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CASE REPORT

Emergency department bedside echocardiography diagnosis of massive pulmonary embolism with direct visualization of thrombus in the pulmonary artery

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Abstract A 62-year-old woman presented to the emergency department (ED) with a chief complaint of 3 weeks of progressively worsening shortness of breath. Her physical examination was normal, except for tachypnea. Her lungs were clear, and no murmurs, gallops or rubs were heard on the cardiac examination. ED bedside echocardiography with color, continuous wave and tissue Doppler ultrasound imaging and lower extremity ultrasonography performed by an ED attending physician revealed a massive pulmonary embolism with right ventricle pressure overload, tricuspid and pulmonic regurgitation, right atrial thrombus and a right popliteal thrombus. The right ventricular outflow tract view allowed for the direct visualization of thrombus in the pulmonary artery. Bedside echocardiography with color, continuous wave and tissue Doppler ultrasound imaging and lower extremity compression ultrasonography can assist the emergency physician and the critical care physician in the diagnosis of massive pulmonary embolism and deep venous thrombosis. Direct visualization of embolic thrombus in the pulmonary artery can help the ED physician accelerate both medical intensive care consultation and therapy and surgical consultation for possible thromboembolectomy and inferior vena cava filter placement.

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Keywords Emergency department · Bedside echocardiography · Massive pulmonary embolism · Direct visualization of thrombus in pulmonary artery · Pulmonary hypertension · Tricuspid regurgitation · Pulmonic regurgitation · Right ventricle tissue Doppler · Right ventricle pressure overload · Hepatic vein pulse Doppler systolic reversal

Case report

A 62-year-old woman with a past medical history of asthma and chronic obstructive pulmonary disease, presented to the emergency department (ED) with a chief complaint of 3 weeks of progressively worsening shortness of breath distinctly different from her usual asthma exacerbation symptoms. She had no history of cancer, pulmonary embolism or deep venous thrombosis, and she denied any recent plane flights or upper or lower extremity trauma. She reported no abdominal, back or chest pain. She had no history of coughing, fever, chills or chemical exposures. She denied smoking. Her ED vital signs were temperature 98.0°F, blood pressure 123/89 mmHg, respiratory rate 18 bpm, room air oxygen saturation 96%, and a heart rate of 109 bpm. Her ECG was normal sinus rhythm with inverted T waves in leads, III, V1–V4, and a right axis deviation. Her chest X-ray showed no consolidations or effusions (Fig. 1). Her physical examination was normal, except for her tachypnea. Her lungs were clear, and no murmurs, gallops or rubs were heard on the cardiac examination. She had no femoral or popliteal tenderness to palpation. Her stool exam was guaiac negative. Laboratory studies were normal, except for a BNP level of 774.7 (normal 0–100 pg/mL).

Emergency department bedside echocardiography and lower extremity ultrasonography were performed by an ED

