Huge Atrial Thrombus
Causing Massive Pulmonary and Paradoxical Embolism via Patent Foramen Ovale

In August 2003, a 44-year-old woman suffering from postpartum dilated cardiomyopathy was referred to our coronary care unit with the complaint of sudden-onset dyspnea. She had been monitored since 1995 because of a diagnosis of primary antiphospholipid antibody syndrome. On admission to our hospital, the patient was in respiratory distress, severely cyanotic and hypotensive. Upon physical examination, her arterial blood pressure was severely depressed (70/40 mmHg), her heart rate was 110 beats/min, and her respiration rate was 35/min. Arterial blood gases demonstrated a PaO₂ of 40 mmHg and PaCO₂ of 35 mmHg, with respiratory alkalosis. Therefore, she emergently underwent mechanical ventilation. Her electrocardiogram showed sinus tachycardia at a rate of 110 beats/min, poor R wave progression in all precordial leads, Q waves in lead III, and deep S waves in lead I. We immediately performed bedside transthoracic (TTE) and transesophageal (TEE) echocardiographic studies, which revealed a large mobile serpentine thrombus, entrapped in a patent foramen ovale (PFO), in the right atrium. The thrombus also extended into the left atrium and protruded into the right ventricle during the diastolic phase of the cardiac cycle (Figs. 1 and 2). Significant tricuspid regurgitation was observed; from the measurement of tricuspid regurgitant velocity, we estimated pulmonary artery systolic pressure to be 60 mmHg.

Surgical thrombectomy was not attempted due to the patient’s hemodynamic instability and to the development of right hemiparesis from a presumed paradoxical embolus. The patient died despite all resuscitative attempts.

Comment

Floating right heart thrombi are uncommon but probably under-diagnosed in patients with pulmonary thromboembolism (PTE). Most echocardiographic reports of right-sided cardiac mobile thrombi “in transit” from the systemic venous system...
are found in case reports or case series in which clots were detected incidentally or during acute PTE. Echocardiographic examination is of great importance in managing patients with suspicion of massive PTE (as in the case presented here), because it might reveal not only thromboemboli trapped in the right or left heart chamber, but right chamber overload and right ventricular dysfunction. By using bedside TTE, we clearly demonstrated that floating right heart thrombi were entrapped in the PFO and were prolapsing into the right ventricle. One can surmise that right atrial pressure, increased by acute loading of the right ventricle, might cause floating thrombi to protrude into the left atrium via the PFO.

In conclusion, we believe that the coexistence of 2 prothrombotic diseases (postpartum dilated cardiomyopathy and antiphospholipid antibody syndrome) resulted in right atrial thrombus in our patient.

References


Fig. 2 Transesophageal echocardiographic images obtained from basal short-axis view show A) thrombus entrapped in patent foramen ovale and B) magnified image of thrombus.

LA = left atrium; RA = right atrium

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