

A Case of Chronic Pulmonary Embolism Successfully Treated by Thrombectomy

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ABSTRACT. A 48-year-old female complained of dyspnea and frontal chest pain five days after surgery for uterine cancer. The diagnosis was acute pulmonary embolism and she was treated with fibrinolytic therapy. Although the pulmonary embolism was reduced and hypoxemia was improved, there was still an unresolved pulmonary thrombus and pulmonary hypertension. Therefore, a cardiopulmonary bypass was performed under moderate hypothermia and without aortic cross-clamping. An organizing thrombus was revealed in the bilateral pulmonary arteries distal to and across from the mediastinal pleura, and a thrombectomy was performed. The postoperative course was uneventful. Postoperative pulmonary arteriography revealed some residual thrombus, and improvement of pulmonary hypertension was noted on right heart catheterization.

Key words: chronic pulmonary embolism — thrombectomy

Acute pulmonary embolism is fatal disorder that must be diagnosed and treated immediately. Recently, the incidence of acute pulmonary embolism has been gradually increasing in Japan. Chronic pulmonary embolism is defined as the occurrence of pulmonary hypertension and right ventricular failure due to an organizing residual thrombus. We report a case of chronic pulmonary embolism successfully treated by pulmonary thrombectomy.

A 48-year-old female underwent surgery for uterine cancer on September 5, 1995. Five days after operation, she suddenly complained of dyspnea and frontal chest pain. Blood gas analysis showed hypoxemia (paO₂ 38 mmHg, paCO₂ 32 mmHg). A diagnosis of pneumonia was made and she was treated with antibiotics, but her symptoms did not improve. Pulmonary ventilation and perfusion scintigraphy led to a revised diagnosis of acute pulmonary embolism on October 11. Pulmonary arteriography suggested a massive thrombus in the bilateral pulmonary arteries (Fig 1). Right heart catheterization suggested severe pulmonary hypertension (pulmonary arterial pressure was 80/30 mmHg). Anticoagulant and fibrinolytic therapy was done, using heparin (10,000 unit/day, 7days), urokinase (360,000 unit/day, 7days) and

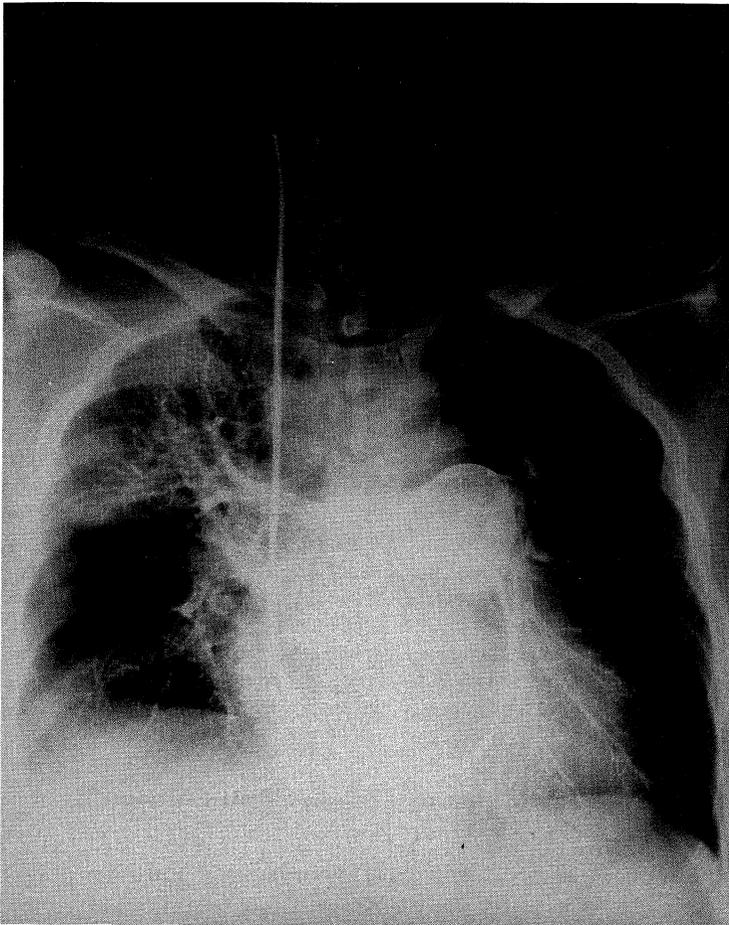


Fig 1. Preoperative pulmonary arteriogram suggested a massive thrombus in the bilateral pulmonary arteries

tissue plasminogen activator (600,000 unit/day, 3days). The thrombus decreased in pulmonary arteriogram. She was admitted to our hospital for an operation for residual thrombus on October 19. Her chief complaint was shortness of breath on effort. On physical examination, her blood pressure was 106/66 mmHg, heart rate 72/min, height 151 cm, body weight 68 kg. She had clear breathing sounds and no heart murmur. Hepatomegaly or dilatation of bilateral jugular vein was not shown. There was no history of deep vein thrombosis. The electrocardiographic findings showed right ventricular hypertrophy. A chest X-ray showed no vascular marking in bilateral lung fields (Fig 2). Analysis of blood gas values indicated increased PaO_2 (88 mmHg). Pulmonary pressure was 40/20 mmHg (mean pressure 32 mmHg).