Fig. 1 Chest X-ray

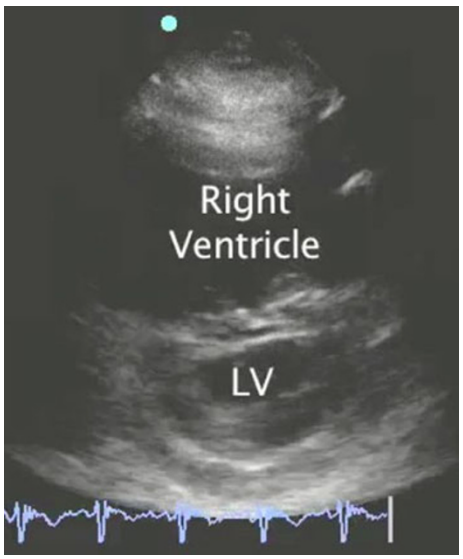

Fig. 2 Parasternal short-axis view

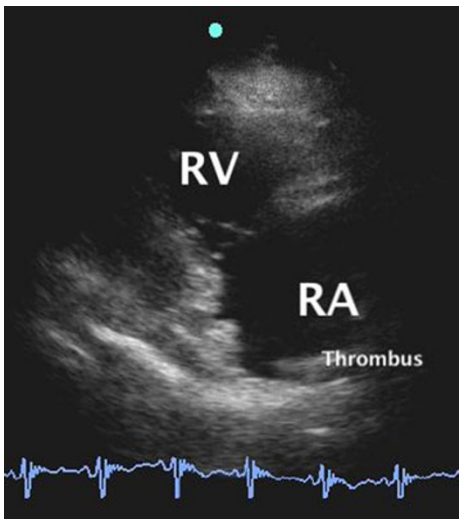

Fig. 3 Right ventricular inflow tract view-R. Atrial thrombus

attending physician (see Video Clips S1–S9 available as supporting information in the online version of this paper). Examination of the heart with a low-frequency array cardiac probe in the parasternal long-axis view revealed no pericardial effusion and an enlarged right ventricular outflow tract (Video Clip S1). Right ventricular pressure overload with a D-shaped left ventricle present during both systole and diastole, and a large dilated right ventricle was present in parasternal short-axis view of the heart (Fig. 2 and Video Clip S2). The right ventricular inflow tract view (RVIT) revealed a right atrial thrombus (Fig. 3 and Video Clip S3). The apical four chamber view revealed a dilated right ventricle with apical and basal segment contractions with bulging out of the mid-right ventricle (McConnell sign) and color Doppler evacuation showed tricuspid regurgitation (Figs. 4 and 5, Video Clips S4 and S5). Continuous wave Doppler evaluation of the tricuspid valve in the apical four chamber view revealed tricuspid regurgitation with a 3.83 m/s regurgitant jet (Fig. 6). Tissue Doppler evaluation of the lateral right ventricular wall in the apical four chamber view revealed an S-wave velocity of 8.93 cm/s (Fig. 7). ED bedside echocardiography evaluation in the right ventricular outflow tract (RVOT) view revealed the direct visualization of an occlusive thrombus in the right main pulmonary artery (Fig. 8 and Video Clip S6). Color Doppler evaluation of the pulmonic valve in the RVOT view showed pulmonic regurgitation (Fig. 9 and Video Clip S7). Right atrial pressure was estimated at 15 mmHg, as our patient had an inferior vena cava (IVC) diameter >2.1 cm and had less that 50% inspiratory collapse of the IVC in the subcostal view of the IVC going into the right atrium (Fig. 10 and Video Clip S8) [1]. Using the modified Bernoulli equation and RAP estimation by IVC inspiratory collapse method recommended by the American Society of Echocardiography: right ventricle systolic pressure (RVSP) = pressure gradient between the RV and RA = 4 × [tricuspid regurgitant Doppler jet velocity (m/s)]^2 + right atrial pressure (RAP) [1]:

In our patient:

$$\begin{aligned} \mathrm{RVSP} &= 4V^2 + \mathrm{RAP} \\ &= 4 \times (3.83\ \mathrm{m/s})^2 + 15\ \mathrm{mmHg} \\ &= 73.7\ \mathrm{mmHg} \end{aligned}$$

Subcostal evaluation of the patient's most vertical hepatic vein with pulse wave Doppler revealed systolic reversal and biphasic hepatic vein blood flow due to the patient's tricuspid regurgitation (Fig. 11). With a high-frequency linear array probe, ED bedside compression

