Ultrasound in Emergency Medicine

MCCONNELL’S SIGN IS NOT SPECIFIC FOR PULMONARY EMBOLISM: CASE REPORT AND REVIEW OF THE LITERATURE

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Abstract—Background: McConnell’s sign (right ventricular [RV] free wall hypokinesis with apical sparing on echocardiography) is often described as very specific for the diagnosis of pulmonary embolism (PE). We present the case of a patient who, despite manifesting a classic McConnell’s sign, was not found to have a PE. Case Report: A 58-year-old woman presented to the emergency department with a cough, dyspnea, and leg swelling. A bedside focused cardiac ultrasound revealed hypokinesis of the RV free wall, with apical sparing, in the apical four-chamber view. A computed tomography angiogram for PE was negative. Ultrasounds of both lower extremities were negative for deep venous thrombosis, and a D-dimer was only marginally elevated. The patient was ultimately diagnosed with pulmonary hypertension due to chronic obstructive pulmonary disease and systemic lupus erythematosus. Why Should an Emergency Physician Be Aware of This?: Emergency physicians should be aware that McConnell’s sign is not completely specific for acute right heart strain from PE.

INTRODUCTION

Emergency physicians use focused cardiac ultrasound (FOCUS) to assess for a limited number of emergent conditions (1,2). Most “goal-directed” protocols for FOCUS in the emergency department (ED) include an assessment for signs of a pulmonary embolism (PE), such as right ventricle (RV) dilatation, RV hypokinesis, and septal flattening (3). One echocardiographic finding, RV free wall hypokinesis with apical sparing, or “McConnell’s sign,” has been described as very specific for the diagnosis of PE (4). We present the case of a patient with a classic McConnell’s sign but no evidence of PE.

CASE REPORT

A 58-year-old woman presented to the ED with a cough, dyspnea, and leg swelling. She described the subacute onset of “bronchitis” about 1 month earlier, consisting of a cough and transient subjective fever. She had been seen by her primary physician on multiple occasions, and had completed two courses of azithromycin, followed by two courses of ciprofloxacin, all without relief of the cough. Four days before her ED visit, she started having lower-extremity swelling, left greater than right. She was also increasingly dyspneic on ambulation. She denied any type of chest or back discomfort. She had a history of chronic obstructive pulmonary disease...
(COPD), systemic lupus erythematosus (SLE), and hypertension. Medications were albuterol, fluticasone propionate, and lisnopril. She described smoking 1 pack of cigarettes per day, but denied drugs or alcohol. Her vital signs showed her to be afebrile, but mildly tachycardic and hypoxic, with oxygen saturations that fell to 92% on room air. Lungs were clear, and no murmurs were appreciated. Both lower extremities were significantly edematous, the left greater than the right. An electrocardiogram (ECG) revealed shallow T wave inversions in leads III and V1–V3. The chest x-ray study was unremarkable. Complete blood count, basic electrolytes, and a troponin were within reference ranges. The pro-brain natriuretic peptide was elevated, at 9700 (>900 pg/mL).

The emergency physician performed a FOCUS. Left ventricular function appeared grossly normal, and no pericardial effusion or aortic root dilatation was seen on the parasternal long axis view (Video 1). On subcostal view, however, the RV appeared significantly dilated, with an end-diastolic RV diameter almost twice that of the left (Figure 1). Septal bowing during systole was observed in the parasternal short axis view (Figure 2 and Video 2). Akinesis of the RV free wall, with apical sparing (i.e., McConnell’s sign), was seen in an apical four-chamber view (Figure 3) (Video 3). The apical sparing was better visualized on a second apical view (Video 4). Of note, an echocardiogram from 4 years earlier was essentially normal. A computed tomography angiogram (CTA) for PE was ordered, and intravenous heparin was started before radiology interpretation. No pulmonary vascular occlusion was found on the CTA though, despite optimal opacification. Incidental note of a 7-mm pulmonary nodule was made, but otherwise there was no sign of PE, infiltrate, or other parenchymal abnormalities. The heparin infusion was stopped at this point. D-dimer was found to be only minimally elevated at 738 ng/mL (>500 ng/mL). Ultrasounds of both lower extremities were negative for deep venous thrombosis.

The patient was admitted to the medical service with a preliminary diagnosis of cor pulmonale secondary to either COPD or SLE. She received furosemide, ceftriaxone, and inhaled beta-agonists. She was initially admitted to the telemetry floor. While she remained alert and coherent, an arterial blood gases test obtained 2 days later found a PaCO₂ of 104 mm Hg and a PaO₂ of 44%. She was then transferred to the intensive care unit, where she received noninvasive ventilation. Before discharge, her PaCO₂ remained elevated at 82 mm Hg, with pH 7.37. A comprehensive echocardiogram was performed by the cardiology service 4 days after admission. A dilated, hypokinetic RV was again seen. Moderate tricuspid regurgitation was observed, and the RV systolic pressure was found to be severely elevated at 50 to 60 mm Hg.

