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Case report

Bubbles in the Heart: A Case of Venous Air Thromboembolism

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ABSTRACT

Introduction: Venous air embolism (VAE) due to central venous catheter (CVC) placement is a rare but preventable complication which is potentially fatal. We describe a case highlighting unique patient characteristics which increase the risk of developing VAE.

Case description: A sixty-year-old gentleman was admitted to the hospital with dyspnea and altered mental status. His comorbidities include cancer of the neck and tongue, currently in remission, and schizophrenia. On presentation, he was found to be in acute respiratory failure, due to pneumonia, and required mechanical ventilation. Following extubation, his CVC was removed from the right internal jugular vein. While ambulating around the unit, he experienced a coughing fit and dizziness. He rapidly developed cardiopulmonary collapse requiring re-intubation and vasopressor support. Chest x-ray demonstrated a radiolucent column along the lateral aspect of the right neck. Due to concern for VAE, an echocardiogram was performed, revealing multiple air-bubbles in the right and left chambers of the heart.

Discussion: Our patient was predisposed to developing VAE due to the extensive radiation induced skin changes, from his cancer treatment, on the neck and upper thorax. This resulted in loss of underlying subcutaneous tissue and decreased skin pliability. He had a large, open puncture wound at the catheter site on his neck, probably resulting in air entry. Anxiety and agitation, due to schizophrenia, made it difficult to maintain our patient in a supine or Trendelenburg position following CVC removal. This case highlights the importance of recognizing patient factors that may increase the risk of VAE.

1. Presentation

We present the case of a sixty-year-old gentleman admitted to the hospital with a chief complaint of dyspnea and altered mental status. His relevant comorbidities included cancer of the tongue and neck, chronic obstructive pulmonary disease, schizophrenia, and anxiety. Emergency department course was remarkable for acute exacerbation of chronic hypoxic and hypercapnic respiratory failure, which required intubation with mechanical ventilation. Subsequently, the patient was admitted to the intensive care unit (ICU) for ventilatory and circulatory support. Broad spectrum antibiotics were initiated for pneumonia. His sputum cultures grew *Streptococcus pneumoniae* and a nasopharyngeal swab was positive for *influenza B*. In the interim, due to agitation and anxiety, he required pharmacologic therapy to allow for adequate oxygenation and ventilation.

Following good response to treatment, a trial of extubation was attempted on day eight. He responded well, and the following day, his central venous catheter (CVC) was removed from the right internal jugular vein in anticipation of transfer to an intermediate care unit. He was hemodynamically stable and was oxygenating well on room air at the time of CVC removal. However, shortly after the removal of his CVC, he ambulated around the unit with assistance due to ongoing restlessness and agitation related to his underlying psychiatric disease.

Approximately 20 min following CVC removal, our patient experienced a coughing spell and dizziness. He rapidly developed cardiopulmonary collapse as evident by atrial fibrillation with rapid ventricular response, hypotension, hypoxia, and respiratory failure. Thus, he required re-intubation and vasopressor support. Pulmonary embolism and venous air embolism (VAE) were considered as potential causes of his acute decompensation; hence, a stat chest X-ray and bedside echocardiogram were performed. Heparin infusion was empirically initiated for possible pulmonary embolism.

His echocardiogram (Figs. 1 and 2) revealed multiple air-bubbles in the right and left chambers of the heart due to a patent foramen

