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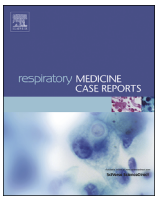

Case Report

Fatal venous air embolism in the setting of hemodialysis and pulmonary hypertension: A point of care ultrasound diagnosis

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A B S T R A C T

Air emboli are rare and often difficult to diagnose. Transesophageal echocardiography remains the most definitive method of diagnosis, but this is not feasible in emergencies.

We present a case of fatal air embolism in the setting of hemodialysis with recent evidence of pulmonary hypertension. The diagnosis was made by visualizing air in the right ventricle using bedside point of care ultrasound (POCUS). While POCUS is not routinely used for the diagnosis of air embolism, its accessibility makes it a powerful yet practical emerging tool for the diagnosis of respiratory and cardiovascular emergencies.

1. Background

The immediate availability of point of care ultrasound (POCUS) makes it a valuable, emerging tool for the evaluation of respiratory and cardiovascular emergencies with increasing interest in this field in the past few decades [1]. In this paper, we present a case of venous air embolism in a hemodialysis patient diagnosed by visualizing air bubbles in the right ventricle (RV) on bedside ultrasonography.

An air embolism is an uncommon but potentially fatal event that can result from air entering the vasculature. Air emboli can be arterial or venous. Venous air emboli can occur when there is a direct communication between a source of air and a vein with the addition of a pressure gradient favoring the passage of air into the vein. Rarely, the use of hemodialysis catheters can cause an air embolism which can in turn lead to potentially devastating clinical consequences [4].

Echocardiography has been used to document the presence of air in the cardiac chambers in some cases of venous air emboli. The detection of air bubbles in cardiac chambers on echocardiography can confirm the diagnosis of an air embolism even in the preclinical setting [2,3]; however, bedside ultrasonography has not been routinely used to diagnose or monitor high-risk patients for venous air emboli. In this article, we describe a case of a fatal venous air embolism in which a bedside ultrasound was used to confirm the diagnosis.

2. Case description

An 82-year-old woman with a past medical history of heart failure with preserved ejection fraction, atrial fibrillation, end-stage renal disease on hemodialysis, chronic obstructive pulmonary disease, and type 2 diabetes mellitus was admitted to the intensive care unit (ICU) for acute on chronic hypoxic respiratory failure. She was admitted to the hospital after a scheduled outpatient endoscopic evaluation of a pancreatic growth, which was complicated by respiratory failure attributed to the initiation of anesthesia. During the

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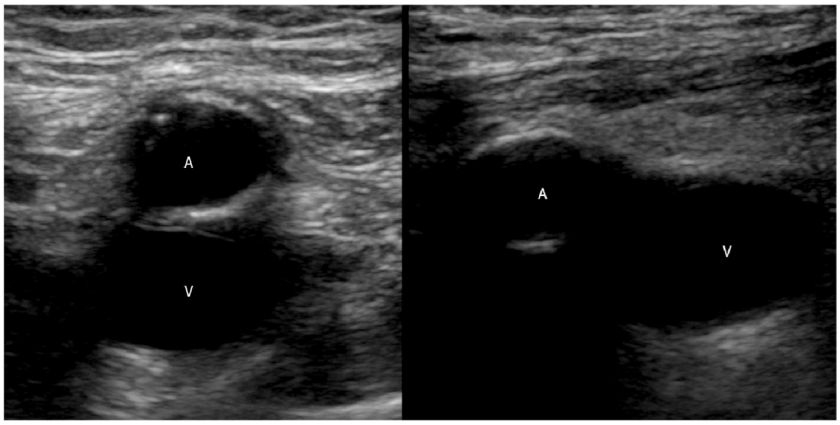

Fig. 1. Patent lower extremity arteries and veins were visualized with no evidence of deep vein thrombosis. A, artery; V, vein.

