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Case Report

Utilization of bedside ultrasound in the diagnosis and management of massive pulmonary embolism: a case report

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ABSTRACT

The prompt diagnosis and treatment of massive pulmonary embolism is a well-known challenge for physicians. We report a case of a 61-year-old hemodynamically unstable man who presented to the emergency department with complaints of acute dyspnea. After performing a focused history and physical, we used bedside ultrasound to diagnose significant right heart strain, which suggested massive bilateral pulmonary embolisms. This diagnosis was further supported by the visualization of deep venous thrombosis in the left lower extremity. The patient was treated with IV tissue plasminogen activator in the emergency department and survived to discharge in his usual state of health.

Case report

Accurate diagnosis of pulmonary embolism (PE) can be difficult due to the nonspecific signs and symptoms that can be seen with similar cardiopulmonary disease presentations [1–3]. In the emergency room setting, alternative diagnostic strategies should be implemented when obtaining a computed tomography pulmonary angiogram (CTPA) results in a delay of care. A massive PE is defined as an acute PE with obstructive shock or systolic blood pressure less than 90 mm Hg. We present a case in which unprovoked deep venous thrombosis caused massive PEs in an unstable patient.

A 61-year-old ill-appearing man was evaluated in our emergency department complaining of dyspnea for one week, along with generalized weakness and near syncope before calling 911. Emergency Medical Services found the patient with unstable vital signs and a prehospital electrocardiogram with indeterminate supraventricular tachycardia. Emergency Medical Services-administered 2 rounds of adenosine per advanced cardiac life support protocol without improvement. The patient arrived to the emergency department with a systolic blood pressure of 70 mm Hg, heart rate of 140-160 bpm, respiratory rate of 22 bpm, and a SpO_2 of 88%-90% on 10L non-rebreather mask. On arrival, the patient was immediately

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placed on a monitor with a code cart, airway cart, and ultrasound machine present for emergent intervention.

While unstable supraventricular tachycardia was a possible diagnosis, a focused history suggested otherwise. The patient complained of a "charley horse" pain in his left calf for a few days before presentation. The patient, however, denied a history of venous thromboembolic disease, recent surgery or trauma, past or current malignancy, or family history of genetic thrombophilia disease. Using the Wells score for PE, the patient's presenting symptoms of tachycardia and calf pain supported a high risk of PE [4].

Current guidelines suggest that the best diagnostic strategy to confirm or exclude acute PE requires appropriate clinical assessment and risk factor stratification using either plasma D-dimer measurement and/or the gold standard CTPA [5]. However, CTPA is not always practical due to the lack of immediate availability of equipment or staff, or its limited utility in patients with severe renal insufficiency or unstable patients [2]. In this case, the patient's unstable vital signs necessitated an immediate diagnostic tool. Bedside ultrasound of the lower extremities revealed extensive thromboses in the left femoral vein and left popliteal vein with no compressibility (Figs. 1, 2). These sonographic findings were suggestive of thromboembolic lesions according to the criteria reported in the literature supporting the diagnosis of a massive PE [2,6,7]. Although silent PE can develop in up to 50% of patients, more often than not, PE is diagnosed in patients with lower limb deep vein thromboses [8].

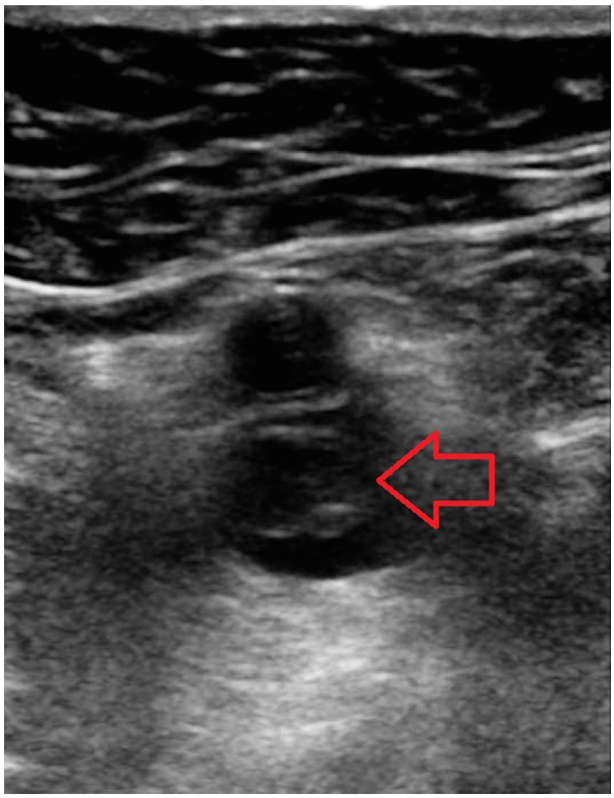

Fig. 1 – Sonogram of the left femoral vein in a 61-year-old man shows a large occlusive thrombus observed in the noncompressible left femoral vein (arrow).

