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Multiorgan Ultrasonography for the Diagnosis of Pulmonary Embolism

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ABSTRACT

The main ultrasound criterion for diagnosing pulmonary infarction is the presence of triangular/wedge-shaped or rounded, hypoechogenic, homogeneous, pleura-based lesions. When used in point-of-care, ultrasonography of several organs can facilitate the diagnosis of pulmonary embolism in a patient presenting with chest pain. We describe a case of chest pain which we thought was due to angina, but point-of-care ultrasonography directed us to a diagnosis of pulmonary embolism.

LEARNING POINTS

- Point-of-care ultrasonography can help the clinician make a rapid diagnosis in patients with acute respiratory failure.
- The main ultrasound criterion for diagnosing pulmonary infarction is the presence of triangular/wedge-shaped or rounded, hypoechogenic, pleura-based lesions.

KEYWORDS

Ultrasonography, pulmonary embolism, POCUS

INTRODUCTION

Venous thromboembolism (VTE), including pulmonary embolism (PE) and deep vein thrombosis (DVT), is the third leading cause of cardiovascular death worldwide (ranked after stroke and heart attack) [1]. PE is heterogeneous with multiple vague presentations and non-specific signs and symptoms [1]. It should be suspected in patients presenting with dyspnoea, chest pain, syncope, shock/hypotension, or cardiac arrest without an obvious alternative cause of an ischaemic cardiac event [2]. As a bedside tool, and in conjunction with clinical assessment, point-of-care ultrasonography (POCUS) can provide faster anatomic, functional and physiological information in patients suspected of having PE in acute presentations [3]. Here we present the case of a patient with exertional dyspnoea and chest pain with an early diagnosis of angina who actually had PE as indicated by POCUS.

CASE DESCRIPTION

A 59-year-old man with hypertension was on a diet and had smoked 20 cigarettes/day for the past 20 years. He had undergone sleeve surgery in 2013 and lost 50 kg. He admitted a history of admission to another facility in 2009, with an issue with his left scapula keeping him in the CCU for 2 days, following which he was discharged after 5 days. A stress ECG at the time was normal, and he was told that there was no need for coronary angiography.

The patient presented with a history of left-sided compressing chest pain and shortness of breath 4 days previously, after which his symptoms had improved. However, the next day, he had a similar attack of central chest pain with shortness of breath. There was no associated nausea or vomiting. Moreover, there was no cough or fever and no contact with sick people. His blood pressure was 140/80 mmHg, he was afebrile,

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his pulse was 84 beats/minute and regular, and his respiratory rate was 24 breaths/minute with saturation O2 at 92% on room air, which was increased to 98% on nasal prongs at 2 l/minute. Physical examination was normal, and there was no lower limb swelling or calf muscle tenderness. A serial electrocardiogram showed sinus rhythm and persistent minimal ST elevation in leads V2–4 with an early repolarization pattern. Serial high-sensitivity troponin levels were 80, 70 and 60 ng/l at 0, 4 and 8 hours, respectively (normal value: <20 ng/l). D-dimer was 13 µg/ml (normal: <0.5). Moreover, NT-proBNP was elevated at 1111 pg/ml. The CBC and renal panels were normal. In addition, the RT-PCR was negative for COVID-19 pneumonia. A focused echocardiogram showed normal left ventricle internal dimensions and normal systolic function. The right ventricle was borderline in size, with mild tricuspid regurgitation, and the systolic peak pulmonary pressure was 45 mmHg. A lung ultrasound performed as part of the assessment for pulmonary oedema showed two subpleural hypoechoic areas in the shape of triangles, with the base facing the pleura, which was suggestive of pulmonary infarction (*Fig. 1*, *Video 1*).