**DISCUSSION**

In 1996, McConnell et al. described a characteristic echocardiographic finding of acute PE and RV free wall hypokinesis with preserved apical contractility (4). Further, they suggested that this finding could distinguish the acute right ventricular strain caused by PE vs. the chronic right ventricular strain caused by pulmonary hypertension (PH). Several studies have supported this initial result. Using a retrospective design, Lodato et al. studied the echocardiograms of 67 patients who had received a CT scan to evaluate for possible PE, and who subsequently received a transthoracic echocardiogram during hospitalization (5). More than 60% of these patients were shown to have a PE, and McConnell’s sign was found to be a very specific
finding. Similarly, a recent ED-based, prospective observational trial also found McConnell’s sign to be a specific predictor of acute PE. Dresden et al. analyzed emergency physician-performed echocardiograms in 146 patients at moderate to high risk of PE, and found McConnell’s sign to be 100% specific (6). Several reference works on echocardiography, accordingly, describe McConnell’s sign as a highly specific, if not very sensitive, echocardiographic sign of acute PE (7,8).

Not all studies, however, have confirmed the high specificity of McConnell’s sign. A 2005 study by Casazza et al. compared the echocardiograms of 201 patients who had been diagnosed with either acute PE or RV myocardial infarction (9). The prevalence of McConnell’s sign was equal in both groups, however, this study did not include subjects with the chronic RV strain. A small study by López-Candales et al. compared the echocardiograms of 10 patients with severe acute PE, all of whom manifested McConnell’s sign, with 10 patients with stable, chronic PH (10). They found that patients with PE and those with PH appeared to manifest an equivalent degree of RV apical strain. Vaid et al. conducted a retrospective study of 73 patients who had been found to have McConnell’s sign on echocardiogram; only about half of them were later confirmed to have an acute venous thromboembolism on subsequent duplex ultrasound, CTA, ventilation-perfusion (V/Q) scan, or other imaging study (11). Unfortunately, the investigators did not collect data on the final diagnosis in those patients who were subsequently shown not to have a PE.

Our patient was unlikely to have had undiagnosed acute PE. The CTA was well opacified, and had no motion artifact or other issues that limited interpretation. Furthermore, although the D-dimer exceeded the threshold set by our laboratory (500 ng/mL), the elevation was slight. Lastly, an ultrasound of the lower extremities was negative. Although each of these three tests has limitations, the combination of all three weighs heavily against acute PE as the cause of RV strain. RV enlargement and failure may occur due to a number of etiologies. RV infarction, with subsequent RV dysfunction, occurs frequently with acute occlusions of the proximal right coronary artery (12). This was unlikely in our patient’s case, given the normal troponin, and lack of suggestive ECG findings or left ventricular segmental wall motion abnormalities. Although it can be an imperfect test, her echocardiographic estimate of RV systolic pressure suggested moderate PH (13). PH of this severity can have many causes, among which are COPD and SLE (14,15). RV dysfunction may be seen in severe COPD, although outright RV failure is far less common (16). However, a subset of COPD patients can manifest acute RV failure, with characteristic peripheral edema and increased pulmonary mean artery pressures, during acute exacerbations of COPD (17).

Given the mixed data on the specificity of McConnell’s sign, it is important to clarify the role of FOCUS in the diagnosis and management of PE. Certain findings in a symptomatic patient, such as an RV diameter exceeding the LV diameter, may suggest the diagnosis of PE. However, the poor sensitivity of most echocardiographic signs of PE make them insufficient to rule out the diagnosis (1). Conversely, these indirect signs must be confirmed in all but the most unstable patients through other imaging tests (e.g., CTA, V/Q). Only in the most “severely compromised” patients should FOCUS be employed to justify treatment of presumed PE without further confirmation (1). Three publications in the emergency medicine literature report using a finding of McConnell’s sign during the diagnosis and management of PE. In a 2010 case report, Liao et al. described the presumptive diagnosis and treatment of PE in a patient who was initially unable to undergo a CTA (18). Given the significant tachycardia and tachypnea, the finding of a

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Figure 3. Apical four-chamber view. (A) In diastole, the right ventricle (RV) appears grossly dilated. (B) In systole, the left ventricular (LV) cavity is obliterated, while the right ventricular free wall appears akinetic. The arrows mark the apparent preserved contractility of the right ventricular apex. RA = right atrium.
McConnell’s sign prompted immediate anticoagulation. By contrast, Haller et al. used FOCUS only after having established the diagnosis of PE by means of CTA (19). The presence of McConnell’s sign, along with marked tachycardia, suggested the need for intensive care unit monitoring, per the authors. In a third case report, a persistently hypotensive patient was found to have a McConnell’s sign on FOCUS, and received thrombolysis for massive PE after the diagnosis of PE was confirmed by CTA (20). The authors do not specify the role that McConnell’s sign played in their evaluation and management, but they describe it as very supportive diagnostically.

While our patient with McConnell’s sign did not appear to have PE, the vital signs were stable, allowing further diagnostic evaluation. In a patient who presents with signs or symptoms of massive PE and is hemodynamically unstable, a McConnell’s sign still likely indicates PE and may be used to support aggressive treatment, such as systemic thrombolysis. However, in stable patients, other imaging modalities should be pursued before initiating the more aggressive therapies.

WHY SHOULD AN EMERGENCY PHYSICIAN BE AWARE OF THIS?

Despite having a classic McConnell’s sign on the focused echocardiogram performed by the emergency physician, our patient was ultimately found not to have a PE. Instead, her RV strain was likely from PH, resulting either from COPD or SLE. Emergency physicians should be aware that McConnell’s sign is not completely specific for acute right heart strain from PE.

REFERENCES


SUPPLEMENTARY DATA

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.jemermed.2014.12.089.