Pulmonary artery thrombosis caused by hyperinflation of the native lung six years after single lung transplantation for emphysema

Alessandro Baisi, MD, Mario Nosotti, MD, Ugo Cioffi, MD, PhD, Matilde De Simone, MD, PhD, Lorenzo Rosso, MD, Alessandro Palleschi, MD, Barbara Chella, MD, PhD, and Luigi Santambrogio, MD, Milan, Italy

Hyperinflation of the native lung is a widely known complication after single lung transplantation for emphysema.[1,2] This may be verified a few months or a few years after the transplant.[3] We report the case of a patient in whom hyperinflation of the native lung was first the cause of chronic respiratory insufficiency for compression of the transplanted lung and then of acute respiratory insufficiency because of pulmonary artery thrombosis.

Clinical Summary

A 49-year-old, former heavy smoker, oxygen-dependent patient underwent a left single lung transplant for emphysema in February 1999. The postoperative course was uneventful. Long-term immunosuppression was maintained with a regimen of cyclosporine (INN: ciclosporin), azathioprine, and steroids. Three months later the patient's respiratory function was excellent (forced expiratory volume in 1 second [FEV_1] = 58%, P_{O_2} = 87 mm Hg, P_{CO_2} = 37.9 mm Hg), and he returned to work and a normal lifestyle. One year after the transplant a decrease in FEV_1 (26% of the best postoperative value) was recorded. Results of a bronchoscopic study were negative for anastomotic stenosis. Viral and bacterial infections were excluded. On computed tomography (CT) a hyperinflation of the native lung was evident (Figure 1). Results of transbronchial biopsies (TBBs) were negative for acute rejection or bronchiolitis obliterans. A diagnosis of hyperinflation of the native emphysematous lung was made. After 6 months the FEV_1 was stabilized at 40% of predicted value, and the TBBs were negative for acute rejection or bronchiolitis obliterans. Lung volume reduction surgery (LVRS) on the native lung was proposed to the patient, but he refused because his respiratory function was still satisfactory and he was worried about undergoing a new operation. In the following 4 years, TBBs were repeated 5 times and were always negative. The hyperinflation of the native lung remained stable. Respiratory function decreased very slowly. The FEV_1 reached 38% of predicted value. The patient's lifestyle was still normal. On January 2005 the patient had a sudden onset of dyspnea. Physical examination results were negative. The x-ray film was unchanged. The P_{O_2} was 53 mm Hg, and the P_{CO_2} was 35.4 mm Hg. The electrocardiogram was normal. No sign of pulmonary infection was evident. The patient underwent a perfusion scan to plan an LVRS on the native lung that unexpectedly showed an area of hypoperfusion in the lower lobe of the left transplanted lung, suggesting pulmonary embolism. The D-dimer was 3450 (normal value < 300). The patient was therefore studied with multidetector CT angiography of the pulmonary arteries. A thrombus in the inferior interlobar branch of the left pulmonary artery was present, completely occluding the artery (Figure 2, 3 and 4). Sections were also made during the venous phase, and deep venous thrombosis of the legs and pelvis was excluded. The thrombosis was attributed to severe kinking of the pulmonary artery because of the displacement of the mediastinum. Heparin was administered to the patient, and then dicumarol. The respiratory function slowly returned to prethrombosis values. After 6 months, multidetector CT angiography shows no filling defect in the pulmonary artery. The patient is now scheduled for a LVRS of the native lung.

From the University of Milan, Department of Surgery—Thoracic Unit, Ospedale Maggiore Policlinico, Fondazione IRCCS, Milan, Italy.

Received for publication June 29, 2005; revisions received Sept 14, 2005; accepted for publication Oct 7, 2005.

Address for reprints: Alessandro Baisi, MD, Department of Surgery—Thoracic Unit, Ospedale Maggiore Policlinico, Fondazione IRCCS, Via F. Sforza 35, 20122 Milano, Italy (E-mail: alessandro.baisi@unimi.it).

J Thorac Cardiovasc Surg 2006;133:746-7

0022-5223/$32.00

doi:10.1016/j.jtcvs.2005.10.006

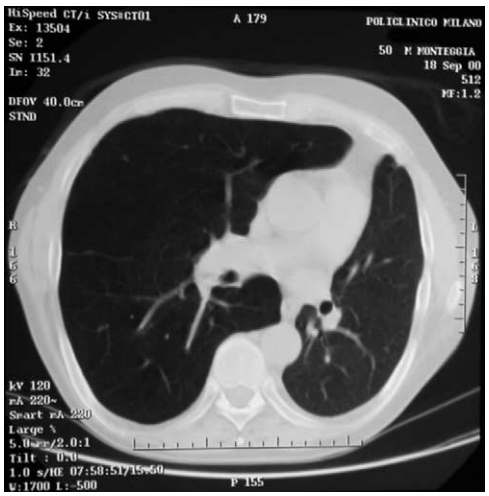

Figure 1. Chest computed tomography (CT) 1 year after the transplant. Hyperinflation of the native emphysematous lung and mediastinal shift toward the transplanted lung are evident.