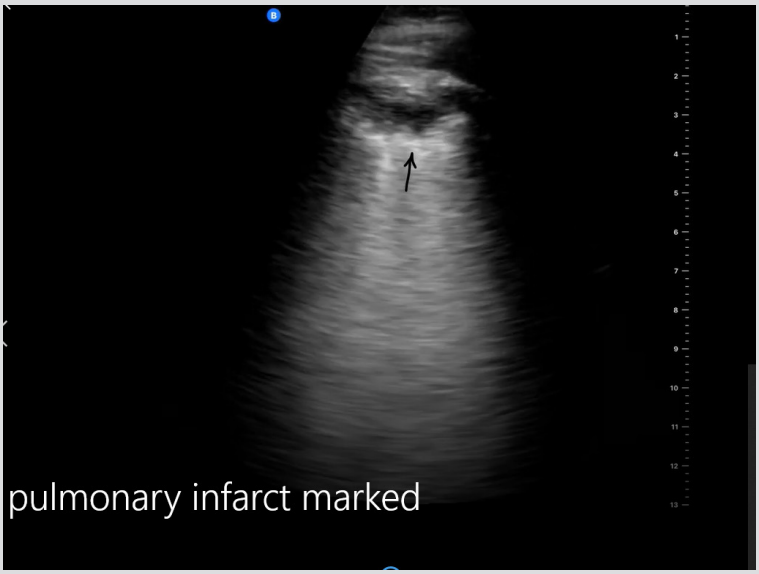
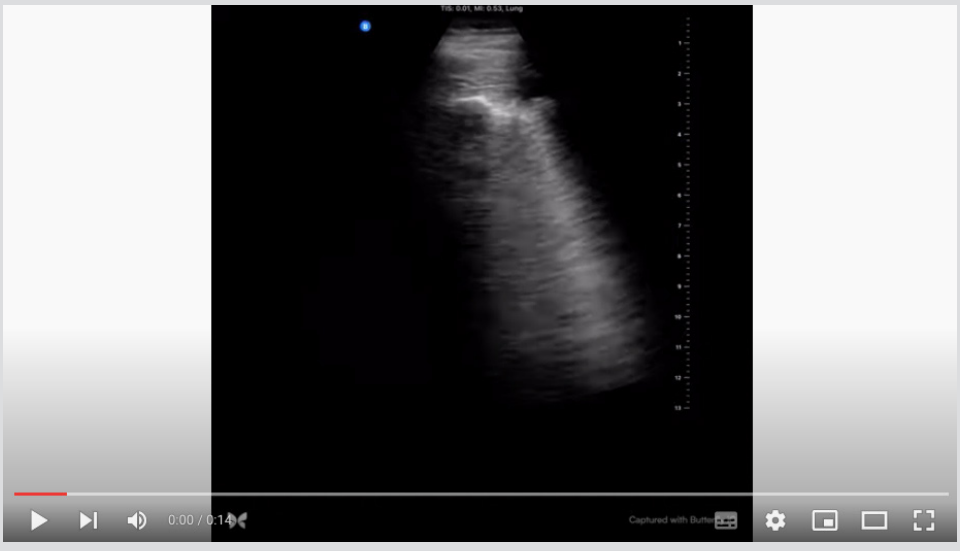


Figure 1. Lung ultrasound in the region of the left anterior upper zone using a curvilinear probe (5 MHz) and showing triangular/wedge-shaped or rounded, hypoechogenic, homogeneous, pleura-based lesions (arrow)



Video 1. Subpleural hypoechoic areas in the shape of a triangle with the base facing the pleura.
View the video: https://youtu.be/80U8TjYXxhI

This finding directed us to Doppler ultrasound of the veins of the left leg which showed a thrombus in the left popliteal vein with limitation of flow and a non-compressible vein (*Fig. 2*). CT pulmonary angiography (CTPA) showed an acute PE in the right and left main pulmonary arteries, which was more evident on the right side (*Fig. 3*). The thrombus was observed in the segmental arteries (*Fig. 3*). The patient was started on enoxaparin 1 mg/kg twice daily, followed by warfarin, and maintained an INR between 2 and 3.

