

## CASE REPORT

# Cerebral air embolism from a Central Venous Catheter: A timely reminder of the importance of rapid diagnosis

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## SUMMARY

Cerebral air embolism (CAE) is a rare, avoidable and potentially fatal iatrogenic complication. Here, we report a case of CAE associated with a central venous catheter in the internal jugular vein that resulted in neurological deficits and generalised epileptic seizures. A 64-year-old man admitted for fasciotomy for compartment syndrome developed CAE with left-sided neurological deficits. The suspected origin was retrograde air flow from the right internal jugular venous catheter. The air spontaneously resorbed without the need for specific therapy, and he made a good recovery. CAE is an infrequent iatrogenic complication that requires prompt diagnosis to avoid significant morbidity and mortality. This case serves as a timely reminder that adverse outcome such as stroke, seizures or death can be avoided by a high index of suspicion and prompt diagnosis. Hyperbaric oxygen is the prime therapeutic measure, but high-quality evidence on its clinical value is lacking.

## BACKGROUND

Cerebral air embolism (CAE) is a preventable iatrogenic complication. Aetiologies include diagnostic and therapeutic interventions such as manipulation of central venous catheters (CVC), bronchoscopy, endoscopy, open heart surgery, thoracotomy and neurosurgical procedures. Non-iatrogenic causes have also been reported including deep sea diving, high altitude accidents, trauma, childbirth and oral-genital sex.<sup>1-3</sup> Here we present a case of a 64-year-old man who developed left-sided sensorimotor defects and status epilepticus due to an air embolism originating from right-sided internal jugular venous catheterisation. This case highlights the importance of prompt diagnosis to avoid adverse and potentially fatal sequelae.

## CASE PRESENTATION

A 64-year-old man with a medical history of chronic obstructive pulmonary disease underwent fasciotomy for compartment syndrome arising from acute limb ischaemia. He had an uneventful recovery and was transferred to the general telemetry floor in stable condition. A CVC was placed in the right internal jugular

vein in the intensive care unit for medicine and fluid administration.

Next day, the patient developed severe, left-sided leg pain that required repeat exploratory fasciotomy resulting in evacuation of a large haematoma. The patient required fluid resuscitation with several bags of normal saline. During fluid resuscitation, he was sitting in a semirecumbent position. Several minutes later, a nurse walked in the room and noticed that he was unresponsive with his hands and feet curled up. His body was noted to have increased tone and bent towards the left side with left-sided tonic-clonic convulsive activity witnessed by the staff. His vital signs obtained at that time were heart rate 120 bpm, blood pressure 85/40 mm Hg, respiratory rate 30 per min and oxygen saturation 60% on ambient air. On chest auscultation, patient had no cardiac murmur and bilaterally equal breath sounds. Neurological examination revealed a lethargic patient who was disoriented and could only follow one-word commands with difficulty. Further examination revealed left-sided hemiplegia with left facial droop. Due to this sudden change in his haemodynamic and neurological status, a medical emergency team was called to stabilise the patient. The patient was treated with 100% oxygen via a non-rebreather mask with little improvement, so bedside intubation was performed.

## INVESTIGATIONS

An emergent CT scan within 30 minute of symptom onset revealed serpiginous gas attenuation over the right posterior parietal lobe indicating gas in the cerebral vessels (figures 1). CT angiography (CTA) (performed only in the arterial phase) within an hour of the first CT scan and 2 hours after symptom onset demonstrated good blood flow without any filling defects. Magnetic Resonance imaging (MRI) with diffusion-weighted imaging performed within 3 hours of symptom onset did not reveal any significant pathology. The possibility of vascular air embolism originating from a mishandled central venous line was considered. The line was checked. All unused hubs on the line were closed, the CVC and delivery devices showed no evidence of leakage. At the time of the event, the patient was



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**Figure 1** Axial view of head CT demonstrating air in the cerebral vessels.

resuscitated with fluids in a semirecumbent position. Authors suspect that it could be due to lack of priming or hubs being open or connections not tight for a shorter amount of time that could have resulted in 15–20 mL of air entry.

CTA of the chest remained negative for pulmonary embolism and showed no air on the right side of heart. Transoesophageal echocardiography (TOE) with agitated normal saline did not reveal right-sided opacification or microbubbles in several cardiac cycles, ruling out intracardiac (right to left atrial shunts) and intrapulmonary shunts, respectively.

### TREATMENT

Over the next few hours, the patient continued to have tonic-clonic jerks on the left side. Electroencephalogram was performed that confirmed the presence of epileptiform discharges. The patient required intravenous levetiracetam. The CVC was removed and alternative access was established. Due to resolution of air on CTA and unavailability of hyperbaric oxygen therapy, he was treated conservatively with 100% oxygen.

### OUTCOME AND FOLLOW-UP

The next day, he was given an awakening trial and did not have neurological deficits on neurological examination.

He underwent successful extubation. The rest of the clinical course was uneventful and, by discharge, he had made a complete recovery.

