—Report on Experiments and Clinical Cases—

Surgical Treatment of Free-floating Thrombi in the Right Side of the Heart in Patients with Pulmonary Thromboembolism


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Abstract

Floating right heart thrombi (FRHTs) are rare in patients with acute pulmonary thromboembolism (PTE). FRHTs are an extreme therapeutic emergency, and any delay in treatment could be lethal. Heparin, thrombolysis, and catheter therapy are used most frequently to treat acute PTE. Here we present three cases involving operative treatment of FRHTs associated with PTE. Diagnoses were made with echocardiography before proceeding to emergency surgery. Thromboembolectomy was performed on cardiopulmonary bypass. One patient became brain dead 10 days postoperatively owing to cardiopulmonary arrest before surgery. The two survivors were subsequently discharged home after uneventful recoveries. (J Nippon Med Sch 2006; 73: 33-37)

Key words: pulmonary thromboembolism, floating right heart thrombi

Introduction

Acute pulmonary thromboembolism (PTE) with free-floating right heart thrombi (FRHTs), although rare, is life-threatening. Prompt and appropriate treatment improve the prognosis. The choice of treatment, i.e., surgical thrombectomy or thrombolysis, remains controversial.

Methods and Materials

From November 2002 through February 2005, three patients were found to have acute PTE with FRHTs. Thrombolysis may cause dislodgement of a thrombus, resulting in additional pulmonary embolic events and hemodynamic deterioration. For these reasons, emergency surgical treatment was considered appropriate.

Case 1. A 55-year-old man suspected of having angina pectoris was admitted complaining of shortness of breath during exercise. Coronary and left ventricular angiography revealed nearly intact arteries and normal cardiac function. A few days later, a skin eruption developed over the entire body. Liver and renal function deteriorated with increased levels of aspartate aminotransferase (181 U/l) and alanine aminotransferase (131 U/l), and blood urea nitrogen (28.7 mg/dl), and creatinine (1.64 mg/dl), which were believed due to the use of
contrast medium. The patient also complained of a sensation of hungering for air. Transthoracic echocardiography revealed a mobile right atrial thrombus that had entered the right ventricle and resulted in severe tricuspid valve regurgitation and right ventricular dilatation (Fig. 1). The pressure gradient between the pulmonary artery and right ventricle was 100 mmHg, which was similar to the systemic pressure. A peripheral deep venous thrombosis (DVT) was found with echocardiography in the left lower extremity. A caval filter was placed in the inferior vena cava before emergent surgery.

Case 2. A 58-year-old woman suspected of having angina pectoris was admitted with concomitant dyspnea. Three weeks previously she had undergone hematoma evacuation of a hypertensive right putaminal intracranial hemorrhage and was recovering in the rehabilitation unit. A pulmonary artery angiogram revealed PTE (Fig. 2). Because of the previous intracranial hemorrhage, thrombolytic therapy was contraindicated. Catheter thrombectomy was performed in the left upper pulmonary artery at that time, and transesophageal echocardiography performed the next day and demonstrated that a large thrombus had crossed the foramen ovale (Fig. 3). A DVT was also observed with echocardiography in the left lower extremity. A caval filter was placed above the entrapped thrombus in the inferior vena cava. Anticoagulation therapy was not performed because of the recent cerebral hemorrhage.

Case 3. A 40-year-old man had undergone irrigation of an intra-abdominal abscess after appendectomy. While the patient recovered, severe dyspnea suddenly developed. Afterward blood pressure dropped suddenly, and the patient fell into a state of shock. Mechanical ventilation, intra-aortic balloon pumping, and percutaneous cardiopulmonary support were begun to maintain the cardiopulmonary function while resuscitation was attempted. Transthoracic echocardiography revealed a FRHT in the atrium.

The operative procedures were performed...
Similarly in all three patients. The heart was exposed by a median sternotomy. With the start of cardiopulmonary bypass with aortobicaval cannulation and right upper pulmonary vein venting, the heart was arrested by cardioplegia. The right atrium was incised, and fresh thrombi were extracted. The main pulmonary artery was incised bilaterally, and the incision was extended to the left beyond the bifurcation. Extraction of the thromboemboli from the distal pulmonary vasculature was carefully performed with a forceps and suction tubing (Fig. 4). Hypothermic circulatory arrest was not used. The patent foramen ovale in case 2 was closed.

