Paradoxical and Pulmonary Embolism
due to a Thrombus Entrapped in a Patent Foramen Ovale

A 70-year-old woman, with a history of recent thromboembolic stroke, was admitted to our hospital because of sudden dyspnea due to pulmonary thromboembolism. Trans-thoracic echocardiography revealed a tubular thrombus entrapped in a patent foramen ovale. Transesophageal echocardiography confirmed this finding and also revealed an atrial septal aneurysm. Because her cerebral status contraindicated surgical intervention, medical therapy with heparin and warfarin was started. Follow-up transthoracic echocardiography, performed 3 weeks after the initiation of therapy, revealed complete resolution of the thrombus. Medical therapy can be an alternative to surgical therapy in high-risk patients who have entrapped thrombi. (Tex Heart Inst J 2006;33:78-80)

Paradoxical embolism has been a challenging diagnosis, since its 1st description. Clinical diagnosis requires at least 2 of the following: deep venous thrombosis or pulmonary thromboembolism (or both); an intracardiac defect with right-to-left shunting; and an arterial embolism without a corresponding source in the left heart.1 Paradoxical embolism can be “proved” only if a thrombus entrapped in an intracardiac defect is demonstrated. Echocardiography has enabled antemortem demonstration of this rare and transient clinical condition. We report the case of a 70-year-old woman, with thromboembolic stroke and pulmonary embolism, in whom a thrombus entrapped in a patent foramen ovale was detected.

Case Report

In November 2002, a 70-year-old woman was admitted to our hospital because of severe dyspnea. One month earlier, she had suffered a debilitating thromboembolic stroke. Cranial computed tomography had revealed multiple ischemic zones. Echocardiography could not be performed at that time. She had a history of diabetes and hypertension. On examination, the patient appeared dyspneic. The vital signs were stable. On auscultation, heart sounds were arrhythmic and tachycardic, and there was no murmur. Pulmonary rales could be heard at the basal third of the right lung. She had left-sided hemiparesis. Her D-dimer level was 364 µg/L (upper normal limit: 375 µg/L). The electrocardiogram revealed sinus tachycardia with frequent supraventricular ectopy and a newly developed complete right bundle branch block. Transthoracic echocardiography, performed to evaluate the cardiac origin of the dyspnea and the previous stroke, showed a 5-× 1.5-cm tubular thrombus extending from the right atrium to the left atrium, through a patent foramen ovale (Fig. 1). The thrombus was mobile and, during diastole, protruded from the tricuspid valve into the right ventricle. The right chambers were enlarged, and the systolic pulmonary artery pressure was elevated. Transesophageal echocardiography confirmed these findings and also revealed an atrial septal aneurysm. The microbubble contrast study had negative results. Results of a ventilation–perfusion lung scan were consistent with bilateral pulmonary emboli (Fig. 2). A Doppler ultrasonogram of the lower extremities revealed thrombus in the right great saphenous vein. Oral anticoagulant therapy and intravenous heparin were started. Surgery was contraindicated because of the patient’s cerebral status; therefore, she was treated conservatively. Repeat transthoracic echocardiography, performed 3 weeks after the initiation of therapy, revealed complete resolution of the thrombus (Fig. 3), and the systolic pulmonary artery pressure was within normal limits. In addition, the
right great saphenous vein was patent in the follow-up Doppler ultrasonogram.

**Discussion**

Paradoxical embolism should be suspected in a patient who has systemic embolism, when signs of pulmonary thromboembolism are also present. A unique form of paradoxical embolism is impending paradoxical embolism, in which an embolic substrate originating from the venous system is entrapped in an intracardiac shunt; patent foramen ovale is the most common such conduit. Pulmonary embolism accompanies most cases of diagnosed paradoxical embolism. Usually, paradoxical embolism follows pulmonary thromboembolism, because elevated right-side pressures enhance right-to-left shunting through a patent foramen ovale. However, in our patient, pulmonary thromboembolism followed paradoxical embolism, and a transient right-to-left shunt might have occurred after a provocative event, such as coughing or a Valsalva maneuver. All patients with the diagnosis of paradoxical embolism should be investigated for the presence of deep venous thrombosis, which can be silent and can predispose the patient to pulmonary thromboembolism. If paradoxical embolism is suspected, transthoracic and transesophageal echocardiography are essential to the diagnosis, the latter with a contrast study for greater sensitivity.

Several therapeutic options exist for right heart thromboemboli. These are anticoagulation therapy, thrombolytic therapy, and surgical removal of the thrombus. Each of these has its own advantages and disadvantages, and the optimal means of treatment is yet unclear. Although cost-effective and easily available, anticoagulant or thrombolytic therapy can result in fragmentation (and further embolization) of the thrombus. Surgery has the unique advantage of providing an opportunity to close the patent foramen ovale, thereby preventing recurrent paradoxical embolism. This advantage may be offset by treatment delays or the risks of cardiopulmonary bypass. In a review of 49 patients who had thromboemboli entrapped in right heart chambers, the mortality rate was 50% with medical therapy and 15% with surgery. However, in a recent review of 177 cases of right heart thromboemboli, the mortality rate of patients undergoing anticoagulation with heparin was comparable to that of surgery. The mortality rate in an untreated group was 100%, and the thrombolysis group had the lowest mortality rate.

Thrombus entrapped in a patent foramen ovale is a rare form of right heart thromboembolism, and our knowledge is derived from reported cases. A recent review recommended initial systemic heparinization, followed by emergent surgical embolectomy when the
surgical risks are acceptable. In our patient, complete resolution of both intracardiac and deep venous thrombi was achieved by intravenous heparin and anticoagulant therapy, with no complications. There are similar cases in the literature. In conclusion, medical therapy can be a safe alternative to surgical therapy in high-risk patients who have an entrapped thrombus.

References
