McConnell sign in a patient with massive acute pulmonary embolism

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CASE

A 69-year-old man with a history of hypertension and diabetes was admitted to the hospital for lower extremity weakness and recurrent falls of one month's duration. Magnetic resonance imaging of the brain showed a $6 \times 3 \times 4$ cm brain mass. Fifteen days prior to his admission, the patient underwent stereotactic brain biopsy with a right frontal craniotomy. He arrived from the nursing home with an altered mental status, was intubated in the emergency room, and was admitted to the intensive care unit (ICU). He had a heart rate of 140 beats per minute and a blood pressure of 90/60 mm Hg. Laboratory workup revealed WBC 11.9 10^9/L, lactic acid 4.1 mmol/L, and troponin 1.2 ng/mL. His electrocardiogram showed a sinus tachycardia but otherwise was within normal limits. Differential diagnosis at this time included septic shock and NSTEMI with cardiogenic shock. A central line was inserted, intravenous normal saline boluses were given, and the patient was started on broad spectrum antibiotics. Transthoracic echocardiogram was performed and revealed marked right ventricular (RV) dilation and a relatively small left ventricular chamber. McConnell's sign was noted (Video 1, Figure 1). The RV systolic pressure was 41 mmHg, the RVGLS was 5%, and the RV annulus S wave was 5.37 cm/sec. Doppler ultrasound of the lower extremities showed a non-occlusive thrombus in the left popliteal vein. Computed tomography of the chest showed extensive bilateral pulmonary emboli with a saddle embolus extending across the main pulmonary arterial bifurcation (Figure 2). The patient was started on a heparin drip; however, given his recent cranial surgery, the decision was made not to start systemic or catheter directed thrombolysis. After a prolonged hospital stay, the patient was eventually discharged to a nursing home.

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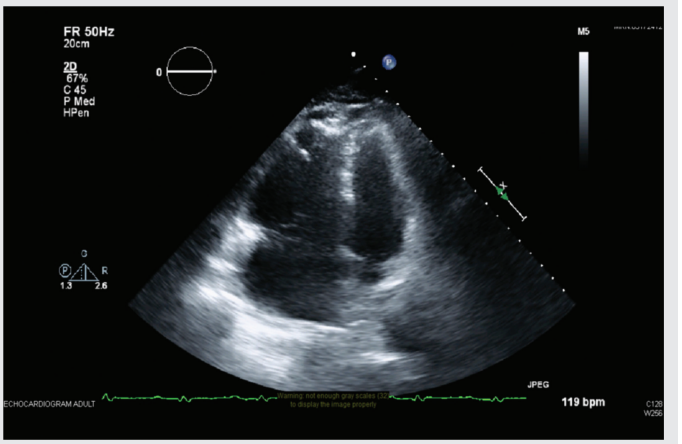

Figure 1. Echocardiographic image showing dilatation of the right ventricle and no movement of the right lateral ventricle wall in comparison to the left ventricle.

DISCUSSION

Pulmonary embolism (PE) can result in a catastrophic event with significant morbidity and mortality, especially when associated with RV dysfunction.[1,2] However, it is still often unsuspected and underdiagnosed. Depending on clinical presentation, the case

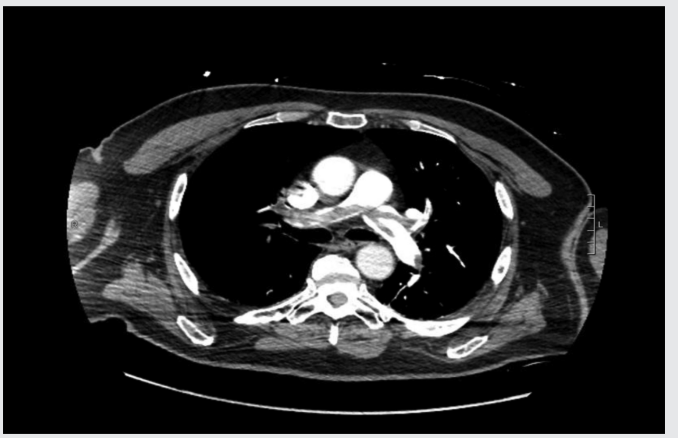

Figure 2. Computed tomography with contrast reveals a large saddle embolus.

fatality rate for acute PE ranges from less than 1% up to 60%.[3] Pulmonary embolism should be suspected in all patients who present with new or worsening dyspnea, chest pain, or sustained hypotension without an alternative or obvious cause. The diagnosis, however, is confirmed by objective testing in only about 20% of patients.[4] In acutely unstable patients, echocardiography can be highly suggestive of PE. Submassive PE has been suggested when RV hypokinesia is identified in otherwise hemodynamically stable patients.[5]

Echocardiographic RV dysfunction is indicative of a poor prognosis, and patients with this finding are at risk for subsequent clinical worsening and PE-related death. These patients may benefit from more aggressive therapeutic strategies, including thrombolytic therapy.[6,7] "McConnell's sign," defined as RV free wall hypokinesis in the presence of normal RV apical contractility, has 77% sensitivity and 94% specificity for the diagnosis of acute PE with a positive predictive value of 71% and a negative predictive value of 96%.[8] Casazza et al. demonstrated that McConnell's sign can also be seen in cases of RV infarction and thus cannot be considered pathognomonic for acute PE.[9] A pulmonary perfusion defect of at least 25% (moderate degree) is required for McConnell's sign to be demonstrated by transthoracic echocardiogram.[10]

Our patient had extensive bilateral pulmonary emboli and a saddle embolus leading to severe RV mid-free wall hypokinesis. Other parameters, including apical contractility, were otherwise within normal limits. Coupling the imaging findings with sudden cardiopulmonary decompensation increased our suspicion for submassive PE, which was confirmed by CT angiography of the chest. Normalization of the regional RV free wall dysfunction with thrombolytic therapy has been demonstrated in patients with massive PE.[11] Our patient was not a candidate for thrombolytic therapy as he was at high risk for major hemorrhage due to a brain neoplasm and recent brain surgery.[12]

Recognition of McConnell's sign and understanding that its pathogenesis involves a moderate-sized pulmonary perfusion defect provide critical care physicians additional information to reach an earlier diagnosis and improve outcomes in cases of acute pulmonary embolism.

Video 1: Echocardiogram demonstrating McConnell sign.

Video 2: Echocardiogram showing significant dilatation of the right ventricle with reduced right ventricular systolic function, flattened septum, large mobile vegetation of uncertain etiology on the tricuspid valve, and McConnell sign.

Keywords: pulmonary emboli, right ventricular dysfunction, McConnell's sign

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