

Venous air embolism: ultrasonographic diagnosis and treatment with hyperbaric oxygen therapy

A. Brodbeck^{1,*}, P. Bothma¹ and J. Pease²

¹James Paget University Hospitals, Anaesthetic Department, Gorleston, UK and ²Norfolk and Norwich University Hospital, Department of medicine, Norwich, UK

*Corresponding author. E-mail: andreas.brodbeck@jpaget.nhs.uk

Abstract

A man with neuromuscular respiratory failure requiring intubation and ventilation suffered a venous air embolism during inadvertent administration of 5 ml of air. Ultrasound (US) imaging confirmed an air embolus in the left subclavian vein, which was only partially treated by US-guided aspiration. The embolus completely resolved on US imaging during hyperbaric oxygen therapy, and the patient recovered with no complications secondary to the embolism. Venous air embolism is under-recognised, and can cause significant neurological morbidity and death if untreated. When available, urgent hyperbaric oxygen therapy appears to be an effective approach.

Keywords: air embolus; hyperbaric oxygen; ultrasound

Gas emboli, whether arterial or venous, can have significant neurological complications. Whilst seemingly rare, it is important to treat them promptly, and to have a high degree of clinical suspicion for at-risk patients. Whilst prevention of air emboli is preferable, interventional radiology-guided aspiration or hyperbaric oxygen therapy (HBOT) are treatment options if such an event is suspected. Much literature supports the efficacy of HBOT as a treatment for cerebral arterial air emboli, but less for cerebral venous air emboli. Aspirating air from a central line is often the first approach. We report an example of a patient in whom aspiration under ultrasound (US) guidance failed, but subsequent HBOT was successful. Aspiration may not be successful and should be reconsidered if such attempts would delay transfer to an HBOT centre.

A 71-yr-old gentleman presented with a 1 week history of ascending lower limb weakness and shortness of breath (written consent given by patient). A CT pulmonary angiogram was unremarkable and non-invasive ventilation was required.

He was treated for Guillain-Barré syndrome/acute motor and sensory axonal polyneuropathy. He became increasingly hypoxaemic, necessitating urgent tracheal intubation and resuscitation, during which he was inadvertently administered 5 ml of air from an empty syringe. Subsequent CT imaging confirmed an air embolus in the left subclavian vein posing the risk of paradoxical embolism or retrograde cerebral venous embolism. US-guided aspiration was attempted, removing ~1 ml of air. To minimise the risk of embolism and subsequent neurological complications, he was treated prophylactically with HBOT. There were no known contraindications to hyperbaric treatment and transfer to the nearest interventional radiology unit would require inter-hospital transport, so he underwent the United States Navy Table 5 protocol (120 min regime) in our Level I (ICU-capable) hyperbaric chamber. On commencing treatment, US imaging in the chamber confirmed some air was still visible in his left subclavian vein (Fig. 1), which subsequently disappeared upon

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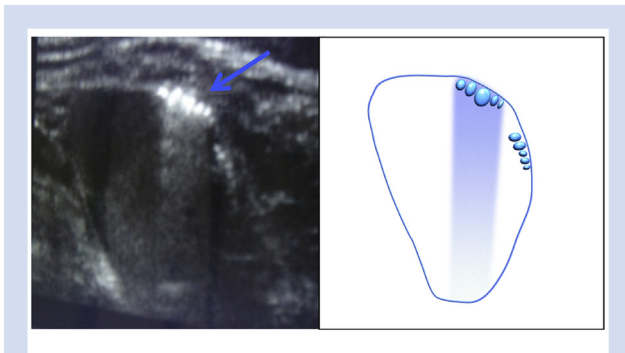


Fig 1. Ultrasound image of subclavian vein with air reflection at anterior wall (arrow)—horizontal, slightly oblique, supraclavicular view.

pressurisation. With reduction of the bubble volume achieved, residual air should be able to flow to the pulmonary capillary system or be safely absorbed.

Documented cerebral gas emboli are rare, with the reported incidence of symptomatic cerebral gas embolism requiring HBOT ranging from 2.5 per 100 000 hospital admissions in Paris and Melbourne to even fewer according to reported data for the UK from the British Hyperbaric Association.^{1,2} Data about air embolism in general gained from the Case Mix Programme of the Intensive Care National Audit And Research Centre reported 4.5 cases per 100 000 ICU admissions, or six cases per year with an admission diagnosis of venous air embolus, arterial air embolus, or both.³ The majority of cases of venous air embolism (VAE) are iatrogenic, classically as a result of neurosurgical intervention in the sitting position, however it is now known to be associated with a variety of procedures including central venous catheterisation, extracorporeal circuits such as haemodialysis, and high-pressure mechanical ventilation.⁴ Most venous air emboli are tiny and do not result in major clinical sequelae or require treatment, with fatal doses of rapidly injected air reported to be 100–300 ml.⁴ However, small gas emboli, as in this case, do carry serious risks, notably the complication of cerebral gas embolism.⁵ There are several ways in which this can occur: 1) direct entry of gas into the cerebral arterial system (i.e. during angiography); 2) pulmonary barotrauma resulting in gas in the pulmonary veins subsequently entering the left heart; 3) paradoxical emboli, whereby a venous embolism enters the arterial system via an intracardiac right-to-left-shunt (e.g. patent foramen ovale) or pulmonary arteriovenous malformations; 4) by overwhelming the pulmonary capillary filter mechanism; and 5) retrograde embolism, a significant concern for this patient, as a result of peripheral venous air bubbles ascending against venous flow and entering the cerebral venous system instead of the pulmonary circulation.⁶

Bubble volume seems to play a major role in the development of a retrograde embolism. Very small bubbles (microbubbles) are usually compensated for.^{7,8} Whilst it is possible that inadvertent air entry into the bloodstream is more common than reported, we hypothesise that the larger the bubble, the greater its buoyancy and potential for retrograde translocation into the cerebral venous system and subsequent infarction.^{2,5} To prevent neurological complications, urgent elimination of the bubble is needed. HBOT leads to a reduction

in volume of the bubble, aids removal of nitrogen, and improves oxygenation of potentially hypoxic tissue.¹⁰ Of 441 (78%) patients with arterial gas embolism, 346 recovered fully when treated with HBOT, with only 20 (4%) dying, compared with 74 of 288 (26%) patients receiving no recompression therapy fully recovering and 52% dying¹¹; reports demonstrated its efficacy even when treatment is delayed up to 21 hours.^{10,12–14} While one can assume that arterial air embolism has a quicker onset and is more severe, VAE can still lead to significant neurological compromise and death.^{9,15}

The risks or practicalities of transferring a patient to a specialist HBOT centre with suspected air embolism need to be considered against the potential treatment or prophylactic benefits of HBOT, especially in acutely unwell individuals. The specific risks of the hyperbaric environment on the patient and clinician also need to be considered. The management of each air embolism therefore needs to be individualised according to clinical context, urgency, and location. We propose that HBOT is an alternative treatment of air embolism, especially in the event where interventional radiology/aspiration has failed or is not available, or for treatment of bubbles too small to be seen.

Although the portal of air entry is clear in this case, VAE remains an under-recognised entity that, if untreated, can result in irreversible neurological deficit and even death. It is imperative that VAE is diagnosed and treated urgently, with HBOT providing a seemingly effective treatment option.

Authors' contributions

Concept, clinical background, treatment details, and images: A.B.

Discussion and literature review, revised drafts: P.B., J.P.

Initial draft: J.P.

Declaration of interest

The authors declare that they have no conflicts of interest.

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