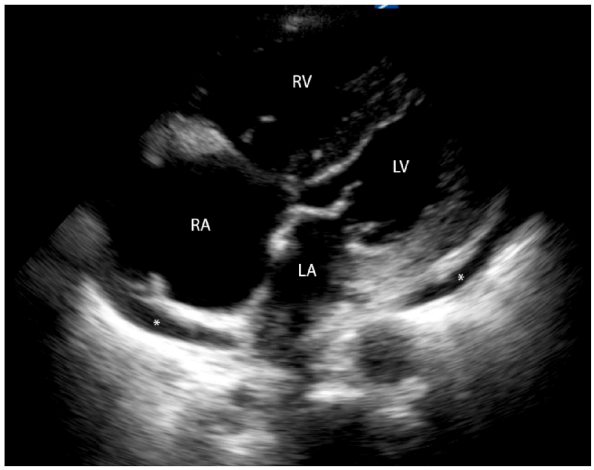

Fig. 2. Apical four-chamber view of the heart on point of care ultrasound on ICU admission. Severely dilated RA and RV, and a small pericardial effusion were seen. The RA appears to bulge into the LA. No air bubbles were visualized in the chambers. RA, right atrium; RV, right ventricle; LA, left atrium; LV, left ventricle; *, small pericardial effusion.

procedure, she was intubated and went into asystole. Return of spontaneous circulation (ROSC) was achieved after one round of cardiopulmonary resuscitation (CPR). She was stabilized and transported to the ICU. Computed tomography angiography was negative for pulmonary emboli. POCUS of lower extremities revealed no evidence of deep vein thrombosis (Fig. 1). POCUS of the heart showed severely elevated RV systolic pressure, moderate to severe RV systolic dysfunction, severe RA and RV dilation (Video 1), and a small pericardial effusion (Fig. 2) with no evidence of tamponade. Additionally, a tricuspid annular plane systolic excursion of 1.2 cm (Fig. 3) was noted. No air bubbles were seen in the chambers at this time. A transthoracic echocardiogram (TTE) three years prior showed normal RV size and systolic function and the patient was not previously known to have pulmonary hypertension.

Supplementary video related to this article can be found at https://doi.org/10.1016/j.rmcr.2023.101819

The next day, since her mental status completely recovered, she underwent routine dialysis with plans to extubate following the completion of dialysis. 30 minutes after initiation of hemodialysis, she became hypotensive and transiently unresponsive prompting re-initiation of intravenous (IV) norepinephrine. Dialysis was paused and, while running saline through the dialysis circuit, a column of air was noted in the dialysis line. She was placed on her left side and monitored for hemodynamic instability. Subsequently, vasoactive agent requirements continued to rise, and the patient became unresponsive. POCUS revealed RV strain and air bubbles in the RV outflow tract, indicating venous air embolism as the likely culprit (Video 2, Fig. 4). She then required multiple vasoactive agents with persistence of her hypotension. Shortly afterwards, she became pulseless, and seven cycles of CPR were performed. Eventually, the patient expired despite resuscitative efforts.

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3. Discussion

Air emboli are a rare complication of using central venous catheters as vascular access for hemodialysis [4]. We present a case of cardiac arrest secondary to venous air embolism in the setting of hemodialysis in a patient with a likely diagnosis of pulmonary hypertension and acute on chronic RV failure. Most cases of air embolism cause minimal or no symptoms. Rarely, venous air embolism can

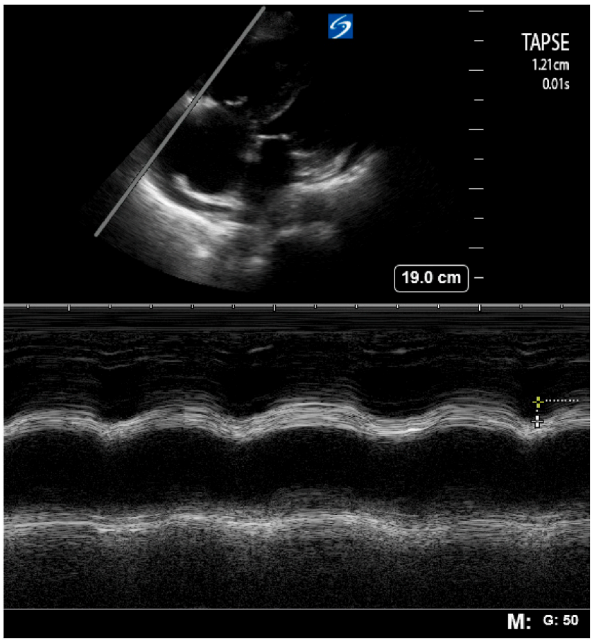

Fig. 3. TAPSE of 1.21 cm. TAPSE, tricuspid annular plane systolic excursion.

