After median sternotomy, heparinization (1 mg/kg) was continued cautiously secondary to history of recent ICH. Activated clotting time (ACT) was 453 sec, and no further heparin was administered. PCPS was converted to

cardiopulmonary bypass (CPB) by cannulation of the ascending aorta and both vena cava, and the left ventricle was vented through the right superior pulmonary vein. When the tympanic temperature reached 28°C, the ascending aorta was cross-clamped, and myocardial protection was achieved with repetitive doses of cold blood

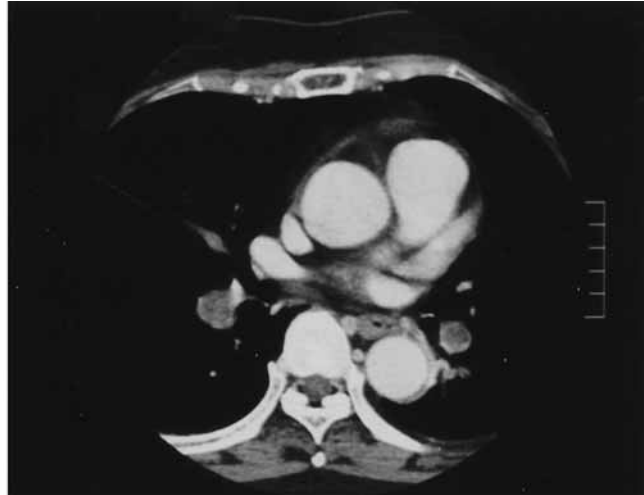
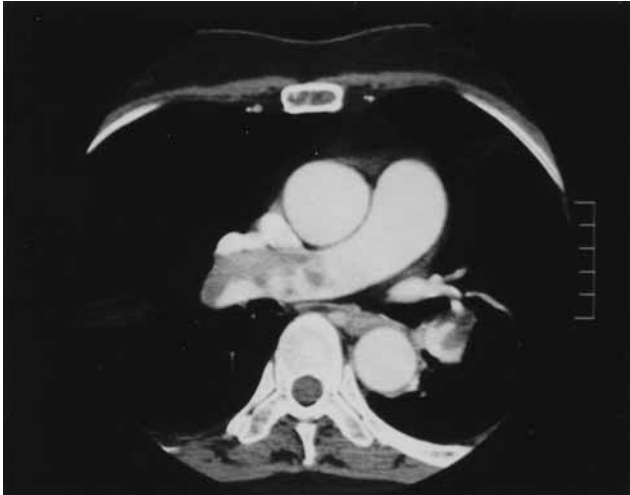


Fig. 3. Preoperative spiral contrast CT scan of the chest.

cardioplegia in an antegrade fashion. A longitudinal incision was made on the left PA, extending from the main PA to the pericardial reflection. The huge saddle clot was gently extracted en bloc under direct visualization. After dissecting the retro-superior vena cava (SVC), a longitudinal incision was made on the right PA, extending from the retro-aortic portion to the retro-SVC portion, by putting traction on tapes passed around the aortic and SVC, and the clot was removed under direct visualization. Residual thrombi were detected in the bilateral PA branches using a fiberoptic choledochoscope and were subsequently removed with forceps. However, thrombus in the left lower PA branch could not be removed. After warming, CPB was terminated and converted back to PCPS. Hemodynamic and respiratory conditions remained stable, and PCPS was weaned to off. Aortic cross-clamp time was 85 min, and operative time was 362 min.