The operation was performed on October 25. The patient underwent a median sternotomy and bilateral pulmonary arteriotomy on a cardiopulmonary bypass without aortic cross clamping (body temperature 25°C). No thrombus was revealed in the main pulmonary artery (PA) or the bilateral PA, but an

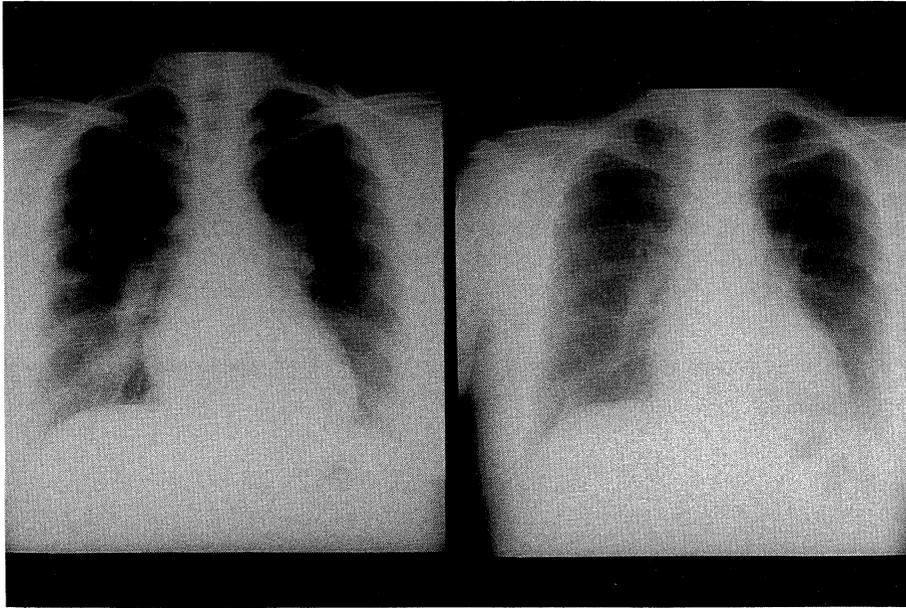


Fig 2. A preoperative chest X-ray film (left side) showed dilatation of a bilateral pulmonary arterial shadow and no vascular marking in the bilateral lung fields. A postoperative chest X-ray film (right side) showed more increment of bilateral vascular marking than preoperatively.

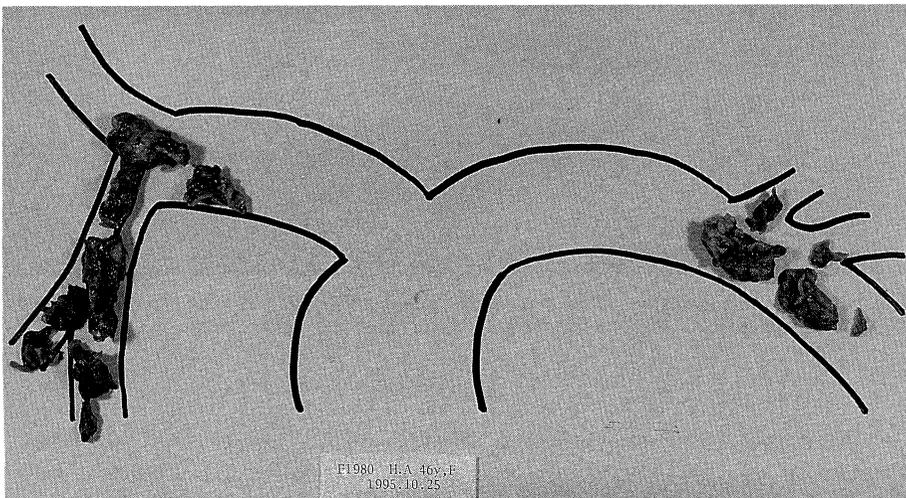


Fig 3. Macroscopic specimen resected from the pulmonary arterial lumen. The removed thrombus was a mainly whitish and organizing one.

organizing thrombus was found in the bilateral PA distal to and across from the mediastinal pleura. A thrombectomy was done using Kelly's forceps and Fogarty's thrombectomy catheter. The opening PA was closed with continuous sutures using 5-0 monofilament polypropylene suture. Pumping time was 129 min and intraoperative blood loss was 2457 ml. The removed thrombus was mainly whitish and an organizing one (Fig 3).

Postoperative PA pressure was 30/10 mmHg (mean pressure 22 mmHg), and her circulatory and respiratory state was stable. The postoperative course was uneventful. Bilateral pulmonary vascular marking was revealed in a chest X-ray (Fig 2). On right heart catheterization, main PA pressure was 24/10 mmHg (mean pressure 15 mmHg), right PA pressure was 28/6 mmHg, pulmonary capillary wedge pressure was 8/4 mmHg and cardiac output was 5.05 l/min. The postoperative pulmonary vascular resistance was 142.6 dyne·sec·cm⁻⁵. In a postoperative pulmonary arteriogram, the pulmonary arteries were recognized to be patent except for a part of the right middle and left upper lobes (Fig 4). The patient was given warfarin (3 mg/day) and beraprost(PGI₂, 120 µg/day) and was discharged on December 18.

