among available tests, would have had the best chance of showing an early change on the valve. Transthoracic echocardiography at that time would not have helped since it was negative even in late January when the transesophageal study showed large vegetations.

There is a great shortage of organs for transplantation. Many potential recipients die because a suitable organ is not available. There is a natural tendency to maximally expand the donor pool and to relax exclusion criteria to the limits of safety. Regulatory requirements also dictate external determination of donor suitability so that potential donor opportunities are not wasted. Organs are now harvested from noninfected immunosuppressed patients and even harvested a second time if a recipient dies a noninfectious death without graft rejection. Organs are sometimes taken from donors with systemic bacterial infections when the infection is highly responsive to antibiotics and does not directly involve the donated organ. This outbreak serves to emphasize that there still is a limit to safety. Liver transplantation is associated with a higher risk of invasive aspergillosis than transplantation of other solid organs (4 to 10% in some series). Exclusion of subclinically present infection in distant organs may not be totally possible. Surveillance cultures positive for Aspergillus organisms should prohibit serial organ donation from a liver recipient, but as in this outbreak, the results may lag the organ transfer. This donor, although lacking specific donor exclusion criteria, was in retrospect too immunosuppressed to serve as an organ donor. Perhaps any liver transplant recipient at day 17 is too immunosuppressed to reliably exclude subclinically present infections and recipient risk from direct transfer of microorganisms.

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Noninvasive Diagnosis and Treatment of a Saddle Pulmonary Embolism*

A Case Report in Support of New Trends in Management of Pulmonary Embolism

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Transesophageal echocardiography and contrast-enhanced spiral CT of the chest helped to avoid a pulmonary angiography in an elderly patient with saddle pulmonary thromboembolism and allowed for direct evaluation of its resolution during treatment with subcutaneous low molecular weight heparin.

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LMWH=low molecular weight heparin; PE=pulmonary embolism; TEE=transesophageal echocardiography

Key words: computed tomography; low molecular weight heparin; pulmonary embolism; transesophageal echocardiography

Definitive confirmation of pulmonary embolism (PE) is still reserved for pulmonary angiography. Only then is...
aggressive treatment with thrombolysis or surgery considered fully justified. We present a case showing an alternative diagnostic approach as well as nonconventional treatment of a saddle PE in an elderly patient.

CASE REPORT

An 80-year-old man with a history of chronic atrial fibrillation that was not treated with anticoagulants presented with dyspnea of recent onset. A few weeks earlier, he experienced pain in his left leg. Physical examination on admission revealed tachycardia and slight swelling of the left calf. The ECG showed atrial fibrillation with a ventricular rate of 130/min with a left axis deviation (−30°). A chest radiograph showed minor pleural effusion. Signs of right ventricular pressure overload were found at Doppler echocardiography; the velocity of the jet of marked tricuspid regurgitation indicated right ventricular to right atrial systolic pressure difference of 48 mm Hg, and systolic flow velocity pattern in the right ventricular outflow tract was characterized by a very short acceleration time of 45 ms and was disturbed by mid-systolic deceleration.

In view of suspected PE, therapy with subcutaneous low molecular weight heparin (LMWH [Fraxiparine]) was started in an anticoagulant dosage (15,000 IU bid). Scintigraphy showed markedly diminished perfusion of the whole left lung with near-normal right lung perfusion. Transesophageal echocardiography (TEE) was performed to differentiate between PE and left pulmonary artery compression by aortic dissection or aneurysm. Despite unilateral scintigraphic changes, massive though nonocclusive thrombi were found at TEE in both pulmonary arteries. Spiral contrast-enhanced (CT) scans of the chest confirmed a large saddle embolus at the bifurcation of the main pulmonary artery, protruding to the left and right pulmonary arteries (Fig 1, top). There were no thrombi in the proximal veins of both legs as shown on duplex Doppler ultrasound examination. The decision was made to continue therapy with subcutaneous LMWH but to start thrombolysis without pulmonary angiography in case of hemodynamic deterioration. However, the patient remained stable, and within a few days dyspnea resolved. On the third day, the tricuspid systolic pressure gradient measured with Doppler echocardiography was the same as initially but acceleration time increased to 65 ms. A CT scan of the chest, repeated after 2 and 4 weeks using the same protocol and interpreted by the same observers, showed progressive reduction of the size of the thrombus, limited at that time to the left pulmonary artery (Fig 1, center and bottom). Similar findings were observed at the time of follow-up TEE.

The patient was discharged on a regimen of acenocoumarol in good clinical condition. He was free from echocardiographic signs of right ventricular overload, with only trivial tricuspid regurgitation, right ventricular to right atrial systolic pressure gradient of 25 mm Hg, and acceleration time of 80 ms.

DISCUSSION

Correct and prompt diagnosis of PE often is difficult because of the invasive and complex character of pulmonary angiography and the lack of specificity of pulmonary scintigraphy. In addition, the immediate availability of both methods in case of emergency generally is poor. A recent report indicates that thrombi can be unequivocally identified by TEE in the main, right, or left pulmonary artery in about 70% of patients with hemodynamically significant PE.1 TEE might be useful not only to confirm immediately the presence of proximal PE but also to exclude aortic dissection. This seems even more important in view of recent reports indicating that aortic dissection may mimic PE not only clinically but also in other ways: right pulmonary artery compression caused by the expanding aorta may result in scintigraphic and even angiographic signs interpreted as PE,2,3 leading to fatal consequences in the case of thrombolytic treatment.4

In our opinion unequivocal TEE visualization of a pulmonary artery thrombus justifies introduction of aggressive treatment especially in an unstable patient when there is a high clinical suspicion of PE. However, though a previous report suggests that TEE, while reasonably sensitive, seems
fully specific for the diagnosis of hemodynamically significant PE,1 confirmation of its findings with other methods should be encouraged until more evidence in support of existing data becomes available. In stable patients, a spiral or electron beam chest CT scan offers a new diagnostic option, which is probably more sensitive than TEE due to imaging more distal branches of the pulmonary artery.5,6 We considered confirmation of the presence of thrombi with two independent imaging methods a definitive, yet noninvasive diagnosis of PE. While our patient remained stable and quickly improved on subcutaneous LMWH, we would be able to introduce thrombolysis without further delay in case of hemodynamic deterioration. In addition, the patient would avoid hemorrhagic complications occurring at the puncture site in patients receiving thrombolysis after pulmonary angiography.7 In our case, CT, TEE, and scintigraphy images suggested that while the left “leg” of the saddle thrombus impeded flow, the right “leg” had no obvious impact on right pulmonary artery flow. Whether the angiographic catheter could have affected the hemodynamic status of the patient by fragmenting proximal pulmonary thrombi remains unclear. While fragmentation and distal migration of an occluding thrombus is hemodynamically beneficial, this might be the opposite in case nonocclusive proximal thrombi were fragmented, resulting in occlusions of multiple distal branches of the pulmonary artery.

Prompt clinical and hemodynamic improvement together with significant size reduction of pulmonary arterial thrombi as evidenced in our patient by imaging methods supports the efficacy of LMWH in pulmonary embolism.8 In comparison with unfractioned heparin, these compounds offer a fixed-dose regimen and the possibility of early ambulation. It is unclear whether prolonged action of LMWH might increase the risk of hemorrhagic complications had thrombolytic treatment been introduced due to hemodynamic deterioration.

Our case supports the clinical value of emerging diagnostic and therapeutic options in acute pulmonary embolism.

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