Right Heart Thrombus in Transit on Point-of-Care Ultrasound: A Rare Finding with Key Management Repercussions

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INTRODUCTION

Right heart thrombi are an uncommon finding, with the majority related to the embolization of deep venous thrombosis and pulmonary embolism (PE).[1] They are typically associated with significant hemodynamic compromise and high early mortality.[2] We present a case of a critically ill patient with acute hemodynamic instability in whom the use of point-of-care ultrasound (POCUS) resulted in rapid diagnosis of right heart thrombus and PE and allowed the timely initiation of appropriate treatment.

CASE PRESENTATION

A 47-year-old man presented with 1 week of productive cough and chills, in the setting of 6 months of gradually progressive shortness of breath. He had no recent surgery, trauma, immobilization, or known malignancy and had no known personal or family history of thrombophilia. He was a lifelong nonsmoker. At presentation, he was hypoxic, with an oxygen saturation of 78% on room air, and febrile to 39.2°C. Chest computed tomography showed diffuse pulmonary ground-glass opacification and interlobular septal thickening ("crazy paving") in addition to dense consolidation in the right lower lobe (Figure 1). The patient required noninvasive positive airway pressure, and subsequently intubation, for ventilatory support. Bronchoalveolar lavage was positive for *Streptococcus pneumoniae*, and periodic acid–Schiff staining showed brightly staining globular material consistent with pulmonary alveolar proteinosis. He received whole-lung lavage and inhaled granulocyte-macrophage colony-stimulating factor, in addition to appropriate antibiotics. Transthoracic echocardiography on admission showed normal left ventricular size and function with mildly enlarged right ventricular (RV) chamber size (measured basal RV end-diastolic diameter 48 mm; normal range, 24-42 mm) and normal RV systolic function (Figure 2, Video 1) with normal tricuspid valve lateral annular systolic velocity (0.16 m/sec; normal range, >0.14 m/sec).

On hospital day 7, the patient developed sudden tachycardia (heart rate 130 beats/min) and hypotension (systolic pressure 80 mm Hg). Electrocardiography demonstrated sinus tachycardia with dynamic ST-T-wave changes. POCUS was performed and revealed a large mobile echodensity measuring 41 mm in diameter traversing the tricuspid valve. The right ventricle was mildly enlarged, with a mild decrease in systolic function by visual estimate (Figure 3, Video 2). During point-of-care imaging, the mobile echodensity, presumed to be thrombus, was no longer visualized. There was a sudden worsening of RV dilatation and marked decrease in RV systolic function, suggesting embolization to the pulmonary arteries (Figure 4, Video 3). This was clinically associated with a worsening of tachycardia (heart rate 150 beats/min) and further hypotension, requiring the initiation of vasopressors.

Following multidisciplinary discussion, thrombolysis was not administered because of concerns for pulmonary hemorrhage given known pulmonary alveolar proteinosis. Marked hemodynamic instability was deemed to make the patient a poor candidate for surgical or catheter embolectomy; therefore, he underwent cannulation for venoarterial extracorporeal membrane oxygenation (ECMO). He was continued on therapeutic anticoagulation with heparin. In the following days, he underwent lower extremity ultrasound, which confirmed extensive bilateral deep vein thrombosis. ECMO was successfully weaned, as was vasopressor and inotropic support, and he was successfully decannulated. Intraprocedural transesophageal echocardiography during venoarterial ECMO decannulation 5 days later showed recovery of RV systolic function (RV fractional area change 37%; normal range, >35%) and decrease in RV size by visual estimate (Figure 5, Video 4). No patent foramen ovale was identified on color flow Doppler imaging. Anticoagulation was transitioned to warfarin, and after a prolonged hospital stay, the patient was discharged to a rehabilitation facility.

