

Notching into a Diagnosis—Incorporating Doppler Interrogation into Point-of-Care Ultrasonography to Diagnose a Submassive Pulmonary Embolism



Raja Ezman Raja Shariff, MBChB, Koh Hui Beng, BM, Beh Ting Yuen, MBBS, Tey Yee Sin, MBBS, and Azmee Mohd Ghazi, MBChB, *Selangor, Malaysia*

INTRODUCTION

Patients with pulmonary embolism (PE) can often present with nonspecific symptoms clinically, posing a diagnostic dilemma. We present a case of submassive PE diagnosed in a patient following utilization of point-of-care ultrasonography (POCUS) in the emergency department (ED), allowing for immediate initiation of appropriate treatment. We highlight important imaging findings that are demonstrated to support the clinical presentation of our patient and the diagnosis of PE.

CASE PRESENTATION

A 69-year-old woman, with no known medical illnesses, presented to the ED with a history of lethargy and reduced effort tolerance over the past 3 months. She denied chest pain, dyspnea, or palpitations during that period and had delayed seeking medical attention in view of the raging COVID-19 pandemic and fear of being hospitalized. Her vital signs on arrival included a blood pressure of 140/85 mm Hg, heart rate of 72 beats per minute, and temperature of 36.9 °C. Although the patient denied being short of breath, her oxygen saturation was 94% on room air and respiratory rate was 18 breaths per minute. Examination of the precordium and chest were, however, both unremarkable. Her electrocardiogram revealed sinus rhythm with no evidence of tachycardia, bundle branch blocks, or right ventricular (RV) strain. Her chest radiograph demonstrated hilar shadowing suggestive of engorged pulmonary arteries (Figure 1). Her initial blood investigations revealed raised high-sensitivity troponin I (35 ng/L), NT-pro brain natriuretic peptide (3,500 pg/mL; normal values < 125 pg/mL), and D-dimer (2.5 µg/mL; normal values < 0.5 µg/mL) levels. Thus, POCUS was performed in the ED to review the patient's cardiac function in view of her abnormal biomarkers.

Cardiac POCUS revealed a normal left ventricular systolic function, with estimated left ventricular ejection fraction ranging between 55% and 60%. There were no obvious regional wall motion abnormalities seen in the left ventricle. There was dilatation of both the right atrium (measuring 65.5 mL and 43.7 mL/m² in volume and indexed volume accordingly) and right ventricle (basal and mid diameter of 4.5 cm and 3.7 cm, respectively, on RV focused apical 4-chamber view; proximal RV outflow tract diameter of 3.7 cm and 4.1 cm on parasternal long-axis and short-axis views, respectively; and distal RV outflow tract diameter of 3.5 cm on parasternal short-axis view) (Video 1). There was evidence of intraventricular septal flattening throughout diastole (Figure 2A–C, Video 2). The RV fractional area change was 30%, and tricuspid annular plane systolic excursion was 1.55 cm, both suggestive of a depressed RV systolic function. There was also the presence of akinesia of the mid-RV lateral wall and hyperkinetic RV apical segment, suggestive of McConnell's sign.

Further Doppler interrogation was performed during the POCUS assessment. The peak velocity across the tricuspid valve measured approximately 3.7 m/sec, with a pulmonary regurgitation end-diastolic peak velocity of 2.0 m/sec (Figure 1D–F). As the right atrial pressure was assumed to be 3 mm Hg (the inferior vena cava measured 1.3 cm and was >50% collapsible upon respiration), estimated RV

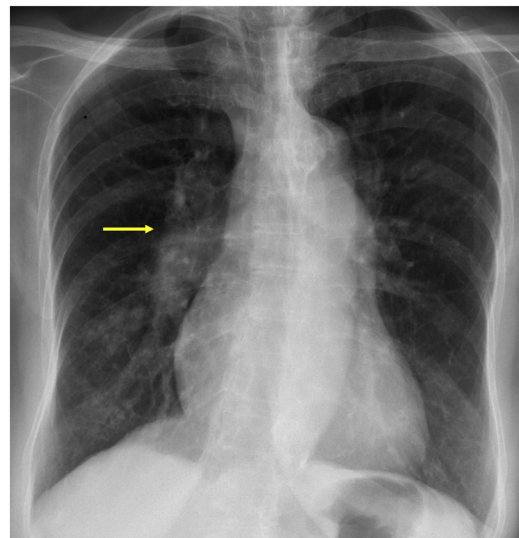


Figure 1 Chest radiograph. There was evidence of right hilar shadowing suggestive of right pulmonary artery engorgement (yellow arrow).

From the Universiti Teknologi Mara Sungai Buloh, Selangor (R.E.R.S.) and Institut Jantung Negara (R.E.R.S., K.H.B., B.T.Y., T.Y.S., A.M.G.), Kuala Lumpur, Malaysia.

