CASE REPORT

Venous and arterial air embolism: a rare phenomenon with fatal consequences

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SUMMARY

Air embolism is often an iatrogenic complication which may occur in venous or arterial circulation depending on the port of entry. We present two cases in which air embolism occurred in venous and arterial circulation after contrast medium injection (CMI) and coronary artery bypass graft (CABG) surgery, respectively. In one case, accumulation of air bubbles was observed in the pulmonary artery after CMI. This was attributed to inadvertent injection of air owing to improper connection of the injector and the catheter. The patient was managed with 100% oxygen in the Trendelenburg and left lateral decubitus position. Repeat imaging demonstrated resorption of the emboli. In another case, air was introduced during CABG in the left atrium and ventricle. Immediate suction of air was attempted however, the patient developed cardiogenic shock requiring vasopressors, and subsequently seizures and coma due to diffuse ischaemic stroke. The patient eventually expired.

BACKGROUND

Air embolism in most cases is iatrogenic in nature and may be life-threatening. The symptoms vary according to the amount of air instilled and the end location of the air bubbles, hence a high level of vigilance needs to be maintained.^{1 2} Awareness of the possible sources, its implications in arterial and venous circulation and the timely management options can help prevent or reduce the risk of potentially catastrophic and fatal outcomes.¹

CASE PRESENTATION

Patient 1

A 65-year-old obese woman with medical history significant for chronic obstructive pulmonary disease, breast cancer status postchemotherapy and coronary artery disease presented with an 8-day history of dyspnoea, non-productive cough and pleuritic chest pain. On arrival, she was noted to have sinus tachycardia and mild respiratory distress, albeit saturating 94% on room air. Her physical examination revealed diffuse expiratory wheezing but was otherwise unremarkable. Laboratory findings and chest radiograph were normal. Chest CT angiography (CTA) was obtained due to a high pretest probability of pulmonary embolism given her history and presentation. Although the CTA was unrevealing for thrombus, two air bubbles in the main pulmonary outflow tract measuring 1.3 and 1.0 cm, respectively, were noted (figure 1). The CT technician reported a leakage of the contrast from one of the hubs on the venous cannula at the

time of contrast injection which likely led to the introduction of air in the venous circulation. Supplemental 100% oxygen via non-rebreather was initiated, and she was placed in the Trendelenburg and left lateral decubitus position. The patient was subsequently transferred to the intensive care unit (ICU) for close monitoring. In the ICU, she was treated with nebulizers and intravenous glucocorticoids with improvement in her symptoms. Repeat CTA chest obtained 12 hours later showed no evidence of air embolism or peripheral thrombosis. She was monitored for 2 days and remained haemodynamically stable. She was subsequently discharged home.

Patient 2

An 81-year-old man with a medical history of hypertension, previous myocardial infarction, severe aortic stenosis and aortic regurgitation presented with symptoms of angina, dyspnoea on exertion and presyncope. On arrival, the patient was in normal sinus rhythm and in no respiratory distress. Physical examination was significant for grade III/VI holosystolic murmur at the right upper sternal border. Transthoracic echocardiogram (TTE) and coronary angiography were obtained, which showed severe aortic stenosis with triple vessel coronary artery disease warranting coronary artery bypass grafting (CABG) and aortic valve replacement. CABG was complicated by

Figure 1 CT angiography of the chest showing two discrete collections of air measuring 1.3 and 1.0 cm within the main pulmonary outflow tract and a single air locule within the right lower lobe pulmonary artery.

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Figure 2 CT head without contrast showing extensive areas of hypodensity in bilateral cerebral hemispheres and anterior and posterior circulation territories.

unexpected air entrapment into the arterial limb of the cardiopulmonary bypass circuit due to unclear source. Transesophageal echocardiogram (TEE) confirmed air in the left atrium and left ventricle. Immediate attempts were made to suction the air from the arterial cannula. The patient was subsequently in cardiogenic shock requiring vasopressor support with low dose epinephrine, and milrinone and intravascular volume repletion to maintain cardiac index. He remained in coma after the procedure and ~3 days later, he was noted to have seizure-like activity. CT head without contrast demonstrated diffuse bilateral cerebral infarcts with increasing oedema consistent with air embolism (figure 2). Family decided to maintain comfort measures only, and soon thereafter the patient succumbed to his ailment.