DISCUSSION

Right heart thrombi are a clinical finding that occur predominantly in the context of PE, where they represent an "embolus in transit" of a deep vein thrombosis, usually from the lower extremities.[1,3] They are a rare finding, present in 3% to 4% of patients with pulmonary embolus who undergo echocardiography,[2,4] but are associated with hemodynamic instability, with up to 18% to 22% of patients with high-risk or massive PE demonstrating right heart thrombi.[4,5]

Echocardiography is the modality of choice for diagnosis of right heart thrombi, though they can also be detected on computed tomography.[4] Right heart thrombi with an embolic source have classically been described as having a worm-like or serpiginous morphology,[6] though they can be more spherical or ball-like, as in our case.[3,7]

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VIDEO HIGHLIGHTS

Video 1: Right ventricle–focused apical four-chamber view demonstrates normal RV systolic function.
Video 2: Subcostal four-chamber view demonstrates a large mobile echodensity traversing the tricuspid valve, with mild RV enlargement and mild decrease in RV function by visual estimate.
Video 3: Subcostal four-chamber view demonstrates a sudden worsening of RV dilatation and systolic function.
Video 4: Midesophageal four chamber view demonstrates an improvement in RV function and decrease in RV size.

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They are typically highly mobile and often prolapse through the tricuspid or pulmonic valves during the cardiac cycle.[1,6] These highly mobile right heart thrombi seen in embolization of deep venous thrombosis have been associated with early thrombus-related mortality of up to 42%, as opposed to 4% early mortality in the comparatively rarer thrombi originating from right heart chambers themselves.[6] Although these highly mobile echodensities may have a characteristic appearance, alternative differential diagnoses, including tumors or endocarditis, must be considered as the clinical situation dictates, and additional image sweeps or unconventional images and windows should be performed to fully assess the density in question and evaluate for additional masses.

POCUS has emerged as a valuable tool to aid in the assessment of critically ill patients, with cardiac POCUS being imperative in those with hemodynamic instability.[8] POCUS can provide rapid identification of potentially catastrophic causes of acute hemodynamic compromise, each with varied emergent interventions required,

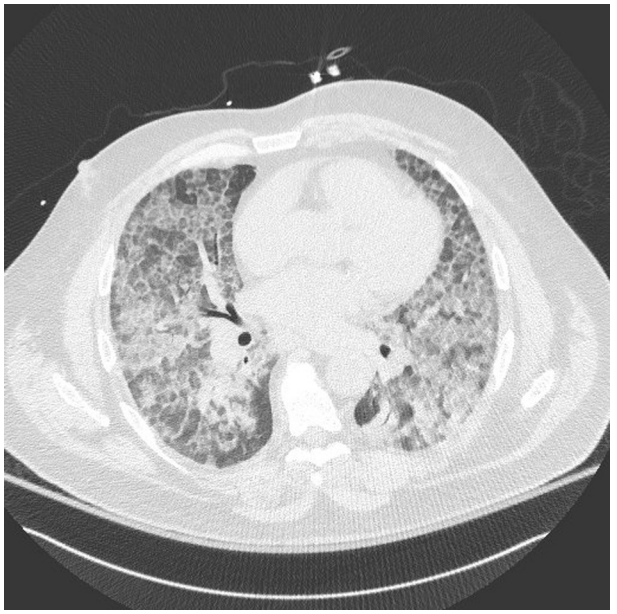

Figure 1 Computed tomography demonstrates ground-glass opacities with interlobular septal thickening consistent with pulmonary alveolar proteinosis.

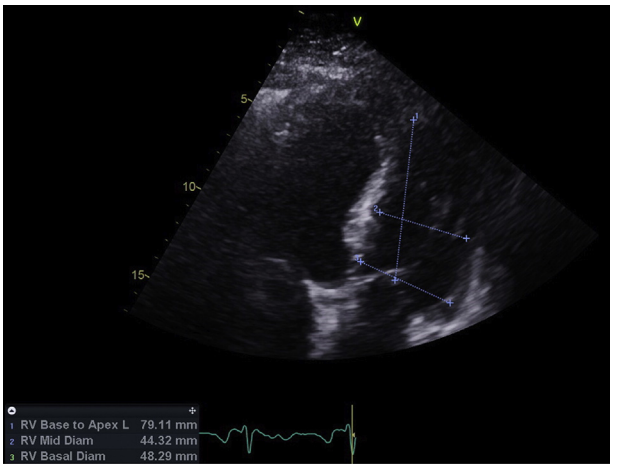

Figure 2 Right ventricle (RV)–focused apical four-chamber view demonstrates mild right ventricular dilatation on admission echocardiogram. *Diam*, Diameter; *L*, length.