Keywords: Pulmonary embolism, Point-of-care ultrasound, POCUS, Case report
Conflicts of Interest: None.

Correspondence: Raja Ezman Raja Shariff, Universiti Teknologi Mara Sungai Buloh, Jalan Hospital, 47000, Sungai Buloh, Selangor, Malaysia (E-mail: rajazeman@gmail.com).

Copyright 2022 by the American Society of Echocardiography. Published by Elsevier Inc. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

2468-6441

<https://doi.org/10.1016/j.case.2022.04.009>

VIDEO HIGHLIGHTS

Video 1: Transthoracic echocardiography in the apical 4-chamber view, demonstrating a dilated right atrium and ventricle, as well as severe tricuspid regurgitation using color-flow imaging. Note the presence of akinesia of the mid-RV free wall and hyperkinetic RV apical segment, consistent with McConnell's sign.

Video 2: Transthoracic echocardiography in the parasternal short-axis view, demonstrating flattening of the interventricular septum throughout diastole and involving systole, to a lesser degree.

Video 3: Computed tomography pulmonary angiography demonstrating a submassive main pulmonary artery embolus, extending into the right pulmonary artery, upper lobar artery, and descending interlobar arteries.

[View the video content online at www.cvcasejournal.com.](http://www.cvcasejournal.com)

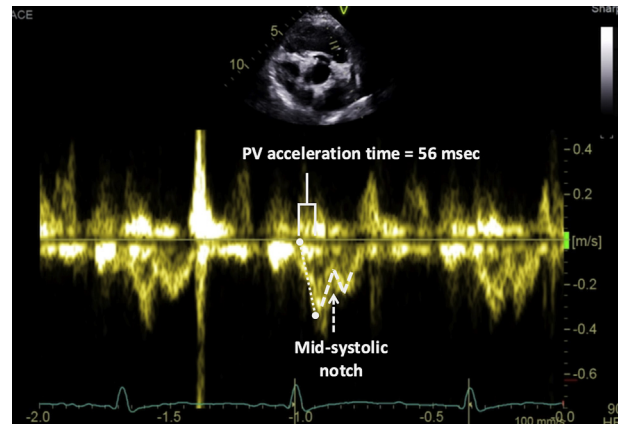


Figure 3 Pulsed-wave Doppler tracing at the pulmonary valve (PV) level, demonstrating an ejection acceleration time of 56 msec and an MSN.

systolic pressure (RVSP) was 58 mm Hg and pulmonary artery diastolic pressure was 19 mm Hg. The vena contracta of the tricuspid regurgitation was 7 mm.

Pulsed-wave Doppler interrogation across the pulmonary valve revealed midsystolic notch (MSN), suggestive of raised pulmonary pressures, with a pulmonary ejection acceleration time of 56 msec (Figure 3). All these findings were suggestive of a possible acute PE. Following this, computed tomography pulmonary angiography was performed revealing a submassive main pulmonary artery embolus, extending down into the right pulmonary artery, upper lobar artery, and descending interlobar arteries (Figure 4, Video 3). As the patient

was hemodynamically stable, but at risk of deterioration, she was started on immediate anticoagulation and was monitored as an inpatient for several days. Lower-limb Doppler and computed tomography imaging of her abdomen showed no evidence of deep vein thrombosis or abnormalities to suggest malignancies, respectively. She was discharged well and continues to be on regular outpatient monitoring on a trimonthly basis and awaiting further investigation into predisposing factors that may have led her to develop PE.

DISCUSSION

The case, although not at all uncommon, highlights key findings linked to a diagnosis of acute PE, which may be underrecognized.