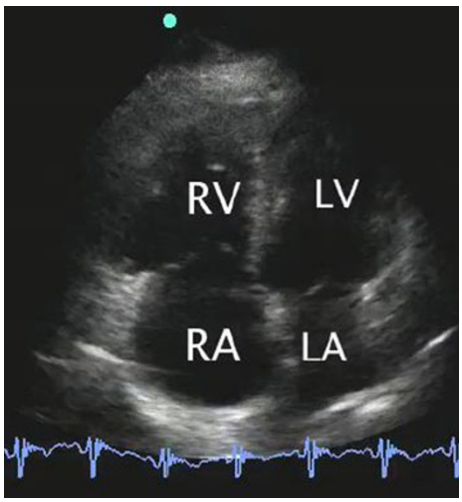

Fig. 4 Apical four chamber view—McConnell sign

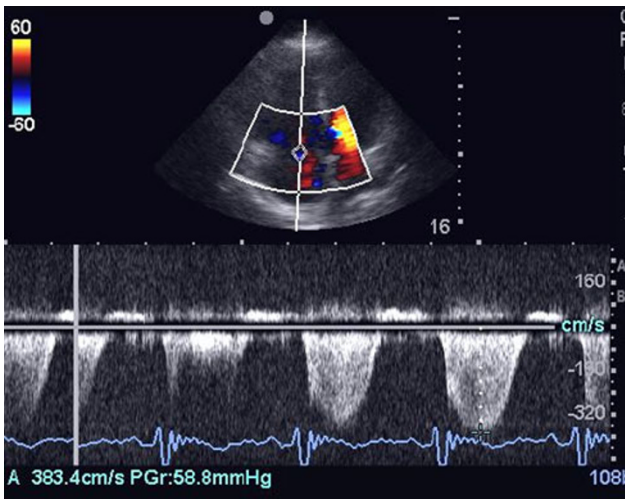

Fig. 6 Tricuspid regurgitation apical 4 view-CW Doppler

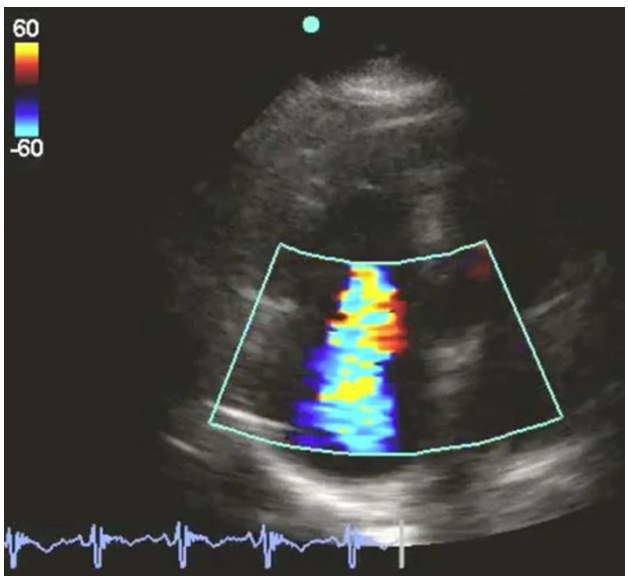

Fig. 5 Tricuspid regurgitation apical 4 view color Doppler

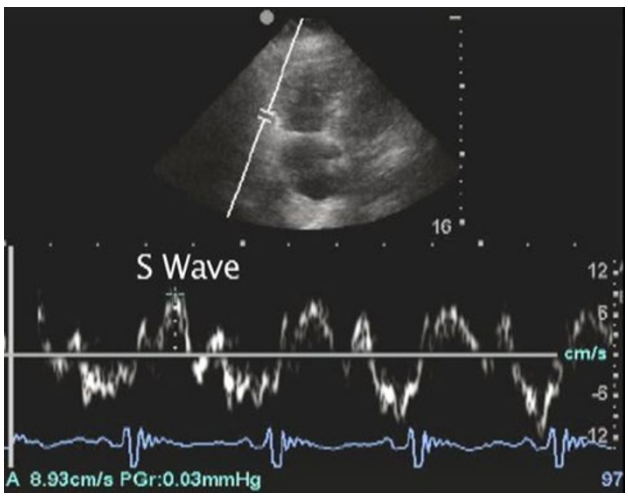

Fig. 7 Apical four-tissue Doppler of right ventricle

ultrasonography evaluation of the lower extremities bilaterally revealed a right popliteal deep venous thrombosis (Fig. 12 and Video Clip S9).

The patient subsequently had a formal computed tomography angiogram of the chest evaluation performed by the Radiology department that revealed massive pulmonary emboli in the left and right main pulmonary arteries extending into the segmental arteries bilaterally (Fig. 13). In addition, a Radiology department performed ultrasound examination of the lower extremities showed a near complete occlusive thrombosis of the right popliteal vein. Consultative cardiology echocardiography examination revealed left ventricular septal flattening consistent with right ventricular pressure overload, a right atrial mobile thrombus, pulmonary valve regurgitation and tricuspid regurgitation and an estimated right ventricular systolic pressure, RVSP, of 75 mmHg. The formal Cardiology echocardiography evaluation also revealed normal apical right ventricular contractility with hypokinetic other right ventricle walls (McConnell sign). Emergency department intravenous heparin therapy was initiated. The patient was admitted to the Medical Intensive Care Unit for further treatment with intravenous tissue plasminogen activator (tPA). Emergency department Cardiothoracic and Vascular Surgery consultation was obtained for the evaluation for possible thromboembolectomy and inferior vena cava filter placement. Our patient was initially hemodynamically stable during most of her ED course; however, after transfer to the MICU, she became hemodynamically unstable during MICU tPA administration, and the Cardiothoracic surgery service took her urgently to the operating room for surgical thromboembolectomy. Early ED

