

Ultrasound in Emergency Medicine

EMERGENCY DEPARTMENT FOCUSED BEDSIDE ECHOCARDIOGRAPHY IN MASSIVE PULMONARY EMBOLISM

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Abstract—Background: Massive pulmonary embolism (PE) is a common consideration in unstable patients presenting to the emergency department (ED) with chest pain, dyspnea, or cardiac arrest. It is a potentially lethal condition necessitating prompt recognition and aggressive management. Conventional diagnostic modalities in the ED, including chest computed tomography angiography and ventilation-perfusion scanning, require the unstable patient to leave the department, and raise concerns over renal injury. Several case reports document findings of massive PE on echocardiography performed in the ED; however, none was performed, interpreted, and acted upon in the form of thrombolytic therapy by an emergency physician without the additional benefit of a cardiologist's interpretation or a confirmatory imaging study. **Objective:** We present a case that illustrates the utility of ED focused bedside echocardiography in suspected massive PE and briefly review direct and indirect ultrasound findings of acute PE. **Case Report:** A case of massive PE in a 61-year-old woman is reported. In this patient with marked dyspnea, progressive hemodynamic instability, and contraindications to definitive imaging, ED focused bedside echocardiography provided valuable information that strongly suggested the diagnosis and led to alteplase administration. To our knowledge, this case represents the first report of thrombolytic therapy administration for acute massive PE based solely on clinical

presentation and an emergency physician-performed bedside echocardiogram. **Conclusion:** In the hands of an experienced emergency physician ultrasonographer, ED focused bedside echocardiography provides a safe, rapid, and non-invasive diagnostic adjunct for evaluation of the patient suspected of having massive PE. © 2011 Elsevier Inc.

Keywords—emergency department; bedside focused ultrasound; echocardiogram; pulmonary embolism; thrombolytic

INTRODUCTION

Pulmonary embolism (PE) is a common diagnostic consideration for patients who present to the emergency department (ED) with chest pain, dyspnea, or both. “Massive” PE describes a hemodynamically significant PE. Chest computed tomography (CT) angiography is now considered the test of choice for diagnosis of PE, although concerns over potential renal injury from intravenous contrast and transportation of an unstable patient away from the ED limit its use in certain scenarios. Although the first issue is adequately addressed by ventilation-perfusion (V/Q) scanning, the latter remains a significant limitation.

Several prior case reports have documented echocardiographic findings leading to a diagnosis of PE in the ED or intensive care unit (1–9). To our knowledge, none of these echocardiograms was performed and interpreted by an emergency physician and guided thrombolytic

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therapy administration before confirmatory imaging or cardiology interpretation.

CASE REPORT

A 61-year-old woman (170 cm, 81 kg) with a history of multiple sclerosis presented to the ED with sudden-onset shortness of breath for 30 min. She denied chest pain and any past cardiac or pulmonary disease. She had no personal or family history of deep vein thrombosis (DVT), PE, or thrombophilia, and denied tobacco and illicit drug use.

Initial emergency physician (EP) evaluation revealed an ill-appearing woman in significant respiratory distress using accessory muscles of respiration. Presenting vital signs were: blood pressure 107/72 mm Hg, heart rate 144 beats/min, respiratory rate 22 breaths/min, SpO₂ 98% on non-rebreather mask, and temperature 36.5°C. The cardiac examination was tachycardic but regular, lungs were clear, and skin was cool and “dusky.” Initial electrocardiogram (ECG) showed sinus tachycardia at 142 beats/min with a right bundle branch block (RBBB), and chest X-ray study was normal.

The patient had a known severe contrast dye allergy and was not, therefore, a candidate for a PE-protocol CT scan of the chest. She was not stable enough to leave the ED for nuclear medicine testing. The EP considered a stat bedside transthoracic echocardiogram; however, efforts to summon an echocardiography technician or cardiologist to the bedside were unsuccessful. An on-duty EP colleague with additional training in emergency ultrasound performed a focused bedside echocardiogram, which demonstrated marked right ventricular (RV) dilatation, a small left ventricle (LV) with impaired filling, and abnormal interventricular septal (IVS) wall motion (Figure 1).

In light of the initial clinical presentation, worsening hemodynamic instability, a normal chest X-ray study, and the concordant focused bedside echocardiogram findings, the patient was presumed to have a massive PE, and an alteplase infusion was begun. One hour into the infusion, the patient appeared much more comfortable, with improved vital signs: blood pressure 130/70 mm Hg, heart rate 118 beats/min, and SpO₂ 100% on 4 L/min of oxygen by nasal cannula. Upon completion of the 2-h infusion, her blood pressure was 147/88 mm Hg, heart rate 103 beats/min, and oxygen saturation remained at 100% on 4 L/min of oxygen. An intravenous unfractionated heparin drip was started, and the patient was transferred to the medical intensive care unit.