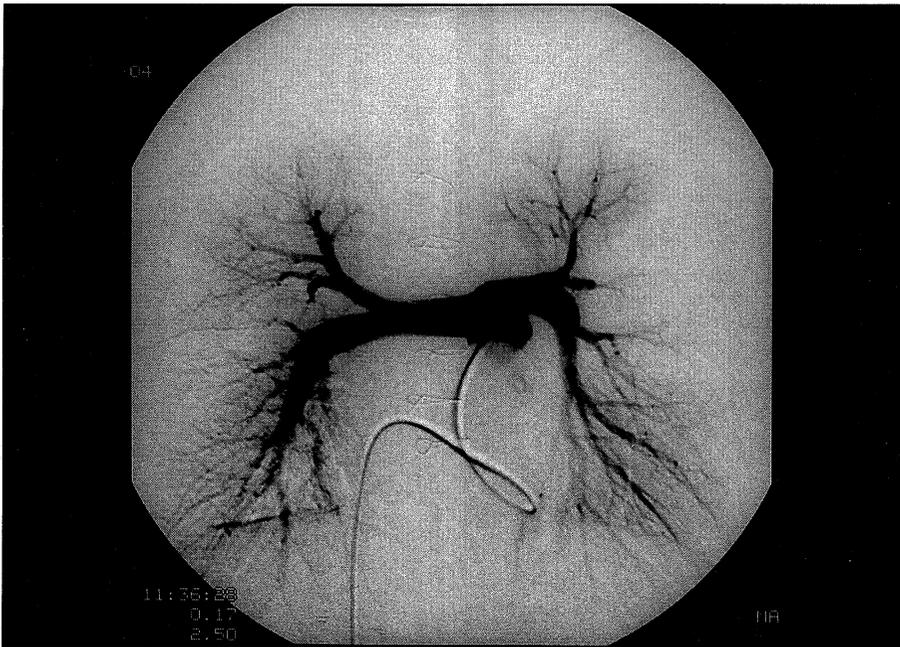


Fig 4. In a postoperative pulmonary arteriogram, pulmonary arteries were recognized to be patent except for a part of the right middle and left upper lobes.

COMMENT

Pulmonary thromboembolism mainly occurs as a result of deep vein thrombosis of the lower limbs and pelvis. Recently, its incidence has been gradually increasing in Japan. Acute massive pulmonary embolism is a fatal disorder that must be diagnosed and treated immediately.¹⁻³⁾ If there is no improvement in the hemodynamic disturbance and respiratory failure by conservative treatment, operative pulmonary embolectomy should be performed without delay. Gray and co-workers²⁾ reported an operative mortality rate for acute massive pulmonary embolism of 29.6% in 71 patients and Mayer and his co-workers³⁾ reported one of 37.5% in 96 patients. A recent report described a salvage case successfully treated by emergency surgery under a percutaneous cardiopulmonary support system before cardiac arrest.¹⁾

Chronic pulmonary embolism (CPE) is defined as the occurrence of pulmonary hypertension and right ventricular failure because of an organizing residual thrombus. The diagnosis of CPE is determined by pulmonary ventilation and perfusion scintigraphy, chest CT and right heart catheterization. The diagnosis of residual thrombus is performed by pulmonary arteriography. Pulmonary arteriography is important for determination of the operative indication. Anticoagulant therapy for CPE prevents formation of secondary thrombosis, but it does not resolve the organizing thrombus. Therefore, anticoagulant therapy cannot prevent progression of pulmonary hypertension and right ventricular failure.⁴⁾

The following operative indications for CPE have been reported⁵⁾; (1) chronic thrombi judged to be surgically accessible, (2) no life-threatening concomitant disease, and (3) a pulmonary vascular resistance greater than 300 dyne·sec·cm⁻⁵. Riedel and his co-workers⁵⁾ reported that the prognosis was poor in non-operative patients with a mean PA pressure above 30 mmHg (cumulative survival rate below 50%). Surgery is indicated for patients with a mean PA pressure greater than 30 mmHg. In our case, anticoagulant therapy was remarkably effective. Although pulmonary hypertension (mean PA pressure 30 mmHg) and a residual thrombus were present, we considered surgical treatment to be a necessity.

Two surgical techniques are used for the treatment of CPE. An embolectomy in unilateral PA is generally performed under a lateral thoracotomy,⁷⁾ whereas bilateral PA is done under a median sternotomy and with a cardiopulmonary bypass.^{3,5,8)} A lateral thoracotomy is used to approach the obstructed arterial branches distally, but the frequency of alveolar hemorrhage is greater than with median sternotomy.^{7,8)} There is a 6-20% operative mortality rate in the current literature.^{4,5,8)} It is important that postoperative anticoagulant therapy should be continuous. If a patient has history of deep vein thrombosis and recurrent pulmonary embolism inferior vena cava filter should be set up.

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