Utility of Echocardiography and Serum Troponin Levels in Pulmonary Embolism

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A 39-year-old female was admitted with a several month history of progressive dyspnea and pleuritic chest pain. The history was significant for 2 packs/day tobacco abuse and a family history of "blood clots." Physical examination was noncontributory, and the electrocardiogram showed sinus tachycardia with a right bundle branch block. The serum troponin level was 0.10 ng/ml (normal is less than 0.05 ng/ml). Brain natriuretic peptide (BNP) levels were not measured. By transthoracic echocardiography, left ventricular function was normal; however, the pulmonary artery and right ventricle (RV) were enlarged, with a D-shaped ventricular septum noted in both systole and diastole (Fig. 1). Moderate-severe tricuspid regurgitation (TR) with a velocity gradient of 4.6 m/sec was measured. Using the modified Bernoulli equation, the tricuspid gradient

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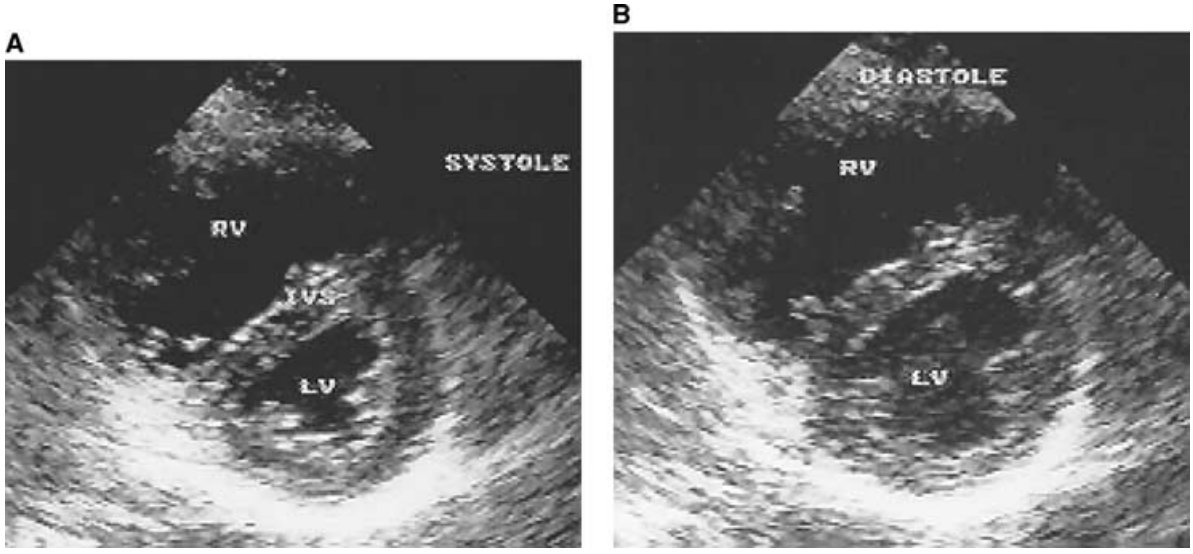

Figure 1. (**A**) *Systolic and* (**B**) *diastolic frames from a parasternal short-axis view. Interventricular septal flattening is noted in both frames. Systolic flattening is consistent with right heart pressure overload, and diastolic flattening with right heart volume overload.*

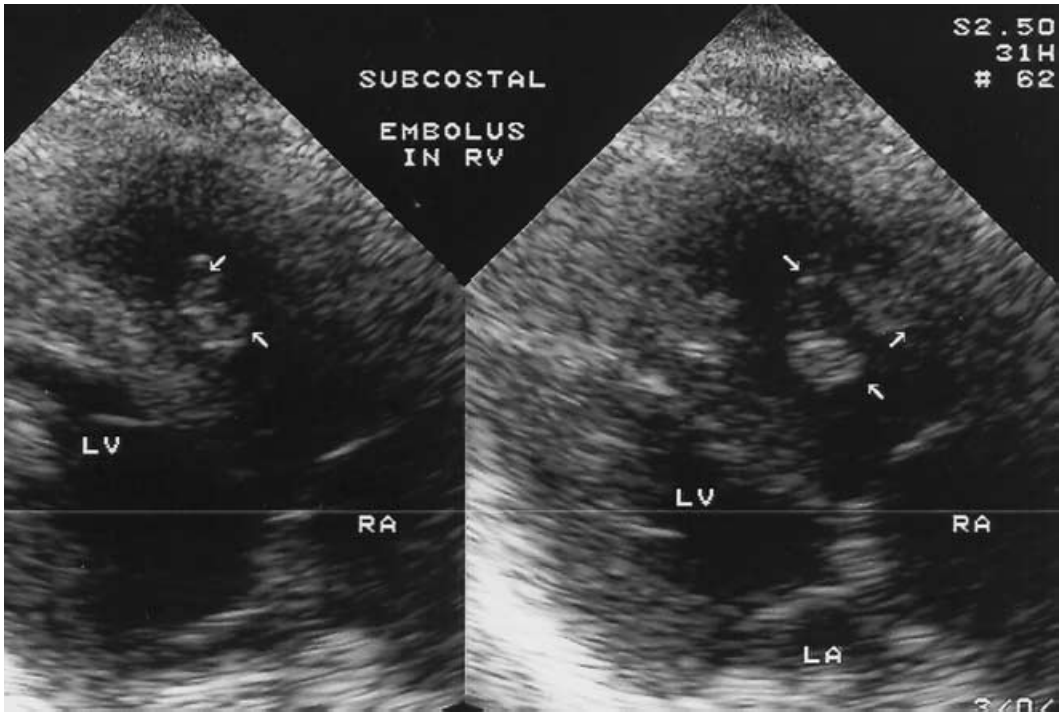

Figure 2. *Two sequential subcostal frames in a patient with acute massive pulmonary embolism. The right heart is dilated and there is a mobile mass "bouncing" within the right ventricular cavity (arrows). LA = left atrium; LV = left ventricle; RA = right atrium.*

was 85 mmHg, and the estimated right atrial pressure 10 mmHg, from evaluation of inferior vena cava (IVC) size with respiratory dynamics. Hence, the calculated systolic PA pressure was 95 mmHg. A ventilation-perfusion scan revealed a large mismatched perfusion defect in the upper lobe of the left lung, consistent with pulmonary embolism (PE). The patient was treated initially with intravenous heparin and subsequently with warfarin.