On hospital day 2, repeat ECG showed resolution of the RBBB. Formal transthoracic echocardiography showed mild RV dilatation with moderately depressed RV systolic function and normal LV function with an LV ejection fraction of 60%. Pulmonary artery systolic

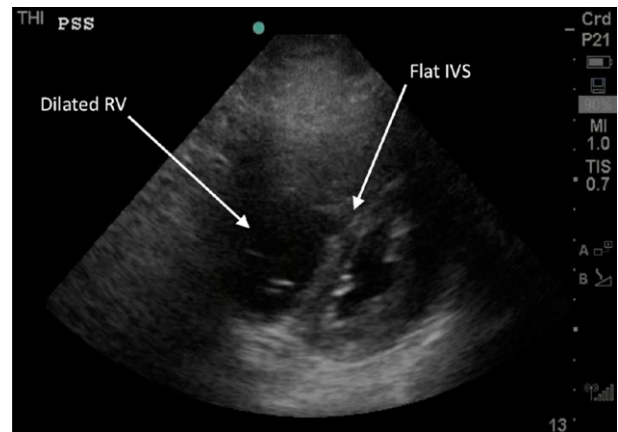


Figure 1. Patient’s parasternal short-axis view at end-diastole demonstrating interventricular septal (IVS) flattening and right ventricular (RV) dilatation.

pressure was estimated at 26 mm Hg. On hospital day 4, a V/Q scan revealed multiple moderate-to-large bilateral segmental and sub-segmental perfusion defects, yielding a “high-probability” interpretation. Bilateral venous duplex ultrasound scans demonstrated non-occlusive thrombus in the left popliteal vein.

After several consultations relating to complications of her multiple sclerosis, the patient was discharged home in good condition 2 weeks after her presentation.

DISCUSSION

This patient presented the treating EP with several dilemmas, and the acuity of her presentation demanded rapid diagnosis and management. Although she was initially normotensive, her blood pressure trended downward to systolic measurements in the 80-mm Hg range several minutes after initial evaluation. The abruptness of her dyspnea, with a clear lung examination and ECG demonstrating tachycardia with RBBB, cued the EP to consider PE as the likely diagnosis within minutes of her arrival. The standard ED work-up for stable patients suspected of having PE includes CT pulmonary angiography in those with normal renal function and without contrast allergy. Alternative testing includes a V/Q scan of the lungs, with or without venous duplex ultrasound of the lower extremities, looking for DVT as a source of PE. Our patient had a contrast dye allergy and was too unstable for transport to the radiology suite or the Nuclear Medicine Department. Focused bedside echocardiography provided a readily available, safe, and non-invasive diagnostic tool that did not require the patient to leave the ED. These features make it an attractive adjunct; however, it is relatively insensitive, as only 30–40% of patients with acute PE will display an echocardiographic abnormality (10). A description of the relevant findings follows (Table 1).

Table 1. Echo/Ultrasound Findings in Acute PE

Direct (high specificity, low sensitivity)
Right heart thrombus (1,7–12)
PA thrombus (6,10–12)
Indirect (low specificity, moderate sensitivity)
RV dilatation (1,6–13)
McConnell sign (better specificity) (7,10,12,13)
RV dysfunction (1,6–8,10,11,13)
IVS flattening (1,6,7,10–12)
IVC dilatation without inspiratory collapse (7,10,12)
DVT seen on lower extremity venous duplex (10,11)

PE = pulmonary embolism; PA = pulmonary artery; RV = right ventricle; IVS = interventricular septum; IVC = inferior vena cava; DVT = deep vein thrombosis.

“McConnell sign,” a specific pattern of RV dysfunction seen in patients with acute PE, is seen as mid-free wall akinesia or hypokinesia with normal apical motion on the apical four-chamber view. It carries a sensitivity of 77%, specificity of 94%, positive predictive value of 71%, and negative predictive value of 96%. Other causes of RV dysfunction (e.g., primary pulmonary hypertension, dilated cardiomyopathy) typically display diffuse, rather than regional, RV dysfunction (13).

The ratio of RV end-diastolic area to LV end-diastolic area should be 0.6 or less. Values in excess of 0.6 indicate RV dilatation. Flattening or bowing of the IVS toward the LV often accompanies this finding and indicates significant RV volume overload. Due to ventricular interdependence, LV diastolic filling is impaired, leading to a reduction in cardiac output (12).

Another indirect finding involves measuring the diameter of the inferior vena cava (IVC) during inspiration and expiration using a subcostal view. Lack of inspiratory collapse of the IVC by 50% suggests a right atrial pressure > 10 mm Hg, whereas the converse indicates a right atrial pressure < 10 mm Hg (10). This non-specific finding may lend further support to a suspicion of elevated right heart pressures.

A comprehensive discussion of thrombolytic therapy in the setting of PE is outside the scope of this article; however, most clinicians agree that in the setting of hemodynamically significant PE (i.e., “massive PE”), thrombolytic administration is supported (14).

CONCLUSION

This case illustrates the utility of ED focused bedside echocardiography in the assessment of patients in

whom acute PE is suspected. Despite its limitations, it provides a safe, non-invasive, rapidly accessible, and inexpensive diagnostic adjunct in the hands of emergency physicians experienced in ultrasonography.

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SUPPLEMENTARY DATA

Supplementary data associated with this article can be found, in the online version, at [doi:10.1016/j.jemermed.2011.05.044](https://doi.org/10.1016/j.jemermed.2011.05.044).

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