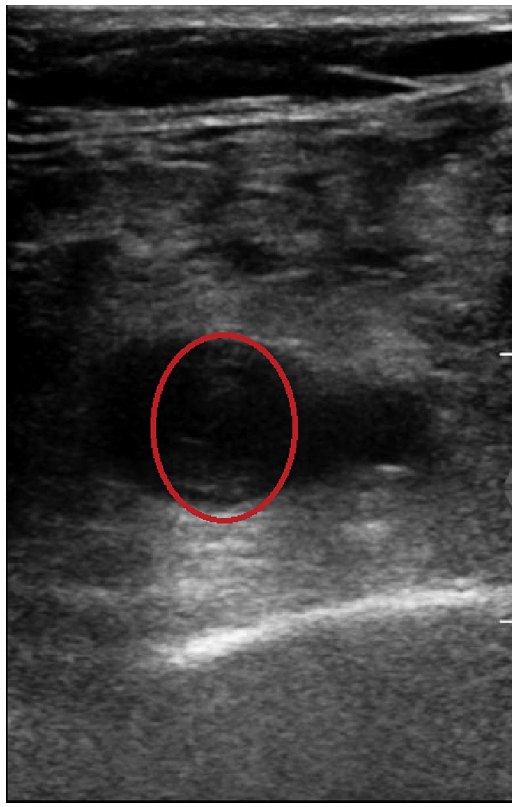

Fig. 2 – Sonogram of the popliteal vein shows a large occlusive thrombus (circled) observed in a noncompressible left popliteal vein.

Studies by Squizzato et al. [2] concluded no single laboratory or imaging test was sufficient to diagnosed PE alone. Similar literature suggests applying a combination of lower-extremity compression venous ultrasonography with echocardiography to diagnose massive PEs [9]. Using bedside

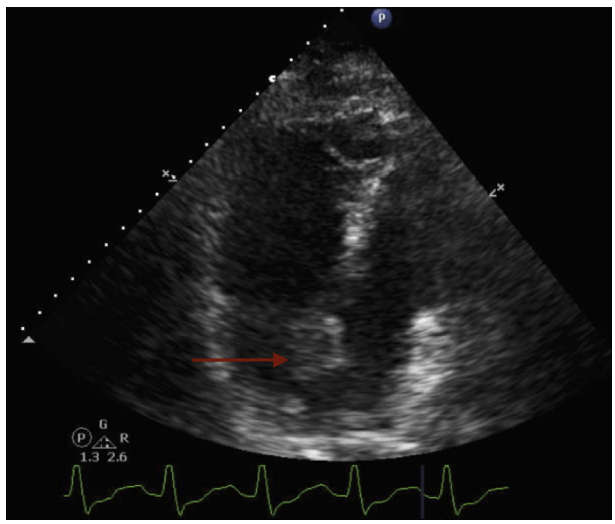

Fig. 3 – Sonogram of the heart (apical view) outlining significant right ventricular strain and thrombus in right atrium (arrow).

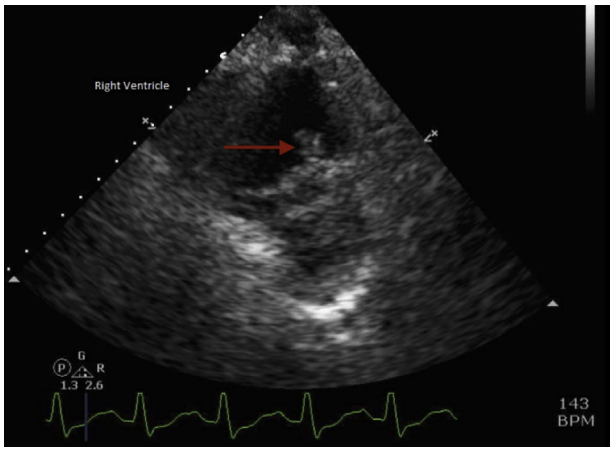

Fig. 4 – Sonogram of the heart (short axis) showing thrombus in right ventricle along with right heart strain (arrow).

ultrasound, we were able to visualize right heart strain. A limited stat transthoracic echocardiogram was used to evaluate right ventricular (RV) function, where we visualized a mobile echogenic thrombus that measured 2.84 cm in length in the right atrium (Figs. 3, 4). Enlarged RV size and hyperdynamic left ventricular systolic function was similarly noted (Figs. 3, 4). There was a distinct regional pattern of RV dysfunction with akinesia of the midfree wall and normal motion at the apex consistent with the McConnell's sign, which increased our certainty of massive PE as the main cause of our patient's clinical symptoms (apical echocardiogram https://www.dropbox.com/s/yajy9id4bbqb6eu/Fig%205%20ApicalEcho2.mpg?dl=0 and parasternal long echocardiogram access at https://www.dropbox.com/s/0t80c7hdt6ukr9s/Fig%206%20ParasternalLongEcho1.mpg?dl=0).

The McConnell's sign observed carries a sensitivity of 77% and specificity of 94% for diagnosis of acute PE, which therefore increased our certainty of massive PE as the main cause of our patient's clinical symptoms [10]. Similar studies have supported the use of echocardiography in cases of high-risk PE in which bedside diagnostic tests were life saving [5,11].

After a brief discussion with our interventional radiology and cardiothoracic surgery teams, we decided to proceed with IV tissue plasminogen activator instead of interventional thrombectomy. This decision was based on the fact that the patient was at risk for rapid decompensation. Literature demonstrates that most clinicians would have given thrombolytic therapy in a hemodynamically unstable patient with PE [12].

Over the course of a 10-day hospital stay, the patient was started on coumadin and received an inferior vena cava filter. He was discharged in his usual state of health. This optimal outcome would not have been as likely if the care team had not moved quickly to the resources available to them in the emergency department. In summary, this case illustrates the teaching point of the advantages clinicians can offer patients if they are skilled in bedside ultrasound utilization. While caution must be exercised in relying only on ultrasound if the clinicians are not skilled in its use, our case underscores the utility of bedside ultrasound in making time-sensitive decisions when precluded from using gold standard diagnostic strategies such as CTPA.

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