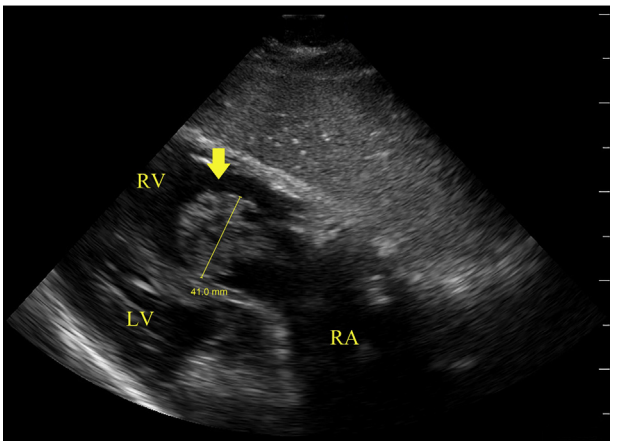

Figure 3 Subcostal view on transthoracic POCUS demonstrates a large echodensity traversing the tricuspid valve *(yellow arrow)*. *LV*, Left ventricle; *RA*, right atrium; *RV*, right ventricle.

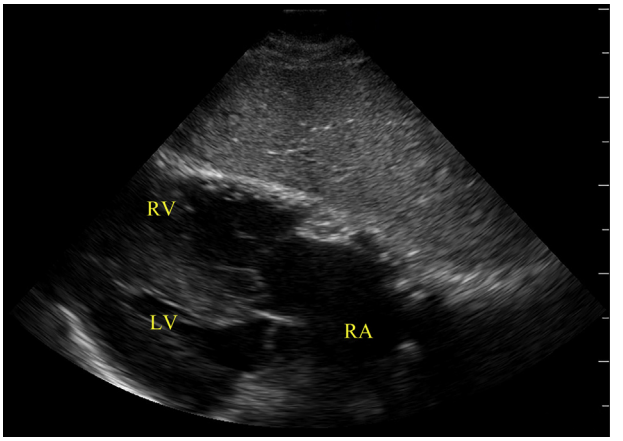

Figure 4 Subcostal view on transthoracic POCUS demonstrates echodensity is no longer visible. *LV*, Left ventricle; *RA*, right atrium; *RV*, right ventricle.

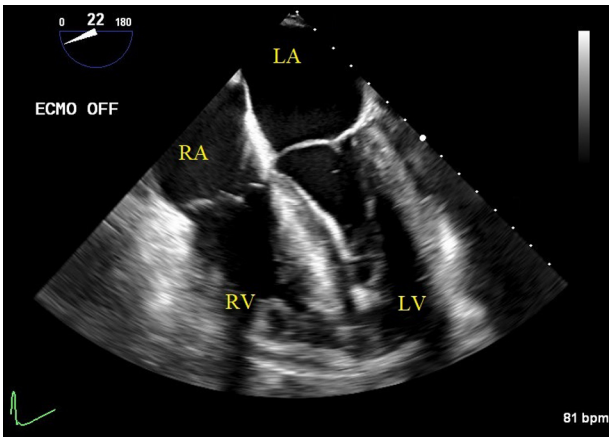

Figure 5 Midesophageal four-chamber view on intraoperative transesophageal echocardiography 5 min after ECMO discontinuation demonstrates improvement in right ventricular function and decrease in right ventricular size. *LA*, Left atrium; *LV*, left ventricle; *RA*, right atrium; *RV*, right ventricle.