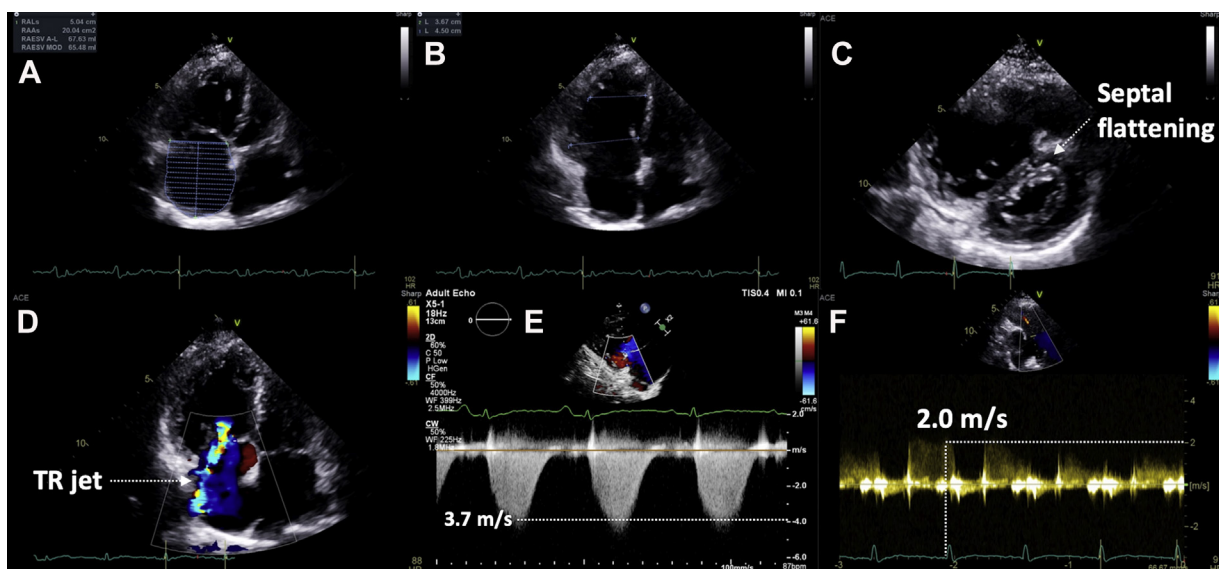


Figure 2 Transthoracic echocardiography. (A) Right atrial and (B) RV dilation shown on apical 4-chamber view. (C) Interventricular septal flattening seen on parasternal short-axis window (arrow). (D) Severe tricuspid regurgitation jet as seen on color flow Doppler on apical 4-chamber view (arrow). (E) Continuous-wave Doppler tracing of tricuspid valve regurgitation, which contributes to the raised calculated RVSP. (F) Continuous-wave Doppler tracing of pulmonary valve regurgitation, which contributes to the high calculated pulmonary artery diastolic pressure. TR, Tricuspid regurgitation.

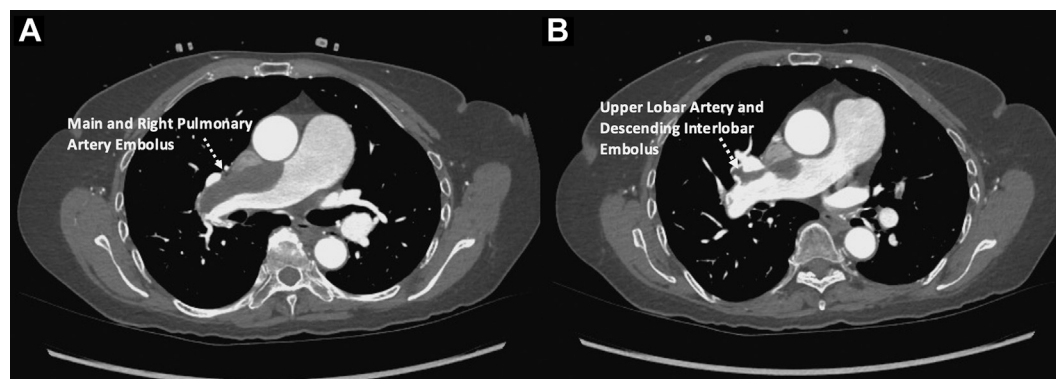


Figure 4 Computed tomography pulmonary angiography, moving caudally from **A** to **B**, revealing a submassive main pulmonary artery embolus (Panel A arrow), extending down into the right pulmonary artery, upper lobar artery, and right descending interlobar arteries (Panel B arrow).

Dilatation of both the right ventricle and atrium, without similar findings seen on the left, should lead clinicians to a diagnosis of raised volumes or pressures within the affected chambers.¹ Other findings on two-dimensional echocardiography include the McConnell's sign, as seen in our case, which is often an early and specific sign suggestive of PE, although this is present in only 12% to 20% of cases.^{2,3} It should be noted that it is merely an appearance of having "spared" the apex, when most likely this relates to tethering of the apex to an otherwise normal or hyperdynamic left ventricle, via transmitted septal forces.⁴ Additionally, absence or presence of pericardial effusion and a depressed RV function are both neither sensitive nor specific. However, when present, this often indicates grave prognosis.¹

Doppler interrogation remains a vital tool that should be incorporated into POCUS within the ED setting. Doppler interrogation through the tricuspid valve using continuous-wave Doppler allows for an estimate of RVSP, which is often raised in the presence of PE (although a caveat would be in cases of severe tricuspid regurgitation, where measurements are often inaccurate).^{1,5-7} Interrogation through the pulmonary valves using pulsed-wave Doppler may help demonstrate the presence of MSN, which has been strongly associated with the presence of both pulmonary hypertension and raised pulmonary vascular resistance.^{1,4-9} Midsystolic notch is caused by reflected waves in the pulmonary vascular system. Normally, reflected waves occur during diastole upon facing distal pulmonary vasculature resistance. However, in the presence of disease, pulmonary vascular tone increases, causing reflections to occur more proximally and arriving earlier, encroaching into systole, and appearing as a notch on the Doppler signal.¹⁰

