A Case of Acute Massive Pulmonary Thromboembolism Treated by Mechanical Clot Fragmentation Using a Percutaneous Transluminal Angioplasty Balloon

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Large, bilateral central pulmonary thromboemboli (PTE) led to cor pulmonale and severe hypoxemia in a patient who had undergone Hardy's operation. After several unsuccessful efforts (thrombolysis using a percutaneous catheter and aspiration of the emboli), mechanical clot fragmentation using a percutaneous transluminal angioplasty (PTA) balloon was attempted. This procedure was successful, resulting in a decrease in pulmonary artery pressure from 58/22 (mean 34) mmHg to 20/10 (mean 13) mmHg together with an increase in aortic pressure from 64/36 (mean 45) mmHg to 112/60 (mean 77) mmHg. Thus, mechanical clot fragmentation using a PTA balloon is a promising method for reducing pulmonary artery pressure and increasing aortic pressure in patients with acute PTE.

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Key Words: Massive pulmonary thromboembolism; Mechanical clot fragmentation; Percutaneous transluminal angioplasty balloon; Inferior vena cava filter

Pulmonary thromboembolism (PTE) is a not uncommon disease in Japan. Life-threatening massive PTE associated with signs of circulatory collapse causing hypotension, right ventricular failure, or syncope requires rapid and effective therapy. Massive PTE is usually treated with systemic or local thrombolytic therapy^{1–4}. However, treatment failure is observed in 15–20% of PTE patients⁵ and the most severe cases result in death^{6,7}.

Here we describe how a PTA balloon proved to be useful for mechanical clot fragmentation in a patient with massive PTE who did not respond to thrombolysis or clot aspiration.

Case Report

A 70-year-old woman underwent Hardy's operation for a nonfunctional pituitary adenoma at our hospital on 11 December 1995. She complained of some shortness of breath during her first postoperative walk 14 days later. On 1 January 1996, she experienced

syncope for the first time about 3 min after developing severe, exercise-induced shortness of breath, and the neurosurgeon referred the patient to us. Laboratory examination revealed a PO₂ of 96 mmHg and PCO₂ of 30 mmHg during administration of 100% oxygen through a non-rebreathing mask, and systemic blood pressure was 84/38 (mean 53) mmHg. An echocardiogram revealed a dilated and hypokinetic right ventricle and severe regurgitation of tricuspid flow. Enhanced computed tomography (CT) revealed large obstructing central thromboemboli in the left and right pulmonary arteries (Fig 1). Heparin anticoagulation therapy (1,000 U/h) was administered and a Swan-Ganz (S-G) catheter was inserted into the pulmonary artery. Pulmonary pressure was 52/21 (mean 31) mmHg and the cardiac index was 1.98 L/min per m².

We planned to infuse 6,000 U of Pro-UK (Nasaru-plase) into the pulmonary artery through the S-G catheter over a 1-h period and, if there was no evidence of clot lysis, then an additional 960,000 U of urokinase was to be administered over 24 h (40,000

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U/h). However, after the intrapulmonary administration of 6,000 U of Pro-UK through the S-G catheter, pulmonary and systemic blood pressure were essentially unchanged [58/22 (mean 34) mmHg and 64/36 (mean 45) mmHg respectively]. Pulmonary arteriography demonstrated total occlusion of the right pulmonary artery and subtotal occlusion of the left pulmonary artery (Fig 2). The cardiothoracic surgeon consulting on this case deemed the risks of surgical embolectomy to be unacceptably high given the right ventricular failure and circulatory collapse with hypotension.

After obtaining informed consent from the patient and her family, we attempted catheter intervention. First, we tried to aspirate the thrombi using a percutaneous transluminal coronary angioplasty (PTCA) guiding catheter (8F Bright tip JR 4.0/Cordis) under a

Fig 1. Enhanced computed tomographic scan showing obstructing central thrombi within both the left and right pulmonary arteries.

standby percutaneous cardiopulmonary support system (PCPS) to create patent channels in both right and left pulmonary arteries in order to improve blood supply to the failed right ventricle. This was unsuccessful. Next, we tried mechanical clot fragmentation using a guide wire (0.035 Radifocus/Terumo), but again the results were unsatisfactory. Finally, mechanical clot fragmentation using a percutaneous transluminal angioplasty (PTA) balloon (8 mm×4 cm Ultra-thin, Medi-tech/Boston Scientific) was attempted. The PTA balloon was introduced into the main pulmonary artery through the 8F PTCA guiding catheter and the balloon was safely directed to the target lesion using a steerable Radifocus guide wire. The balloon was inflated several times (by hand) within the left and right pulmonary arteries (Fig 3). A pulmonary arteriogram taken after mechanical clot fragmentation showed 2 moderate residual thrombi remaining in the right interlobar artery and in the left upper lobe arteries, but re-establishment of blood flow to segmental branches of both lungs was evident (Fig 4). Pulmonary pressure fell to 20/10 (mean 13) mmHg, systemic blood pressure increased to 112/60 (mean 77) mmHg, and the cardiac index increased to 4.86 L/min per m².

To prevent recurrence of PTE, a titanium Greenfield filter (Medi-tech/Boston Scientific) was placed in the inferior vena cava and heparin anticoagulation therapy was administered for 2 weeks, followed by warfarin sodium anticoagulation therapy, which is continuing. Pulmonary pressure and blood pressure have been stable since the catheterization, but improvement in PO₂ was slow. PO₂ in room air reached

Fig 2. Pulmonary arteriogram showing total occlusion of the right pulmonary artery and subtotal occlusion of the left pulmonary artery.

