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Case Report

Acute right ventricular failure and pulseless electrical activity arrest following auto-transfusion of blood

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ABSTRACT

Air embolism is a rare but potentially catastrophic complication of interventional procedures. The occurrence of acute right ventricular dysfunction during intraoperative auto-transfusion of blood, presumably related to pulmonary embolism of agitated air microbubbles and microthrombi, is less commonly recognized. We report a case of auto-transfusion complicated by acute right ventricular failure and pulseless electrical activity arrest. Auto-transfusion of recovered blood is a practical solution to reduce need for post-procedure allogenic transfusions. Although such interventions are frequently performed without complications, they do have inherent risks that should be readily acknowledged. This case clearly describes a severe complication and sequelae of auto-transfusion.

<**Learning objective:** Auto-transfusion of recovered blood is commonly performed in surgical and interventional procedures to reduce the need for allogenic transfusion. Despite this benefit, the risks and complications of auto-transfusion can be severe and must be considered. We report a case of intraprocedural auto-transfusion resulting in introduction of air emboli and subsequent cardiac arrest. Additionally, we provide a brief review of air emboli and underlying pathophysiology that leads to cardiovascular decline.>

Introduction

Air embolism is an uncommon but life-threatening event that occurs when air is introduced into the vasculature [1]. Air emboli are most commonly described in the literature as a complication of cardiac surgery involving cardiopulmonary bypass, as well as neurosurgical and otolaryngologic operations [2]. They rarely occur with routine infusion of intravenous fluids and blood products. However, introduction of air emboli has been described during autologous blood transfusion with intraoperative blood salvage devices [3]. We present a case of acute right ventricular failure and pulseless electrical activity arrest, following auto-transfusion of aspirated blood from iatrogenic pericardial effusion.

Case report

The patient was a 78-year-old woman with past medical history of paroxysmal atrial fibrillation, transient ischemic attack (TIA), hypertension, and gastrointestinal bleeds who was admitted to our hospital for placement of Watchman left atrial appendage (LAA) closure device (Atritech, Inc., Minneapolis, MN, USA).

Preprocedure transthoracic echocardiogram (TEE) was performed which provided visualization of single-lobed "chicken-wing" shaped LAA. Femoral central venous access was obtained. Heparin 7000 units was administered and acute clotting time (ACT) was measured at 333 s and 303 s on repeat. Under fluoroscopic and TEE guidance, transseptal puncture was performed, and Watchman device was deployed in the LAA. Contrast extravasation into the pericardial space was noted, which was concerning for LAA laceration (Fig. 1A). Intra-procedure TEE confirmed an expanding pericardial effusion with hypotension (Fig. 1B). The patient received intravenous fluids and vasopressor support. The subxiphoid area was prepared and draped and

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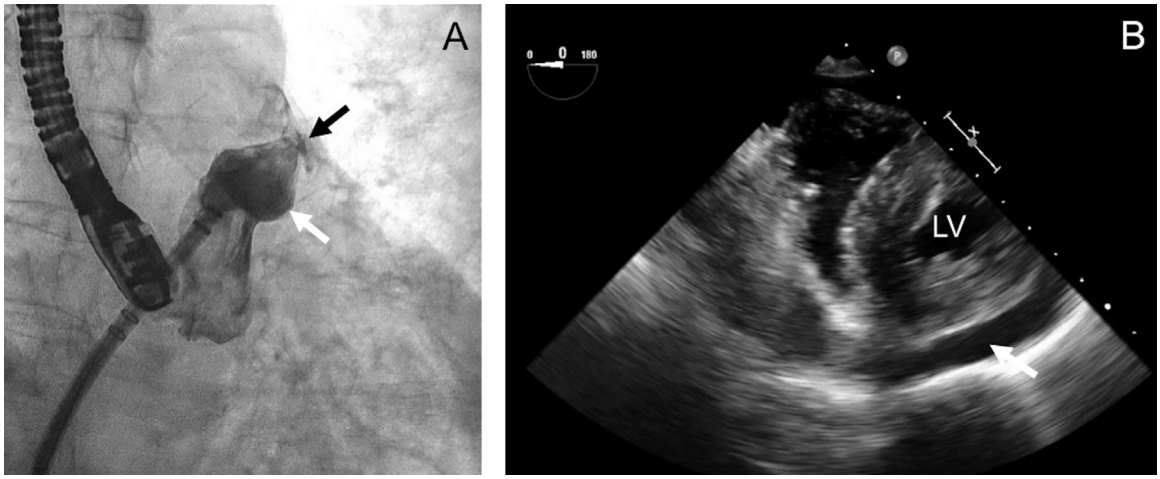