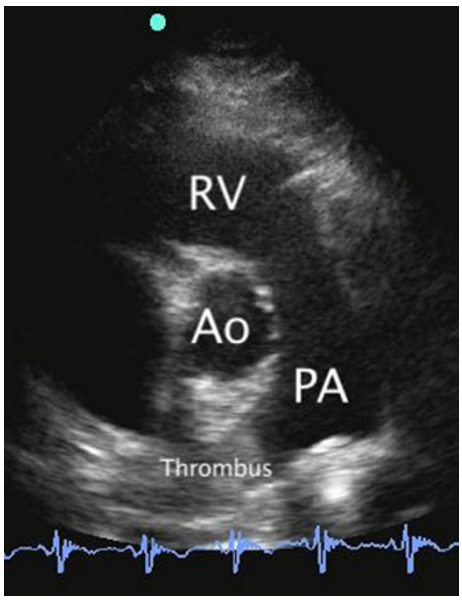

Fig. 8 RVOT view-direct visualization of thrombus in PA

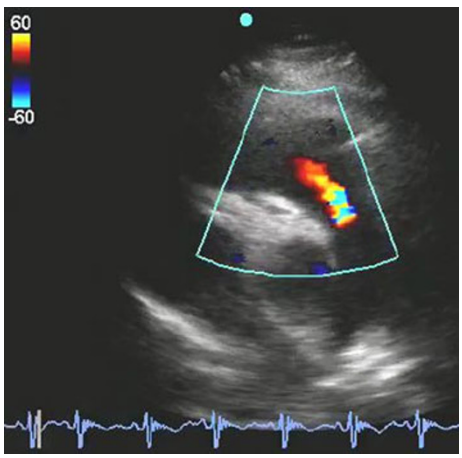

Fig. 9 RVOT view pulmonic regurgitation

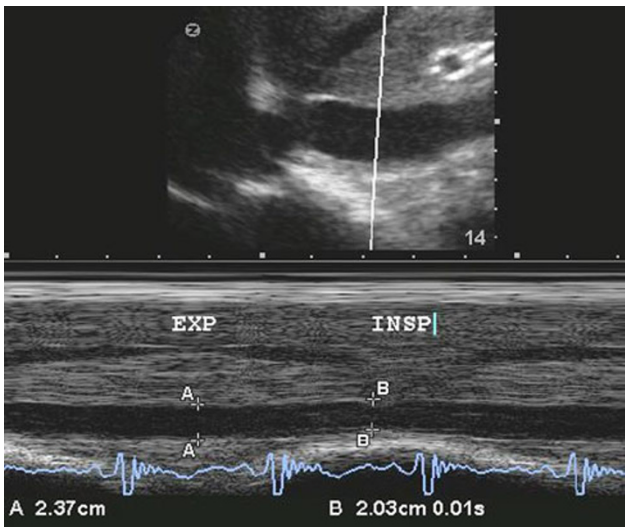

Fig. 10 Inferior vena cava into right atrium

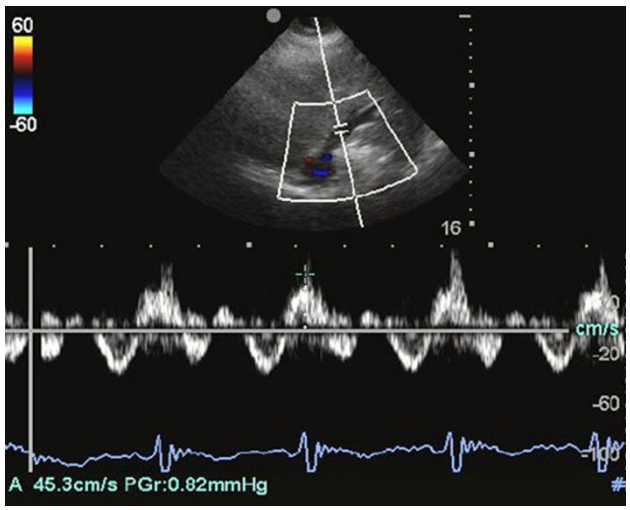

Fig. 11 Hepatic vein systolic reversal due to TR

Cardiothoracic surgery consultation on our patient was critical to help expedite the surgical team to the operating room. The patient recovered from her surgical thrombo-embolectomy and she was discharged from the hospital neurologically intact.