Presence of the 60/60 sign, where RVSP measures <60 mm Hg and pulmonary ejection acceleration time is <60 msec, is also highly suggestive of an acute PE.^{1,4,7-9} Acute PE, especially large proximal thromboembolism, can cause a sudden increase in the pulmonary vascular resistance without raising pulmonary artery pressure. The stroke volume of a normal right ventricle is usually insufficient to maintain an RVSP >60 mm Hg when there is uncoupling of the right ventricle to the pulmonary artery and the pulmonary artery to the pulmonary vasculature. When left untreated, this can lead to RV collapse.⁴ It should be noted that rarely does the RVSP exceed 40 mm Hg, and any doubling of the RVSP may indicate underlying chronic pulmonary hypertension.¹¹ Despite not having any known comorbidities, our patient was still pending several investigations to rule out prothrombotic conditions such as occult malignancies or

thrombophilia, which may have explained a much higher RVSP on presentation.

CONCLUSION

Although the trifecta of POCUS findings suggestive of PE (i.e., McConnell's sign, MSN, and 60/60 sign) may not always exist concurrently, their presence should be actively sought by clinicians as acute PE can lead to rapid deterioration when diagnosis is delayed. Unfortunately, we are aware that not all POCUS systems are equipped with spectral Doppler function, especially handheld devices, but hopefully our case highlights its utility. We believe that our patient had likely averted a more unfortunate fate following her submassive PE due to the incorporation of Doppler into our POCUS practice.

ACKNOWLEDGMENT

We thank Universiti Teknologi Mara Sungai Buloh and Institut Jantung Neagara for supporting the submission of this manuscript.

SUPPLEMENTARY DATA

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.case.2022.04.009>.

REFERENCES

1. Konstantinides SV, Meyer G, Becattini C, Bueno H, Geersing GJ, Harjola VP, et al. 2019 ESC guidelines for the diagnosis and management of acute pulmonary embolism developed in collaboration with the European Respiratory Society (ERS). *Eur Heart J* 2020;41:543-603.
2. Patra S, Math RS, Shankarappa RK, Agrawal N. McConnell's sign: an early and specific indicator of acute pulmonary embolism. *BMJ Case Rep* 2014; 2014:bcr2013200799.
3. Patel VI, Miles M, Shahangian S, Javien J. McConnell's sign: echocardiography in the management of acute pulmonary embolism. *Clin Case Rep* 2021;9:e04994.
4. Parasuraman S, Walker S, Loudon BL, Gollop ND, Wilson AM, Lowery C, et al. Assessment of pulmonary artery pressure by echocardiography—a comprehensive review. *Int J Cardiol Heart Vasc* 2016;12:45-51.

5. Roberts JD, Forfia PR. Diagnosis and assessment of pulmonary vascular disease by Doppler echocardiography. *Pulm Circ* 2011;1:160-81.
6. Augustine DX, Coates-Bradshaw LD, Willis J, Harkness A, Ring L, Grapsa J, et al. Echocardiographic assessment of pulmonary hypertension: a guideline protocol from the British Society of Echocardiography. *Echo Res Pract* 2018;5:G11-24.
7. Bossone E, D'Andrea A, D'Alto M, Citro R, Argiento P, Ferrara F, et al. Echocardiography in pulmonary arterial hypertension: from diagnosis to prognosis. *J Am Soc Echocardiogr* 2013;26:1-14.
8. Afonso L, Sood A, Akintoye E, Gorcsan J 3rd, Rehman MU, Kumar K, et al. A Doppler echocardiographic pulmonary flow marker of massive or submassive acute pulmonary embolus. *J Am Soc Echocardiogr* 2019;32:799-806.
9. Arkles JS, Opatowsky AR, Ojeda J, Rogers F, Liu T, Prassana V, et al. Shape of the right ventricular Doppler envelope predicts hemodynamics and right heart function in pulmonary hypertension. *Am J Respir Crit Care Med* 2011;183:268-76.
10. Parker MW, Gottbrecht MF, Aurigemma GP. Midsystolic notch and pulmonary hypertension: pathophysiologic mechanism and technical considerations. *J Am Soc Echocardiogr* 2021;34:693-5.
11. Goldhaber SZ, Elliott CG. Acute pulmonary embolism: part I: epidemiology, pathophysiology, and diagnosis. *Circulation* 2003;108:2726-9.