DISCUSSION

Air embolism formation requires a pressure gradient and a connection between the gas and the blood vessel.³ These air bubbles can then migrate to various vital organs causing significant morbidity and mortality. Arterial air embolism may result as a complication of esophagogastrosocopy, bronchosocopy, craniotomies and CABG. The air may travel in a retrograde fashion via various arteries up to the cerebral circulation or alternatively, it may travel to the left ventricle and subsequently to the coronary arteries disrupting coronary perfusion;^{1 4} whereas venous air embolism is commonly caused due to intravascular catheters such as peripheral and central venous cannula including Hickman catheters, pulmonary artery catheters, haemodialysis catheters and pressurised infusion systems.⁵ Air embolism in the venous circulation travels to the right side of the heart and eventually to the pulmonary circulation.¹ Additionally, large venous emboli may cause paradoxical arterial embolisation due to a sudden increase in right atrial pressure leading to right to left shunting. This may occur through either a patent foramen ovale

or across the pulmonary capillary bed allowing the air to enter the arterial circulation.⁶

Air embolism not only causes perfusion defects in the vasculature but also creates a surge of inflammatory response. The degree of physiologic impairment depends on the volume of air, rate at which it is introduced and the end location.^{1 7} Generally, a small amount of air is benign in the venous circulation and is absorbed without any adverse effect but large amount, 300–500 mL of air at a rate of 100 mL/s may result in right ventricular strain, cardiogenic shock and death.⁸ Contrarily, even a small amount of air in the arterial circulation can be fatal and lead to myocardial infarction, heart failure, dysrhythmias, motor deficits, convulsions or coma.⁹

The diagnosis of air embolism requires a high index of suspicion and should be considered in the presence of sudden cardiovascular and/or neurological complications after a high-risk invasive procedure. Electrocardiogram changes suggestive of right heart strain, decrease in end tidal carbon dioxide due to increased dead space, ‘mill-wheel’ murmur and aspiration of bubbles through a central catheter can all aid in the diagnosis of venous embolism.^{3 7} TEE is the most sensitive test in detecting intracardiac gas, detecting as little as 0.02 mL/kg of air and air bubbles as small as 5–10 μm .⁸ If neurological findings are present and an air embolism to the cerebral arteries is suspected, CT head without contrast is a fast and often first-line means of diagnosis.¹⁰

Treatment should be initiated immediately on sufficient suspicion of air embolism. Supportive treatment with 100% oxygen should be started promptly regardless of the type of embolism. Additionally, in the setting of haemodynamic compromise, intravascular volume repletion and intravenous catecholamines should be initiated concurrently. In an event of a venous embolism, the patient should be placed in a Trendelenburg and left lateral decubitus position to let the air travel towards the right ventricle apex, thereby preventing or relieving the intrapulmonary vascular obstruction.¹¹ In the case of a cerebral arterial embolism, the patient should also be placed in the Trendelenburg position to prevent cerebral oedema and hyperbaric therapy should be considered within the first 30 hours as it has shown to be effective in reversing neurological deficits.¹² Other treatments such as intravenous lidocaine, corticosteroids and heparin have been suggested for the management of arterial air embolism but none of these have been shown to be effective.⁷

Learning points

- ▶ Air embolism is often an iatrogenic complication and an increased awareness, alertness and vigilance is required for timely diagnosis and management to prevent considerable morbidity and mortality.
- ▶ TEE is a sensitive and definitive test for cardiopulmonary embolism while CT head without contrast is the test of choice for cerebral arterial embolism.
- ▶ For venous embolism, a patient should be immediately placed in the Trendelenburg and left lateral decubitus position with 100% oxygen supplementation.
- ▶ Management of cerebral arterial embolism includes the Trendelenburg position and hyperbaric therapy also should be considered if there is no contraindication within the first 30 hours.

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