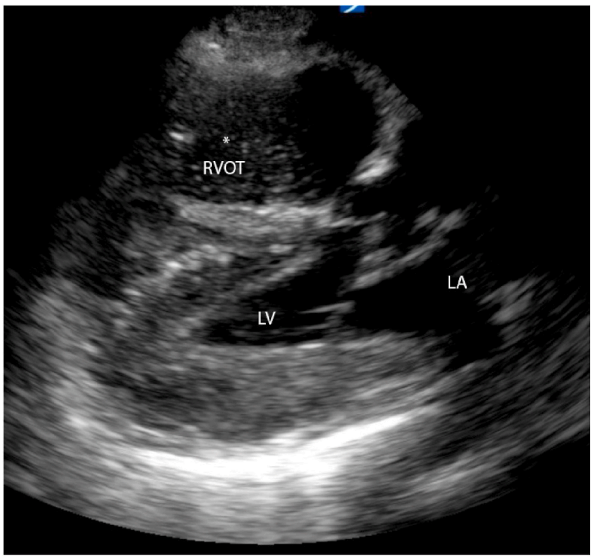

Fig. 4. Parasternal long view of the heart on point of care ultrasound during hemodialysis pause just before cardiac arrest. Severely dilated RVOT with visualization of air bubbles. RVOT, right ventricular outflow tract; LA, left atrium; LV, left ventricle; *, air bubbles.

be immediately life-threatening with a presentation of hemodynamic collapse. The hemodynamic significance of any venous air embolism depends on both the rate and volume of air entrainment in the vasculature in addition to the patient's baseline cardiac function [5]. Our patient had signs of pulmonary hypertension with RV failure on her most recent echocardiogram, just before developing a venous air embolism. This baseline likely diagnosis of pulmonary hypertension likely resulted from other comorbidities such as end-stage renal disease, COPD, and heart failure. Her initial cardiac arrest episode was likely a consequence of the hemodynamic impact of intubation in the setting of undiagnosed pulmonary hypertension. We argue that the presence of pulmonary hypertension in this patient could have contributed to her outcome. Pulmonary hypertension can also be exacerbated by pulmonary vasoconstriction in response to the presence of air in the pulmonary arterioles [4,6].

The diagnosis of a venous air embolism can be challenging, and detection of air in the heart chambers by transesophageal echocardiography (TEE) remains the most definitive method of diagnosis [7,8]. TEE can detect as little as 0.02 ml/kg of air [8,9]; however, TEE is often not readily available or feasible in guiding decision-making during emergencies. In our patient, venous air embolism was diagnosed by POCUS. To our knowledge, the use of bedside ultrasonography to aid in the diagnosis of an air embolism has rarely been

described in the literature. In our patient, severe RV dilatation and air bubbles in the RVOT were visualized (Fig. 4). These findings are consistent with those in prior case reports of air emboli diagnosed using POCUS, including dilatation of the RV and the presence of gas bubbles moving between heart chambers [10–12]. Of note, it is possible that the air bubbles noted in our case are due to infusion of medications in the setting of low cardiac output state. However, this finding was not detected during the initial POCUS we performed after the patient arrived at the ICU. Additionally, a column of air was detected in the hemodialysis circuit and the location of air bubbles in the RV outflow tract matches the clinical picture of an acute on chronic RV failure secondary to RV outflow tract obstruction. POCUS offers clinicians immediate visualization while avoiding additional wait time associated with other formal imaging studies [1]. Several studies investigated the utility of bedside ultrasonography in guiding clinical decisions compared to other methods [13].

Management of a venous air embolism includes immediately placing the patient in Trendelenburg position or in left lateral decubitus head down position, along with starting hemodynamic support. Definitive therapy includes hyperbaric oxygen and withdrawal of air from the RA [14]. Unfortunately, the patient decompensated rapidly, allowing no time to attempt the insertion of a central venous catheter or Swan-Ganz catheter for more definitive therapy. Of note, some authors questioned the benefit of repositioning patients with air emboli in reducing adverse outcomes [15,16]. Further studies on the management of air emboli have yet to be performed.

Our case highlights the utility of POCUS in diagnosing a venous air embolism in an emergently decompensating patient. In the delicate hemodynamic setting of pulmonary hypertension, an air embolism can be the tipping point toward instability. POCUS is a powerful, yet accessible tool that should be considered to aid medical decision-making in critical care [1]. Further studies are needed to investigate the utility of POCUS in monitoring and diagnosing patients at high risk of air embolism, including hemodialysis patients [17].

Declaration of competing interest

We declare that this manuscript is original, has not been published before, and is not currently being considered for publication elsewhere. We know of no conflicts of interest associated with this publication, and there has been no financial support for this work that could have influenced its outcome.

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