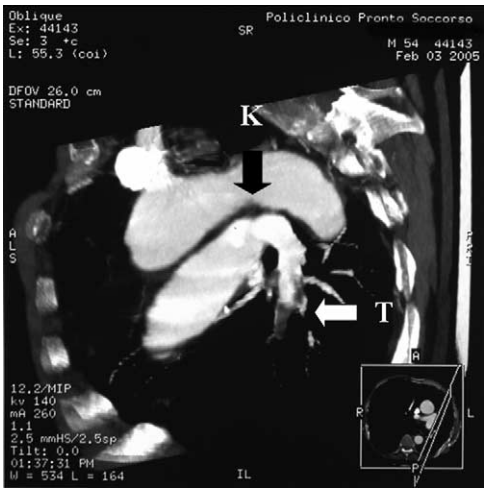

Figure 2. Contrast CT scan of the pulmonary arteries 6 years after the transplant. The hyperinflation of the native lung appears to be stable. However, a thrombus (*white arrow T*) is evident in the interlobar branch of the left pulmonary artery. Its kinking (*black arrow K*) because of the severe mediastinal shift is evident.

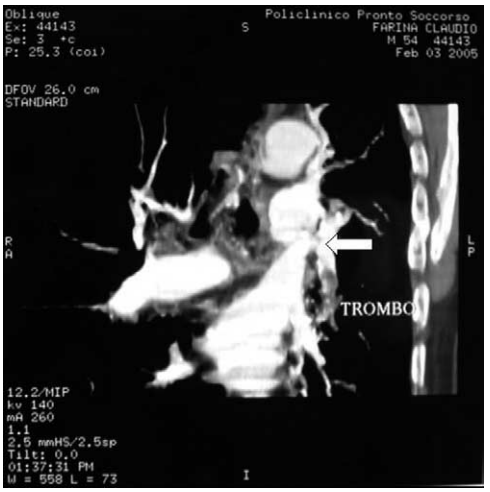

Figure 4. Contrast CT scan: frontal view of the pulmonary artery kinking (white arrow) and thrombosis.

Discussion

Routine use of bilateral lung transplantation in emphysematous patients is still debated.[1,4,5] In a single lung transplant, hyperinflation of the native lung has been reported as a cause of chronic respiratory insufficiency. It usually occurs in bullous emphysema; therefore, bilateral lung transplantation is recommended in these patients. The native lung hyperinflates and compresses the transplanted lung, impairing its function with a perfusion/ventilation mismatch. In our patient this hyperinflation of the native lung occurred after 12 months and remained stable over 4 years, as shown by CT scan and pulmonary function tests. The mediastinal shift toward the transplanted lung caused a kinking of the low-pressure vessels (eg, the pulmonary arteries and veins). The left pulmonary artery was so kinked that intraluminal thrombosis occurred. Other causes of thromboembolism were excluded by CT during the venous phase, which ruled out deep venous thrombosis. Thrombosis of the transplanted pulmonary artery can be a life-threatening complication, but in our patient it caused only an acute respiratory insufficiency because it occluded the interlobar branch. After 6 months of anticoagulant therapy, the thrombosis resolved with its symptoms. It is now imperative to re-alienate the mediastinum, reducing the hyperinflated native lung.

On the basis of this newly reported complication, we believe volume reduction of the native lung should not be delayed when it hyperinflates, which can cause a severe displacement of the mediastinum. LVRS is indicated to reduce respiratory insufficiency, because of the compression of the transplanted functioning lung, and to prevent the thrombosis of the pulmonary artery, which can be an acute life-threatening complication.

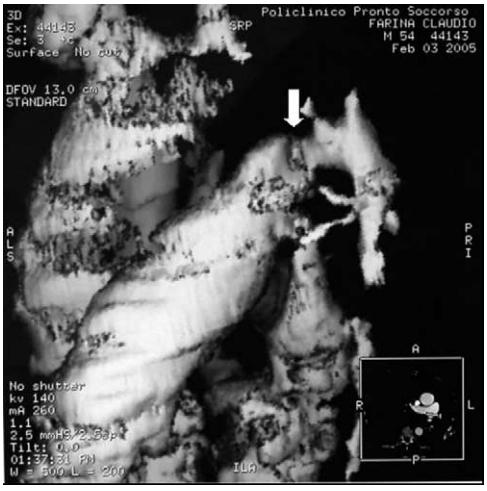

Figure 3. 3 D-angiographic reconstruction of multislice CT scan showing the kinking of the pulmonary artery (white arrow).

References

1. Malchow SC, McAdams HP, Palmer SM, Tapson VF, Putman CE. Does hyperexpansion of the native lung adversely affect outcome after single lung transplantation for emphysema? Preliminary findings. *Acad Radiol*. 1998;5:688-93.
2. Le Pimpec-Barthes F, Debrosse D, Cuenod CA, Gandjbakhch I, Riquet M. Late contralateral lobectomy after single-lung transplantation for emphysema. *Ann Thorac Surg*. 1996;61:231-4.
3. Fitton TP, Bethea BT, Borja MC, Yuh DD, Yang SC, Orens JB, et al. Pulmonary resection following lung transplantation. *Ann Thorac Surg*. 2003;76:1680-6.
4. Mal H, Brugiere O, Sleiman C, Rullon I, Jebrak G, Groussard O, et al. Morbidity and mortality related to the native lung in single lung transplantation for emphysema. *J Heart Lung Transplant*. 2000;19:220-3.
5. Sundaresan RS, Shiraishi Y, Trulock EP, Manley J, Lynch J, Cooper JD, et al. Single or bilateral lung transplantation for emphysema? *J Thorac Cardiovasc Surg*. 1996;112:1485-94.