Discussion

Patients with massive pulmonary embolism usually have both right ventricular and right atrial pressure overload with pulmonic and tricuspid regurgitation with most patients being tachycardic and hypotensive [1–15]. At the patient's bedside in the ED, we were able to directly visualize an occlusive thrombus in the patient's right pulmonary artery using the parasternal short-axis RVOT view. ED physician performed bedside echocardiography which was a very useful tool to allow us to expedite medical therapy and obtain early ED surgical consultation. Although our patient was tachycardic and very dyspneic in the ED, she remained alert and normotensive during most of her ED course. In the ED, however, she decompensated and became hypotensive requiring an intravenous fluid bolus with her blood pressure normalizing prior to medical intensive care unit transfer.

ED cardiology physicians have reported direct visualization of thrombus in the pulmonary artery using bedside transthoracic echocardiography specifically using the parasternal short-axis RVOT view [5–7]. Madan et al. [6] demonstrated a directly visualized decrease in the amount of pulmonary artery thrombus after intravenous tissue

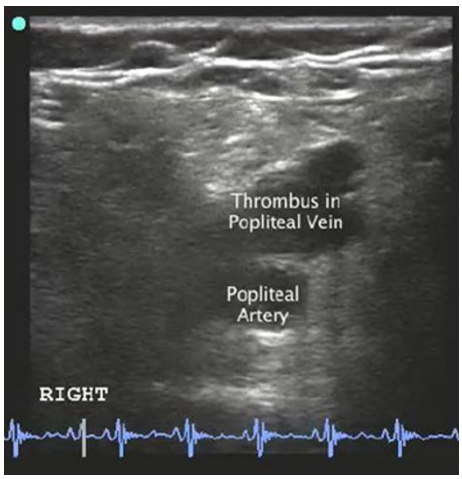

Fig. 12 Popliteal deep venous thrombosis

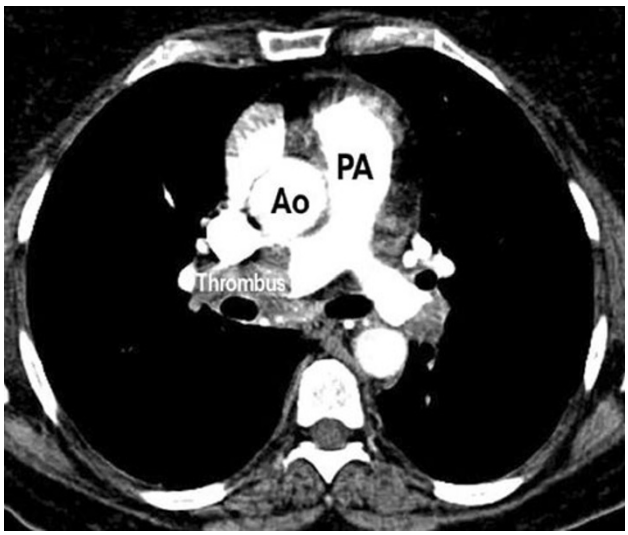

Fig. 13 CT scan of massive pulmonary embolism

plasminogen activator administration. Emergency physicians have reported indirect bedside echocardiography signs of submassive or massive pulmonary embolism such as right ventricle enlargement, a flattened intraventricular septum in both systole and diastole, McConnell sign and tricuspid regurgitation, with no direct visualization of a pulmonary artery thrombus [4, 8, 9]. In our patient, we were able to directly visualize an occlusive thrombus in the right main pulmonary artery in the RVOT view using ED bedside echocardiography (Fig. 8 and Video Clip S6).