including aortic dissection, acute valvular disease, cardiac tamponade, marked left ventricular dysfunction or, as in our case, acute PE.[9] POCUS facilitates observation of many of the standard echocardiographic views, with subcostal imaging being important in critically ill patients in particular if suboptimal image quality is observed with other conventional views, as in our case. Although right heart thrombi are rarely detected on POCUS, the RV pressure overload and dysfunction resulting from acute PE are more commonly seen. Suggestive POCUS findings include a dilated right ventricle with basal right ventricle/left ventricle ratio $>$ 1.0, interventricular septal flattening with "D-shaped" left ventricle on short-axis imaging, decreased tricuspid annular plane systolic excursion or decreased peak systolic velocity of the tricuspid annulus, engorged inferior vena cava with diminished inspiratory collapsibility, and depressed RV free wall contractility compared with RV apex ("McConnell sign").[9] "Triple POCUS," a strategy of POCUS of the lung, heart, and leg veins, has been proposed as a modality that may improve clinical assessment of patients with suspected PE, with the addition of pulmonary ultrasonography for assessment for subpleural infarcts and leg vein ultrasonography for the assessment for deep vein thrombosis. This approach has been shown to increase sensitivity compared with single-organ ultrasonography and may safely rule out PE when combined with other parameters such as negative D-dimer.[10]

In patients with hemodynamic instability thought to relate to PE, the absence of RV overload or dysfunction largely excludes PE as the etiology of the compromise.[9] Therefore, the recommended initial test in a hemodynamically unstable patient suspected of having PE is bedside echocardiography, with confirmatory chest computed tomographic angiography or conventional lower extremity ultrasound sought only after appropriate stabilization has been achieved.[9] In these patients, echocardiographic evidence of RV pressure overload, particularly with PE-specific findings such as right heart thrombi, can justify emergency treatment for PE.[9] In our case, the identification of right heart thrombi on POCUS led to the identification of pulmonary embolic disease and expedited necessary treatment and management decisions without the need for additional emergent investigations. Although POCUS allows rapid, point-of-care assessment, formal echocardiography should be sought once the clinical situation allows.[11]

The optimal treatment of right heart thrombi remains without consensus, though mortality approaches 100% in those who do not receive any treatment.[1] Given the clinical rarity, prior studies have been hampered by small sample sizes and selection bias, and they have differed in their observations and subsequent treatment recommendations. Kinney and Wright[12] in 1989 reported a meta-analysis of 71 studies involving 119 patients, concluding that the efficacies of heparin, thrombolysis, and surgical embolectomy were similar (probabilities of survival were 0.7, 0.62, and 0.62 respectively). In 1989, the European Working Group on Echocardiography reported among 119 patients the superiority of surgical embolectomy compared with full-dose anticoagulation (thrombus-related early mortality rates were 27% and 64%, respectively).[6] Chartier *et al.*[3] in 1999 reported that the choice of therapy had no effect on mortality, but they did advocate for emergency surgery as the preferred treatment and thrombolysis as an alternative. Rose *et al.*[1] in 2002 reported a retrospective pooled analysis of 177 cases and concluded that thrombolysis had a superior association with mortality compared with anticoagulation therapy or surgical embolectomy (mortality rates of 11.3% vs 28.6% and 23.8%, respectively). Torbicki *et al.*[2] in 2003 reported on 43 patients with right heart thrombus from a PE registry and concluded that heparin alone might not be adequate treatment for right heart thrombi. More recently, Athappan *et al.*[13] in 2015 reported on a pooled analysis of 328 patients with right heart thrombi and found significantly higher mortality in those treated with anticoagulation alone compared with surgical embolectomy or thrombolysis (probabilities of survival were 47.7%, 70.45%, and 81.5%, respectively). A number of prospective studies have described the efficacy and safety of thrombolysis for right heart thrombi in massive PE,[7,14] with proceeding to surgical embolectomy in patients with persistent thrombus as a potential algorithm for management.[7] Lastly, ECMO has been described as a viable option for cardiopulmonary support in patients with massive PE.[15] The heterogeneity of the results suggests that, as in our case, a plan of care individualized to each patient agreed upon by a multidisciplinary team on the basis of clinical characteristics and comorbidities is required.

CONCLUSION

Right heart thrombi are a rare echocardiographic finding associated with hemodynamic compromise and high early mortality in PE. As our case demonstrates, POCUS can allow early recognition of this entity in hemodynamically unstable patients, leading to the prompt initiation of treatment without the need for additional testing. The optimal management strategy for mobile right heart thrombi with high embolic risk remains uncertain and therefore should be individualized to each patient on the basis of multidisciplinary discussions.

SUPPLEMENTARY DATA

Supplementary data to this article can be found online at https://doi.org/10.1016/j.case.2022.04.013.

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