Based upon echocardiographic findings, PE may be categorized as either *acute massive*, *subacute massive*, or *minor*.[1] In patients without preexisting pulmonary hypertension, massive PE is associated with RV enlargement, dilatation of pulmonary vessels, RV free-wall hypokinesis with preservation of apical contractility, and partial/absence of collapse of the inferior vena cava during inspiration.[2] A mean PA pressure $\geq$20 mmHg is consistent with

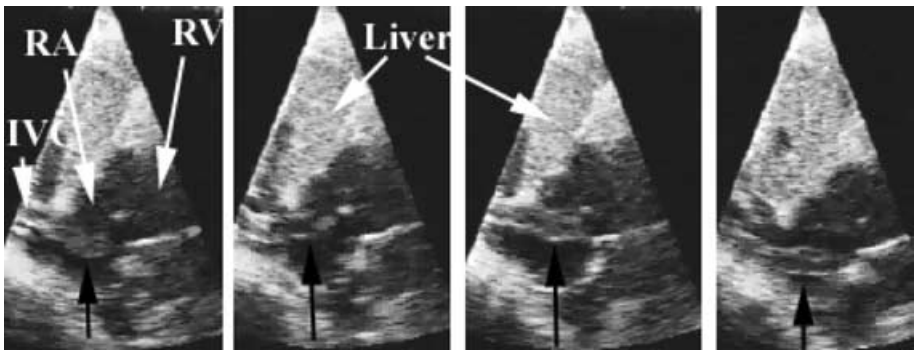

Figure 3. *Sequential subcostal images demonstrating a mobile thrombus with the distal end "stuck" in the IVC and its proximal mobile end in the RA (black arrows). IVC = inferior vena cava; RA = right atrium; RV = right ventricle.*

acute massive PE, but a mean PA pressure >40 mmHg suggests subacute massive PE. In addition, the RV wall is thinner in patients with acute PE than in those with subacute PE. With minor PE, PA pressure remains normal.[3] The presence of RV hypertrophy with dilatation should raise the possibility of an underlying chronic cardiopulmonary condition. Based on the patient's history and echocardiographic findings, our patient was categorized as having a subacute massive PE.

Thrombi that originate in the lower extremities usually appear as mobile "casts" of the veins, and may appear to "bounce" around the right heart chambers (Fig. 2) or IVC (Fig. 3). Intravenous leiomyomatosis, a vascular embolic tumor from the uterus, may have a similar mobile appearance within the right heart.[4] In patients with major pulmonary embolism, detection of a PFO may predict arterial embolic events from paradoxical embolism (Fig. 4) and an increased risk of death.[5]

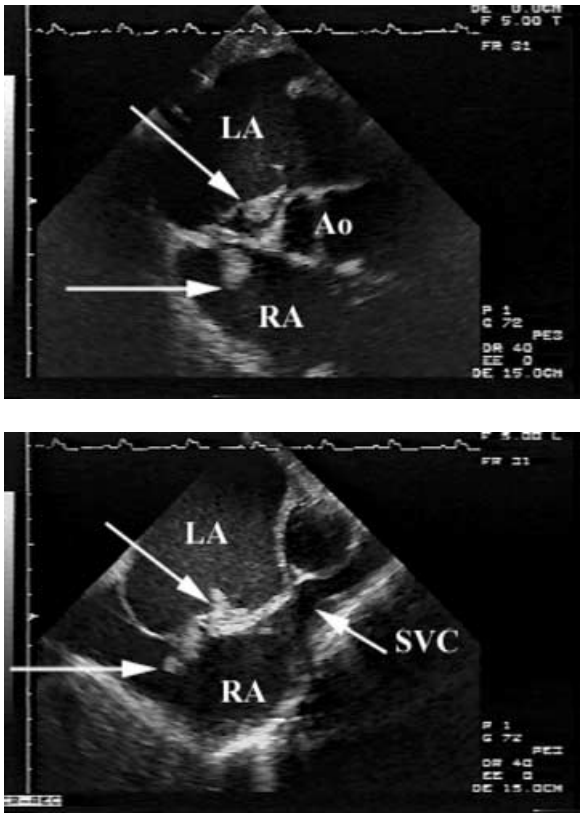

Figure 4. *TEE imaging* (**A**) *in the horizontal and* (**B**) *vertical plane demonstrating a thrombus (arrows) "jammed" in a PFO. The patient presented clinically with an acute pulmonary embolism and stroke. Ao = aortic valve; LA = left atrium; RA = right atrium; SVC = superior vena cava.*

It is important for the clinician to be aware that pulmonary embolism may be associated with elevated troponin levels.[6] The measurement of troponin levels has emerged as a promising tool for risk stratification.[7] Often a mild elevation of serum troponin levels due to acute right ventricular dysfunction will be noted. In normotensive patients with acute PE, elevated levels identify those at increased risk for a complicated clinical course and increased mortality.[8] Although BNP was not measured in our patient, a low level is predictive of an uneventful hospital course with acute PE.[9]

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