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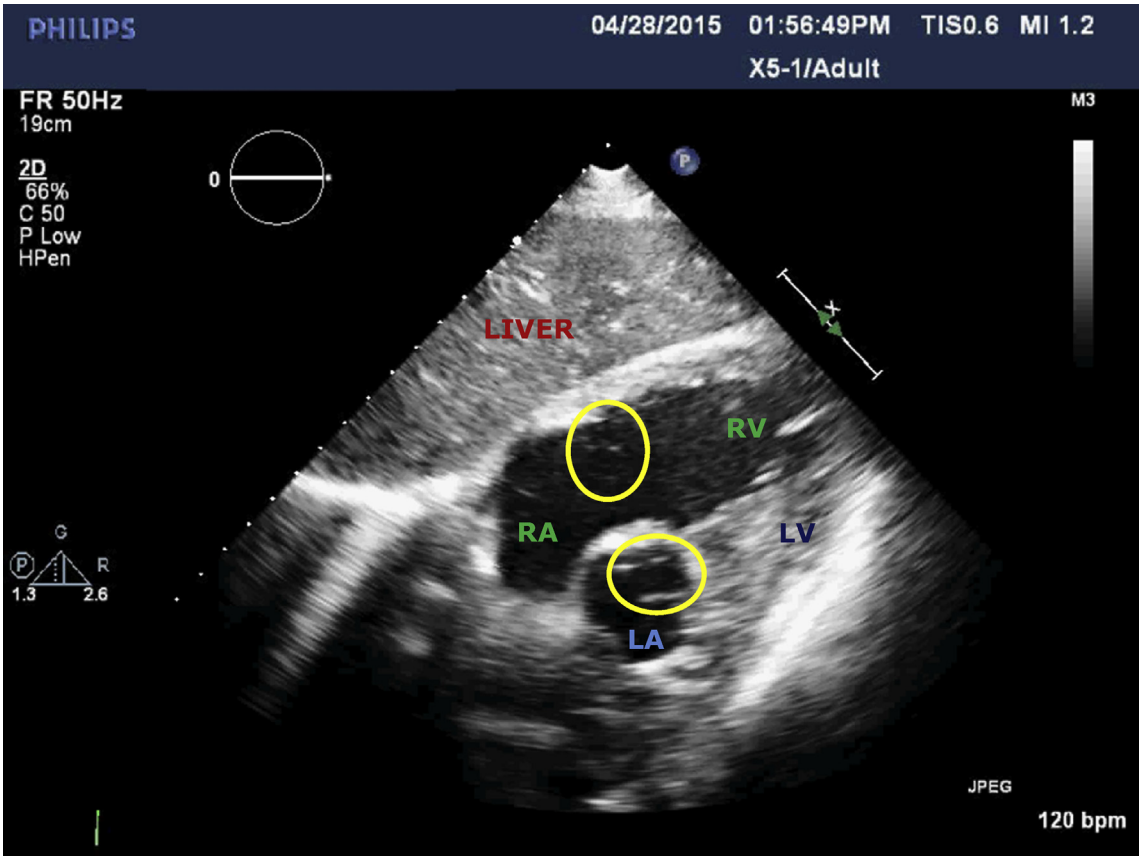

Fig. 1. Echocardiogram demonstrating multiple air-bubbles (yellow circles) in the right and left chambers of the heart. RA = right atrium, RV = right ventricle, LA = left atrium, LV = left ventricle.

ovale. These bubbles dissipated with time during the course of the echocardiogram study, without the use of agitated saline. The chest X-ray (Fig. 3) demonstrated a radiolucent column along the lateral aspect of the right neck where his previous CVC had been located. Both of these findings were thought to be consistent with VAE. A computerized tomography (*CT*) angiogram of the chest excluded pulmonary embolism; therefore, his heparin infusion was discontinued. He was immediately placed in the left lateral decubitus position. He also received 100% fraction of inspired oxygen (FiO2) through the ventilator. The patient ultimately recovered from this acute episode. Within twenty-four hours he was on minimal ventilator settings, and his vasopressors were weaned.

2. Discussion

VAE due to CVC is a preventable, hospital-acquired complication that is potentially fatal [1]. The number of cases of VAE due to CVC is underestimated due to the transient and nonspecific nature of its presentation [1]. It is also very difficult to diagnose with confirmatory testing. Estimates of the frequency of VAE related to CVCs range from 1 in 47 to 1 in 3000 cases [2]. However, its corresponding mortality rate ranges from 23% to 50% [2].

The Pennsylvania Patient Safety Authority (PSA) review in 2012 noted that the highest frequency of confirmed or suspected VAE occurred during insertion and removal of CVCs [2]. According to Brockmeyer et al. the most common site of CVC placement complicated by VAE is in the subclavian vein [3]. This finding may simply be a consequence of the higher number of CVCs inserted in this location in their study [3].

Developing clinically significant VAE is dependent on the rate and volume of air introduced into the circulation. Studies using canine models show that the ability of the lungs to filter air from the venous vasculature is overwhelmed when more than 0.30 mL/kg/minute are introduced into the system causing tissue ischemia [3]. A volume of 300–500 mL infused at a rate of 100 mL/s is estimated to be fatal, and this rate can occur using a 14 G catheter with pressure gradient of 5 cm H_2O [3]. The diversity of presenting symptoms is reflected by the movement of the air within the vasculature. A patient may present with symptoms of an ischemic stroke, cardiopulmonary collapse, severe chest pain, or dyspnea [1,3].

VAE related to CVCs occurs at the time of insertion, during CVC use, and at the time of removal. Common factors identified by the Pennsylvania PSA contributing to VAE due to CVCs included inadvertent catheter placement in the artery, injection of air, failure to occlude needle hub until the catheter was capped, upright positioning of the patient, and failure to perform Valsalva maneuver at the time of removal [2]. Peter and Saxman in 2003 noted that only 26% of physicians cited concern for the occurrence of VAE during CVC removal [5]. Additionally, they do not take precautions for its prevention [1]. Despite organization-based guidance on removal of CVCs, only 31% of critical care nurses reported that they always implement these recommendations [1].

Treatment for VAE is supportive. Using the Durant maneuver, the patient is placed on the left lateral decubitus position with the head lying down [3]. This positioning helps to keep the trapped air within the heart away from the right ventricle outflow tract and may consequently reduce or dislodge the blockage caused by air bubbles within the vasculature [3]. It is also recommended that patients receive high flow oxygenation. Hyperbaric oxygen has