Results

Case 1. While the patient was being weaned from cardiopulmonary bypass, massive bleeding occurred from the right pulmonary artery suture line, which was repaired with a large pericardial patch. The tracheal tube was removed on the third postoperative day. For anticoagulant therapy, a dose of 20,000 units of heparin a day was continued for 20 days, after which oral warfarin and antiplatelet therapy was begun. Many perfusion deficits were observed in both lungs 2 weeks later, and a complete absence of the tracer was observed in the upper lung field (Fig. 5). Pulmonary arterial pressure measurement using a Swan-Ganz catheter on the 34th postoperative day showed that the pulmonary arterial pressure had normalized (28/13 mmHg when the systemic pressure was 110/70 mmHg). Finally pulmonary perfusion markedly improved, and no significant filling defects were observed.

Case 2. Weaning from the cardiopulmonary bypass was uneventful. The tracheal tube was removed on the 5th postoperative day, and oral warfarin therapy was started on the 6th postoperative day. Three weeks later lung perfusion deficits were observed only in the periphery of the right upper lobe. The patient was hospitalized for 3 months to undergo rehabilitation after intracranial hemorrhage. She was subsequently discharged home after an uneventful recovery.

Case 3. An emergency operation was successfully performed. However, the patient never regained consciousness because of brain damage caused by hemodynamic shock in. Although the respiratory status had transiently improved, the hemodynamic condition deteriorated and the patient died 10 days postoperatively owing to the previous brain damage.

Discussion

FRHTs are rare and are usually diagnosed when echocardiography is performed in patients with a suspected or proven pulmonary embolism. FRHTs are present in 7% to 18% of patients with PTE.
Moreover, the overall mortality is 40% or more and the first-day mortality rate is 21%\textsuperscript{14}. FRHTs present an extreme therapeutic emergency, and any delay in treatment could be lethal. Thus, FRHTs require emergency treatment, but there is no clear consensus regarding the most appropriate method.

Heparin, thrombolysis, and catheter therapy have been used most frequently for acute PTEs, whereas surgery has been selected most frequently for chronic thromboembolic pulmonary hypertension\textsuperscript{7}. In general, thrombolysis may be advocated as a first-line therapy for a PTE. It may favorably affect outcomes in patients with acute massive PTEs. Its theoretical advantages are numerous. It accelerates thrombus lysis and pulmonary reperfusion and reduces pulmonary hypertension\textsuperscript{36}. In contrast, in the case of FRHTs, death frequently occurs when the thrombus enters the pulmonary trunk\textsuperscript{25}. Thus, the thrombus must be extracted from the right heart immediately. Therefore, we recommend that when FRHTs with PTE are detected with echocardiography, thrombectomy is necessary with or without cardiopulmonary bypass. The use of cardiopulmonary bypass has an advantage over thrombolysis therapy. FRHTs can be carefully extracted from the atrium and evacuated from the pulmonary vasculature at the same time\textsuperscript{310}. Successful thrombolysis of FRHTs and PTEs has been accompanied by severe adverse events. Thrombolysis may cause dislodgement of a fixed thrombus into the pulmonary artery trunk, and treating them could result in an additional PTEs, hemodynamic deterioration or death. Massive PTEs result in circulatory collapse\textsuperscript{41}.

The thrombi observed were characteristic of venous emboli origin. FRHTs can travel from the legs to the pulmonary arteries. These three patients had DVTs. The DVT arose from compression of the iliac vein during the previous manipulation of the intrapelvic organs during surgery for colon carcinoma in case 1. The DVTs arose owing to long-term recumbence in cases 2 and 3. We recommend that anticoagulant therapy are used to prevent PTEs in patients who already have had a DVT or undergo long-term recumbence. In particular, in case 2, a large thrombus had crossed the foramen ovale. Paradoxical thromboembolism through a patent foramen ovale did not occur at that time. The thrombus could have paradoxically entered the systemic circulation. Such an event is life-threatening.

Echocardiographic examination in all cases of suspected PTE must be performed quickly so that emergency treatment can be started in patients with atrial FRHTs. Echocardiography revealed right ventricular dilatation and extremely mobile floating structures in the right atrium. Serial echocardiographic examinations are useful when the clinical status deteriorates because they may demonstrate thrombi that were not detected on the initial examination.

**Conclusion**

FRHTs present an extreme therapeutic emergency. Thrombolysis may cause the dislodgement of a fixed thrombus, resulting in an additional PTEs, which can cause circulatory collapse. The use of cardiopulmonary bypass has an advantage over thrombolysis therapy because thrombi can be safely removed from the right atrium and the pulmonary vasculature at the same time.

**References**

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