Fig. 1. Fluoroscopic image depicting deployed Watchman device in left atrial appendage (white arrow) with contrast extravasation (black arrow) into pericardial space (A). Intra-procedure transesophageal echocardiographic image of pericardial effusion (white arrow) following Watchman deployment (B).
LV, left ventricle.

emergent pericardiocentesis was performed resulting in improvement in blood pressure. Protamine sulfate was administered to reverse heparin anticoagulation, while a total of 1200 mL of blood was eventually aspirated from the pericardial space. As the pericardial space was being drained using 60 cc syringes, simultaneously, approximately 800 mL of aspirated blood were autotransfused via the femoral vein with care to prevent any obvious injection of air. Protamine 50 mg was administered and ACT corrected to 145 s.

Following auto-transfusion of blood, the patient acutely became hemodynamically unstable. TEE showed complete resolution of pericardial effusion but right ventricular enlargement and marked systolic dysfunction with septal flattening were noted, suggestive of right ventricular volume and pressure overload (Fig. 2A). A small underfilled left ventricular chamber due to loss of preload was also seen. The patient continued to decompensate and ultimately PEA arrested despite inotropic and vasopressor support. Cardiopulmonary resuscitation was performed with two minutes of chest compressions. The patient received bicarbonate, calcium gluconate, epinephrine, norepinephrine, and intravenous fluids. Return of spontaneous circulation was achieved and improvement in right ventricular systolic function with return of preload to the left ventricle (Fig. 2B). Based on the temporality of the hemodynamic decompensation, despite the volume resuscitation and evacuation of the pericardial space, immediately following the auto-transfusion of blood, the acute right ventricular failure was thought to be due to introduction of air microemboli, microthrombi, and/or other sediments in hemolyzed blood during reinfusion of aspirated pericardial blood.

Appropriate positioning of the Watchman device was reconfirmed, the device released and the remainder of the access/delivery apparatus removed from the left atrium. Right heart catheterization for hemodynamic evaluation showed right atrial pressure 19/16 mmHg (mean 13), right ventricular pressure 43/10 mmHg (end-diastolic 16), pulmonary capillary wedge pressure 15/16 mmHg (mean 15), and pulmonary artery pressure 43/23 mmHg (mean 29). Pulmonary arteriogram was performed that showed absence of obvious perfusion defects in the pulmonary

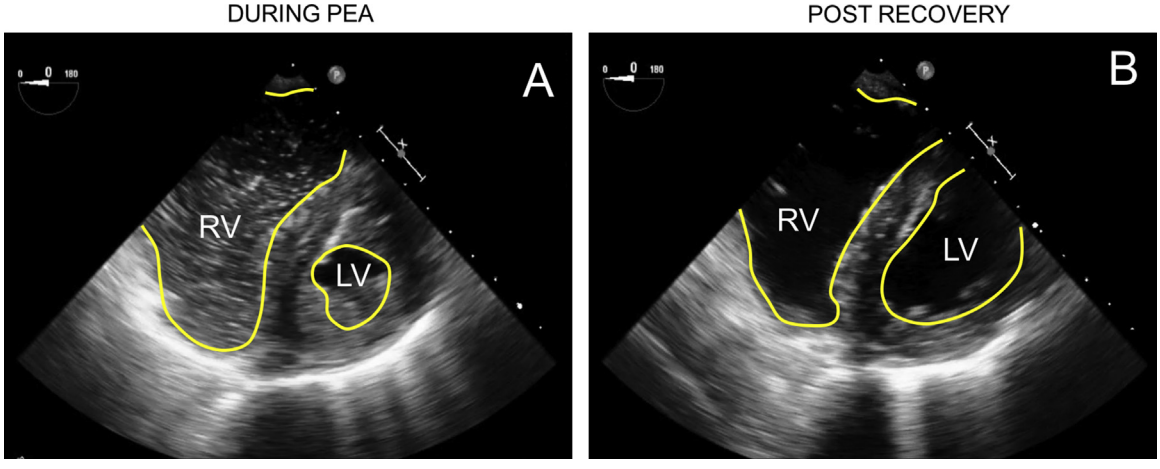

Fig. 2. Transesophageal echocardiographic (TEE) video clip during pulseless electrical activity (PEA) arrest showing right ventricle enlargement, interventricular septal flattening, and reduced left ventricle chamber volume (A). Post recovery TEE video clip with reduced right ventricle chamber volume and improved left ventricle filling (B). Thumbnail images represent diastolic still frames from corresponding video clips, described above. Yellow line within thumbnail images demarcates endocardial border of right and left ventricles, during PEA and post recovery.
LV, left ventricle; RV right ventricle.

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