Indirect bedside echocardiography signs of massive or submassive acute pulmonary embolism include: the McConnell sign of right ventricular mid-segment dilation with normal apical contractility, tricuspid and pulmonic regurgitation with elevated RVSP and RV pressure overload, pulmonary hypertension, D-shaped left ventricle with a flattened intraventricular septum during the entire cardiac cycle, right atrial thrombus, decreased tissue Doppler S-wave in the apical four chamber RV lateral basal wall view, and hepatic vein pulse wave Doppler systolic reversal and biphasic hepatic vein blood flow due to tricuspid regurgitation [1–13]. Our patient demonstrated all of these indirect signs of massive pulmonary embolism yet the direct visualization of the thrombus in the pulmonary artery with ED bedside echocardiography was the most clinically diagnostic and useful as chronic conditions such as chronic pulmonary hypertension with resulting RV hypertrophy may also result in RV strain.

Patients with RV infarction can have a positive McConnell sign and Casazza et al. [11] have reported that the McConnell sign is only 33% specific for pulmonary embolism. They noted, however, that continuous wave Doppler helps differentiate RV infarction from submassive pulmonary embolism by demonstrating an increased tricuspid regurgitation right atrial to right ventricle pressure gradient in submassive pulmonary embolism yet a normal gradient in RV infarction [11]. Both RV infarction and massive pulmonary embolism will have a decreased lateral RV apical four chamber tissue Doppler S-wave velocity and less than 10 cm/s is considered abnormal [12]. Our patient had a right ventricular tissue Doppler S-wave velocity of 8.93 cm/s confirming RV pressure overload with decreased RV systolic function. Patients with acute massive pulmonary embolism and right atrial pressure overload can have a dilated right ventricle with tricuspid regurgitation, elevated right ventricular systolic pressure which is an estimate of pulmonary artery systolic pressure. We estimated our patients RVSP to be 73.7 mmHg and the formal Cardiology RVSP estimation was 75 mmHg, both measurements indicating RV pressure overload. When estimating right atrial pressure with IVC collapse, Randazzo et al. [16] has reported fair-to-moderate inter-rater reliability (kappa of 0.41) of cardiologist performed versus emergency physician performed echocardiography evaluation of IVC inspiratory collapse in non-intubated patients to estimate central venous pressure. We also measured our patient's most vertical hepatic vein with pulse Doppler revealing systolic reversal and biphasic hepatic vein Doppler waves (Fig. 11). This hepatic vein Doppler signal is usually the result of a combination of tricuspid regurgitation and decreased right ventricular systolic function, as our patient had a decreased lateral wall RV tissue Doppler velocity, less than 10 cm/s [13].

Burnside et al. [17] in their systematic review of pooled data from six articles and 936 patients suggest that emergency physician-performed ultrasonography for the diagnosis and exclusion of deep venous thrombosis compared to Radiology-performed ultrasonography imaging may be accurate with overall sensitivity of 95% (95% CI = 87–99%) and overall specificity of 96% (95%

CI = 87–99%). Our ED bedside diagnosis of a right popliteal deep venous thrombosis was confirmed by a formal Radiology study and Vascular surgery consultation was initiated to evaluate for possible inferior vena cava filter placement.

The prognosis of a patient with a submassive or massive pulmonary embolism and a right atrial or right ventricle thrombus is markedly decreased [14, 15]. Patients who have a large thrombus in their pulmonary artery and thrombus in either the right atrium or right ventricle are at risk for rapid hemodynamic decline and they may need medical thrombolytic therapy or surgical thrombus removal [14, 15]. We were able to visualize thrombus in our patient's pulmonary artery and right atrium. Cardiothoracic surgery was consulted early, while the patient was still in the ED.

The supplemental video clips illustrate how ED bedside echocardiography can assist the emergency physician in the diagnosis of massive pulmonary embolism with both direct visualization of thrombus in the pulmonary artery and with examining indirect signs of RV pressure overload.

Advanced bedside echocardiography evaluation of patients for massive pulmonary embolism with direct thrombus visualization in the pulmonary artery and right ventricular pressure overload is an operator-dependant modality, and emergency and critical care physician's screening-advanced echocardiography should always be followed by formal cardiology echocardiography examination.

Conclusion

Bedside echocardiography with color Doppler imaging, continuous wave Doppler imaging and tissue Doppler imaging and lower extremity compression ultrasonography can assist the emergency physician and the critical care physician in the diagnosis of massive pulmonary embolism and deep venous thrombosis. Direct visualization of embolic thrombus in the pulmonary artery can help the ED physician accelerate both medical intensive care consultation and therapy and surgical consultation for possible thromboembolectomy and inferior vena cava filter placement.

Conflict of interest None.

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