### DISCUSSION

CAE is a rare, preventable, but serious iatrogenic complication resulting in significant morbidity and mortality.<sup>4</sup> CAE is widely under-reported. CAE should be considered in patients with sudden neurological deterioration, particularly after central venous or arterial manipulations, neurological procedures, or interventional procedures such as bronchoscopy, endoscopy and haemodialysis when the patient is in the sitting, semi-sitting, or standing posture.<sup>1–3</sup> The most common neurological symptoms reported are focal neurological deficits, coma, seizures, encephalopathy and headache. Neurological manifestations mainly arise from damage occurring in the right brain hemisphere owing to the more central position of the right carotid artery and right internal jugular vein, as was the case with our patient, who developed left-sided sensorimotor deficits.

CAE can be arterial, venous or both. It is difficult to differentiate arterial from venous embolism as air entry can occur concomitantly. Air embolism can take different pathways depending on patient position during CVC insertion or manipulation. Cerebral air entry can take place in a retrograde fashion in the venous circulation when the patient is in an upright position, which we suspect in our case.<sup>5</sup> More commonly, air can travel to the heart while the patient is in the supine position and pass into the ventricle and lungs, causing dyspnoea, cough, chest tightness and arrhythmias. It can also migrate to the arterial side from the venous side via intracardiac shunts (eg, patent foramen ovale) or intrapulmonary shunts (eg, arteriovenous malformations, intrapulmonary physiological shunts).<sup>6</sup> Physiologically, pulmonary capillaries can filter out air bubbles, thereby protecting the venous and arterial circulations. However, if the air bubble exceeds the filtration capacity threshold of the pulmonary capillaries, pulmonary oedema or arterial air emboli can occur as mentioned above.<sup>7,8</sup> History, physical examination, imaging including CT/CTA/MR angiography/MR venography, CTA chest, transthoracic echocardiography, TOE and transcranial Doppler can provide helpful cues to the aetiology and for differentiating arterial from venous embolism.

Early diagnosis and urgent intervention are crucial and may reduce morbidity and mortality from CAE.<sup>4</sup> Current treatment includes immediate stopping of the embolic procedure, placing the patient in the supine position in cases of retrograde ascension of air (rather than in a Trendelenburg position to avoid any potential worsening of cerebral oedema), aspirating air through a CVC, external cardiac massage and administering 100% supplementary oxygen to decrease nitrogen in the alveoli.<sup>6,9</sup>

Hyperbaric oxygenation (HBO) should be considered in the presence of neurological deficits and is probably the only management approach that is proven effective for both venous and arterial air embolism.<sup>10</sup> HBO works by increasing the partial pressures of dissolved oxygen in the plasma, leading to increase oxygenation in the ischaemic tissue and favouring denitrogenation.<sup>10</sup> The optimal time frame for initiating HBO is uncertain. The evidence suggests early initiation is preferred and improves outcomes.<sup>10</sup> However, a delayed diagnosis should not discourage treatment with hyperbaric oxygen, and there is some evidence of recovery of

neurological function in patients treated late with HBO.<sup>11 12</sup> In our case, the patient developed sudden neurological deficits and was found to have air on CT scan that resolved within an hour on repeat CT with 100% oxygen. Lack of availability of HBO in our tertiary care centre, full resorption of air on repeat CT scan and the critical condition of our patient prevented transfer to an HBO facility.

In our case, no mishandling was reported by the staff during CVC use, but we suspect this was due to under-reporting, lack of knowledge or awareness. The patient had fluid administration prior to the event, and CAE could be attributed to the lack of priming or capping of the unused hubs. Further, his semirecumbent position made him vulnerable to air aspiration during inspiration, hence retrograde ascension of air directly to the brain. Some studies have reported rapid symptom onset; however, we noticed a delay in our case as reported by Yesilaras and colleagues.<sup>13</sup> This could be due to only a small volume of air entering at a slow rate.<sup>14</sup>

Since CAE associated with CVC is largely preventable, we emphasise the importance of careful handling of CVCs and reiterate some precautions. After catheter is inserted, adhesive tape and sutures should be applied to prevent dislodging. It is imperative to ensure all connections are tight and unused hubs are locked when the catheter is not in use. As learnt from our case, it is crucial to prime the tubing and expel air from all syringes to be connected to the catheter. During CVC removal, the same position, that is, head down or Trendelenburg, should be used. This prevents the central venous pressure (CVP) from dropping below atmospheric pressure, and the patient should be instructed to avoid talking, coughing and breathing in. If possible, the highest CVP can be achieved with the Valsalva manoeuvre, resulting in the greatest protection against air embolism. Adequate pressure should be applied to achieve haemostasis after catheter removal followed by application of an air-occlusive dressing for 24 hours. It is recommended that the patient remains supine for 30 min after central venous access removal. Institution protocols should be followed for insertion and removal, along with regular training of staff to reduce or prevent any complications.<sup>6</sup>

This iatrogenic case is an important reminder of the danger of CAE and that clinical vigilance by all staff is required to prevent this avoidable complication and unnecessary morbidity and mortality.

## Learning points

- ▶ Cerebral air embolism is an avoidable complication that can result from central venous catheterisation.
- ▶ Early diagnosis by relevant imaging is important to reduce morbidity and mortality.
- ▶ Treatment with hyperbaric oxygen should be considered, if available.
- ▶ Precautionary measures can significantly reduce the chances of developing air embolism.

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