Pulmonary Vein Thrombosis Treated Successfully by Thrombectomy After Bilateral Sequential Lung Transplantation: Report of a Case

ITARU NAGAHIRO1, MATTHEW HORTON2, MICHAEL WILSON2, JAYME BENNETT2, PHILIP SPRATT2, and ALLAN R. GLANVILLE2

1 Department of Cancer and Thoracic Surgery, Okayama University Medical School, 2-5-1 Shikata-cho, Okayama 700-8558, Japan
2 Lung Transplant Unit, St. Vincent’s Hospital, Sydney, Australia

Abstract
We report the case of a 35-year-old man in whom an acute pulmonary vein thrombosis developed following bilateral sequential lung transplantation for cystic fibrosis. The thrombus was detected by transesophageal echocardiography 12h after transplantation and an emergency thrombectomy was successfully performed.

Key words Pulmonary vein thrombosis · Lung transplantation · Thrombectomy

Introduction
Anastomotic pulmonary vein thrombosis is a potentially fatal complication of lung transplantation. We present herein a case of pulmonary vein thrombosis after bilateral sequential lung transplantation, which was successfully treated by thrombectomy, and review the available literature on this subject.

Case Report
A 35-year-old man with cystic fibrosis underwent bilateral sequential lung transplantation without cardiopulmonary bypass. The preoperative prothrombin time, partial thromboplastin time, and platelet count were normal. Preoperatively, the patient was given mycophenolate mofetil 1 g orally, as well as FK506 2 mg and methylprednisolone 500 mg intravenously. Heparin 5000 IU was given intravenously before ligation of the right pulmonary artery and veins. The donor lungs were thoroughly flushed, and the total ischemic time was 4h 25 min for the right lung and 6h 10min for the left lung. Immediately after reperfusion of the right lung, 10 parts per million nitric oxide inhalation was started. Postoperative immunosuppression consisted of FK506, mycophenolate mofetil, and methylprednisolone, given as standard doses. A chest X-ray taken immediately after the operation showed a unilateral diffuse interstitial opacity of the right lung. Initially, this was thought to be reperfusion injury, occasionally seen after sequential bilateral lung transplantation. However, a persistently high central venous pressure of 9–11 mmHg and a pulmonary capillary wedge pressure of 14–16 mmHg, aggravating hypoxia (arterial partial oxygen tension /H1100592 mmHg at oxygen fraction /H110050.6), and increasing interstitial opacity on chest X-ray (Fig. 1) raised the suspicion of pulmonary vein thrombosis. A transesophageal echocardiography (TEE) performed about 12h after the operation confirmed the presence of a bilateral pulmonary vein thrombosis. A large thrombus was seen in the origin of the right pulmonary veins, which propagated into the left atrium (Fig. 2). This thrombus appeared to produce a significant increase in inflow velocity across the pulmonary vein origin into the left atrium. There was also a small amount of left pulmonary vein thrombus at the level of the anastomosis. There was, however, no significant hemodynamic obstruction to the left pulmonary flow return. The patient underwent emergency surgical exploration. The chest was reopened, cardiopulmonary bypass was established, and antegrade blood cardioplegia was administered into the aortic root. The anterior wall of the right pulmonary vein anastomosis was opened. A large amount of thrombus appeared to fill the inflow of the left atrium. The thrombus extended into the left atrium, and there was no evidence of right-sided pulmonary venous narrowing or anastomotic stricture. With the anastomosis opened, and the thrombus removed, the
left pulmonary vein anastomosis was inspected from the right side. A small amount of left-sided thrombus was removed. There was no left-sided anastomotic stricture or narrowing. The right pulmonary vein anastomosis was reconstructed using a continuous technique with 4-0 polypropylene sutures. The patient was weaned from cardiopulmonary bypass without any difficulty. Intravenous heparin (30,000 U/day) was started after the second operation for systemic anticoagulation. TEE was performed the following morning, which revealed no residual pulmonary vein thrombosis, as well as normal cardiac chambers and normal contraction. The patient was weaned from mechanical ventilation on postoperative day 3. He noticed transient weakness of the left arm on postoperative day 3 due to a small cerebral infarct in the right occipital lobe, possibly caused by embolization from the pulmonary vein thrombus. This resolved, and he was discharged home on postoperative day 22 and returned for regular follow-up visits as an outpatient without any further complications.

Discussion

Anastomotic pulmonary vein thrombosis is an infrequently reported, but potentially fatal, vascular complication of lung transplantation. Although optimal therapy has not been established, it is suggested that small, nonobstructive thrombi may be managed conservatively, whereas larger, obstructive thrombi may require surgical thrombectomy. A case of successful thrombolysis by recombinant tissue plasminogen activator has also been reported. Although there have been several reports of patients undergoing emergency thrombectomy, unfortunately most of them succumbed. Commonly, a delay in diagnosis of pulmonary vein thrombosis resulted in a delay in therapeutic interventions, often with a poor and fatal outcome. The clinical presentation of pulmonary vein thrombosis, namely, hypoxemia and interstitial infiltrate in the transplanted lung, mimics other potential postoperative complications, such as infection, graft rejection, and reperfusion injury. Neurologic deficit due to cerebral emboli may be the sole manifestation.

Leibowitz et al. reported that the incidence of pulmonary vein thrombosis after lung transplantation may be as high as 25%, and emphasized the usefulness of TEE for its detection. Given this high frequency of pulmonary vein thrombosis, it could be recommended to perform TEE routinely in the early postoperative phase to exclude the occurrence of pulmonary vein thrombosis, even in the asymptomatic patient. In our patient, pulmonary vein thrombosis was detected early, prior to the development of severe lung edema due to complete venous obstruction or cardiac infarction due to coronary artery embolism. At this stage, there was minimal and reversible heart-lung injury, compared with the irreversible changes seen in patients with fulminant symptoms. This could be a major reason why we were able to rescue the patient by emergency thrombectomy.

It remains unclear what factors predispose patients to the development of pulmonary vein thrombosis after lung transplantation, except for stricture or stenosis of the left atrial anastomosis. In our patient, the preoperative prothrombin time, partial thromboplastin time, and platelet count were normal, but it is possible that he has a hereditary form of coagulopathy, such as insufficiency of protein C or protein S, which could have predisposed him to the development of pulmonary vein thrombosis. As routine prophylaxis for anastomotic