Fig 3. Catheter intervention during balloon inflation within the right pulmonary artery.

Fig 4. Pulmonary arteriogram obtained after mechanical clot fragmentation showing 2 moderate residual thrombi remaining in the right interlobar artery and in the left upper lobe artery but re-establishment of blood flow to segmental branches of both lungs.

Fig 5. Enhanced computed tomographic scan obtained 3 months after catheterization showing no filling defect within either pulmonary artery.

78 mmHg 2 months later and supplemental oxygen was discontinued. Enhanced CT, performed 3 months after catheterization, detected no filling defect within the left and right pulmonary arteries and both pulmonary arteries were smaller than 3 months earlier (Fig 5). At the 6 months' follow-up the patient was fully active and showed no symptoms of respiratory impairment. Furthermore, no other defect was detected.

Discussion

Thrombolytic therapy can be lifesaving in patients with massive PTE^{1,4,8} and should be considered as a therapeutic approach in patients with major PTE who have syncope, hypotension, severe hypoxemia, or

heart failure^{1,4,8,9} In the present case, we were guided by the dose level generally used in acute myocardial infarction and expected that 6,000 U of Pro-UK (Nasarpulse) and an additional 960,000/24 h of urokinase infused through the S-G catheter would suffice. The 6,000 U of Pro-UK was administered, but the patient threatened to take a turn for the worse before the Pro-UK could take effect. There are 3 possible reasons for this outcome. First, our patient's clot might not have been fresh; she first experienced shortness of breath 8 days previously, so the clot might have been in the pulmonary artery for 8 days or more. In this case, recombinant tissue plasminogen activator (rt-PA) might have been better than Pro-UK; Goldhaber et al¹⁰ found that rt-PA given intravenously over 2 h is more effective and safer than a 24-h regimen of urokinase for PTE patients whose symptoms occurred less than 14 days previously. They reported that, by the second hour of infusion, 82% of rt-PA-treated patients showed clot lysis compared with 48% of urokinase-treated patients. Second, more than 2 h might have been necessary for the patient to experience the maximum therapeutic effect of thrombolytic therapy. Third, it is possible that 6,000 U is too small a dose of Pro-UK in the case of massive PTE. The dose of Pro-UK that we used is the dose regimen established for patients with acute myocardial infarction. It is possible that a much higher dose of Pro-UK is needed for patients with massive PTE, which is why we intended to administer an additional 960,000 U of urokinase over a 24-h period.

As thrombolytic therapy proved to be unsuccessful, we concluded that surgical embolectomy would be needed to remove the proximal pulmonary emboli, but the cardiothoracic surgeon we consulted did not agree to the idea because of the high risk involved. Although the surgical mortality rate in hemodynamically stable patients ranges from 13% to 20%, it rises to 50% in patients with cardiopulmonary disease and hemodynamic instability^{11,12}

The efficacy of PCPS in treating cardiopulmonary failure after PTE has been detailed^{13,14} since Gibbon¹⁵ proposed the concept for the treatment of PTE in 1939. However, as Numaguchi et al¹⁶ discovered, the prognosis of patients with massive PTE receiving PCPS is poor as a result of bleeding complications. Indeed, although a previous patient with massive PTE achieved stable circulation with PCPS, the patient's condition deteriorated during preparation for surgical embolectomy because of a pulmonary hemorrhagic infarct and bleeding at the sites of vascular puncture for PCPS. We felt that the brain blood flow in our patient was sufficient, as she maintained a high level of conscious-

ness throughout. Thus, we believed that mechanical catheter-directed therapies might be more suitable than the introduction of PCPS to create patent channels and treat right ventricular failure as rapidly as possible.

In Japan, the usefulness of transvenous pulmonary embolectomy using a guiding catheter for PTCA^{17,18} and of mechanical clot fragmentation by guide wire¹⁸ for treatment of massive PTE had been reported. Inoue et al¹⁸ reported success rates for clot aspiration and mechanical clot fragmentation of 25% and 100% respectively. Thus, we attempted to aspirate thrombi using the PTCA guiding catheter and to fragment the clot with a guide wire, but with minimal success in each case.

Finally, we attempted mechanical clot fragmentation using a PTA balloon. Handa et al¹⁹ reported that a patient with subacute PTE who did not respond to drug therapy for 4 weeks was treated successfully by balloon angioplasty. In our acute PTE patient, we attempted clot fragmentation rather than angioplasty. This was successful and resulted in a decrease in pulmonary artery pressure from 58/22 (mean 34) mmHg to 20/10 (mean 13) mmHg and a concomitant increase in aortic pressure from 64/36 (mean 45) mmHg to 112/60 (mean 77) mmHg. Dispersal of the central clot into the peripheral pulmonary arteries substantially decreased pulmonary vascular resistance and increased pulmonary blood flow because the volume of pulmonary emboli was much less than the total volume of the pulmonary peripheral vessels. Indeed, fragments of clot could have produced distal microembolisms and resulted in a less than maximal increase in PO₂, but we believe that the creation of patent channels and the treatment of right ventricular failure is more important. Furthermore, in our patient, the microemboli resulting from the clot fragmentation procedure were subsequently dissipated by anticoagulation therapy with heparin and warfarin sodium, so that 2 months later PO₂ had increased and supplemental oxygen was no longer needed.

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