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After 1 month of follow-up, the patient was asymptomatic. The diagnosis was considered to be a provoked PE, as the patient later mentioned a long drive that had occurred 4 weeks before the incident.

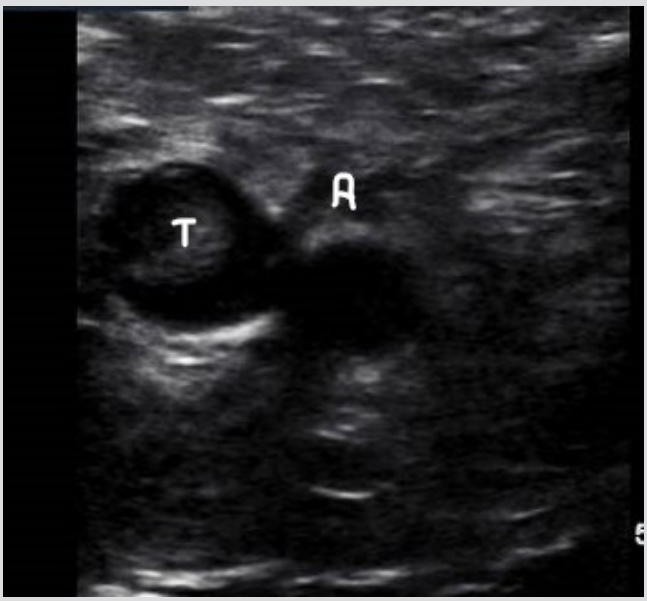

Figure 2. Doppler venous left leg examination showed a thrombus. (T) Left popliteal vein (A) Left popliteal artery

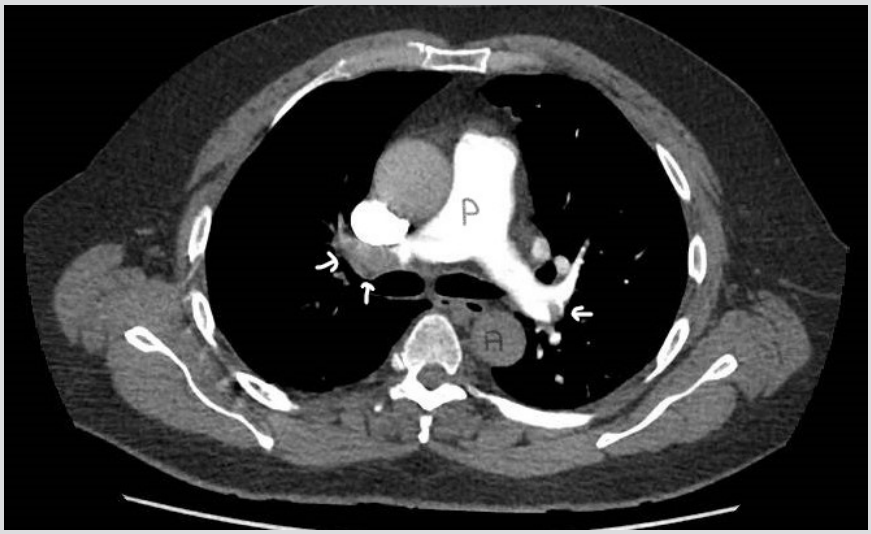

Figure 3. Computerized tomography pulmonary angiography showed acute pulmonary embolisms in the right and left main pulmonary arteries, larger on the right side.
(A) Descending aorta, (P) main pulmonary trunk