The postoperative course was uneventful, and pulmonary hypertension gradually resolved. Extubation was performed on postoperative day (POD) 3. Unfractionated heparin (UFH; 15,000 units/day) was administered intravenously beginning on POD 3 to achieve adjusted activated partial thromboplastin time (aPTT) between 35 and 40. Oral anticoagulant treatment (sodium warfarin) was initiated in POD 5 and overlapped with UFH treatment for 3 days. Postoperative spiral contrast CT scan of the chest performed on POD 3 revealed removal of nearly all the thrombus. Brain CT scan performed on POD 4 demonstrated appropriate evolution and reabsorption of the ICH without recurrence or extension (Fig. 1B). Lung perfusion scintigraphy performed on POD 19 was inter-

preted as normal (Fig. 2B). The patient was transferred to another facility on POD 64 for ongoing rehabilitation. She was discharged with ongoing warfarin therapy, titrated to achieve an international normalized ratio of approximately 2.0.

Discussion

PE is a relatively common disorder in hospitalized patients and source of considerable morbidity and mortality. However, while mortality is approximately 30% for untreated PE, adequate treatment reduces the mortality rate to 2-8%.¹⁾ In the ICOPER study,²⁾ the overall mortality rate was 11.4% during the first two weeks after diagnosis of PE and 17.4% at 90 days. Guidelines for acute PE¹⁾ state that PE can be classified into two main groups: massive and non-massive. Massive PE consists of shock and/or hypotension (defined as a systolic blood pressure <90 mmHg or a pressure drop of ≥ 40 mmHg for 15 min if not caused by new-onset arrhythmia, hypovolemia or sepsis). A subgroup of patients with non-massive PE may be identified by echocardiography signs of right ventricular hypokinesis. This subgroup is called submassive, because there is growing evidence that the prognosis of this patient group may be different from those with non-massive PE and normal right ventricular function. It is thought that massive PE is defined by not diagnostic imaging but circulatory condition. In patients who were hemodynamically unstable secondary to massive PE at the time of presentation, the mortality rate increased to 58.3%.

PE with concomitant neurological disease is rare but

is associated with high mortality. Inci et al.³⁾ reported that the incidence of clinical PE among 6,081 operated neurosurgical patients was 0.42%, and the mortality was 59.4%, while Yamada et al. reported that the incidence of PE in 9,101 stroke patients was 0.45%, and the mortality was 39%.⁴⁾ Inci et al. described various etiologies for PE in patients with neurological disease, including glucocorticoid-dehydrating osmotic treatment, release of brain thromboplastin substances, limb weakness, advanced age and, in particular, immobilization.³⁾ Moreover, in patients with cerebrovascular pathologies (subarachnoid hemorrhage and ICH), the incidence of PE was high (3.57%), because of longer periods of immobilization secondary to neurologic disabilities.

The choice between thrombolytic therapy and surgical embolectomy for treatment of massive PE remains controversial. Guidelines for acute PE¹⁾ state that thrombolytic therapy is indicated in patients with massive PE (defined by the presence of shock and/or hypotension) and that acute pulmonary embolectomy has a limited role in massive life-threatening PE. However, Gulba and colleagues reviewed 37 consecutive patients with massive PE and shock and reported that the mortality was 23% for patients treated with surgical embolectomy and 25% for patients treated with medical therapy.⁵⁾ Further, the PE recurrence rate was 7.7% for surgical embolectomy and 21% for medical therapy. These data suggest that medically-treated patients have a higher death rate and an increased PE recurrence rate and are in contrast with the treatment guidelines.

The present patient was diagnosed with massive PE on post-ICH day 11, and surgical embolectomy was elected over thrombolytic therapy. While heparin therapy is clearly indicated in the setting of PE, the appropriate

use and timing of heparin therapy in the context of PE with concomitant ICH remains unknown. In this case, treatment of the life-threatening massive PE took precedence over the risk of recurrent ICH that accompanies heparin therapy and surgical embolectomy with CPB. However, heparin was given in one-third of the conventional dose to reduce the risk of recurrent ICH, and an ACT of greater than 400 sec was achieved.

In summary, surgical embolectomy was successfully performed in a patient with massive PE and pre-existing recent ICH. Although minimizing heparin therapy during CPB is advocated, further study to determine appropriate use and timing of heparin in this patient population would be of benefit.

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