DISCUSSION

The main ultrasound criterion for diagnosing PE is the presence of triangular/wedge-shaped or rounded, hypoechogenic, homogeneous, pleura-based lesions [4–6]. A comparison of the diagnostic performance of chest sonography, MRI angiography and ventilation/perfusion scintigraphy in PE demonstrated that chest sonography revealed rounded or wedge-shaped hypoechoic lesions with a sensitivity and specificity of 81% and 84%, respectively, in a prospective clinical study in 55 patients [7]. Another study observed that a diagnosis of PE was suggested if at least one typical pleural-based/subpleural wedge-shaped or round hypoechoic lesion (with or without pleural effusion) was reported using thoracic ultrasound [8]. Mathis et al. diagnosed PE via thoracic ultrasound according to the following criteria: (1) confirmed PE, represented by two or more typical triangular or rounded pleural-based lesions; (2) probable PE, represented by one typical lesion with pleural effusion; (3) possible PE, represented by small (<5 mm) subpleural lesions or a single pleural effusion alone; or (4) normal thoracic ultrasound findings. CTPA was used as the reference method [9]. It was found that thoracic ultrasound had a sensitivity of 74%, a specificity of 95%, a positive predictive value of 95%, a negative predictive value of 75%, and an accuracy of 84%, at a prevalence of 55%. The sensitivity in patients with criterion 1 was 43%, and the specificity was 99%.

Two studies with similar results in inpatients with confirmed PE investigated with lung ultrasound and CTPA, found that peripheral parenchymal lesions (mean: 2.6 lesions per patient) associated with PE were detected using lung ultrasound in 35 patients (80%) with a sensitivity of 80% (CT scanning sensitivity: 82%). The specificity of lung ultrasound for identifying pulmonary infarction was 92% [10, 11]. It is important to detect sonographic evidence of pulmonary infarctions, but its absence does not rule out PE.

Different protocols for ultrasound examination have been proposed but do not include criteria for pulmonary infarction in protocols with multisystem ultrasound. Because of the value of lung ultrasound in diagnosing acute respiratory failure (the BLUE protocol), lung ultrasound pattern A (a normal lung ultrasound) and a positive venous Doppler were used to diagnose PE in patients with respiratory failure [12]. *Bataille* et al. integrated echocardiography in using the same principles of lung ultrasonography without including this sign in the protocol [13]. The rapid assessment of dyspnoea via ultrasound (the RADiUS protocol) did not use thoracic ultrasonography to search for triangular or rounded pleural-based lesions and unilateral pleural effusion, which may indicate pulmonary emboli, because of poor sensitivity [14]. Moreover, chest ultrasound is not sufficiently sensitive to rule out PE [14].

As noted earlier, cardiac ultrasound may help to identify haemodynamically significant PE. Some dyspnoea protocols include lower extremity venous ultrasonography to rule out DVT [14, 15]. Several studies have reported that emergency and other clinicians can quickly and accurately diagnose DVT [15–17]. As the inclusion of DVT evaluation significantly increases the time spent assessing dyspnoea using ultrasound, this examination can be selectively included in suspected PE/DVT cases.

In an assessment of point-of-care, multiorgan ultrasonography findings were considered diagnostic for PE when lung ultrasonography visualized at least one pulmonary subpleural infarct, heart ultrasonography detected right ventricular dilatation or thrombi in the right cavities, or leg vein ultrasonography detected DVT [14]. Multiorgan ultrasonography was performed on people with a Well's score >4 or on patients with a Well's score <4 and a positive D-dimer test [18]. Multiorgan ultrasonography had a sensitivity of 90% and a specificity of 86.2%, lung ultrasonography a sensitivity of 60.9% and a specificity of 95.9%, heart ultrasonography a sensitivity of 32.7% and a specificity of 90.9%, and vein ultrasonography a sensitivity of 52.7% and a specificity of 97.6% [18].

In our case, the presence of a rounded pleural-based lesion on ultrasound suggested the use of lower limb venous Doppler examination, resulting in a confident diagnosis before the use of CTPA. Although the absence of pulmonary infarction on ultrasound does not rule out PE, its presence suggests physicians should examine other organs using ultrasound (echocardiography and lower limb venous Doppler) and diagnose PE at the bedside.

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