Cerebral Venous Air Embolism following Peripheral Catheter Insertion: A Case Report

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Abstract

Cerebral venous air embolism (CVAE) is a rare and catastrophic event. Most of the data is related to cerebral arterial air embolism (CAAE). CVAE is usually caused by neurosurgical procedures, cardiovascular operations, and central catheter insertion. The insertion of a peripheral venous catheter carries an extremely low risk of air embolism. A 72-year-old man presented to the emergency department with a two-week history of mild hemoptysis. During the admission, he developed ischemic stroke and CVAE. There was no apparent cause for CVAE apart from the peripherally inserted venous catheter. He was managed with high oxygen concentration, but unfortunately, he passed away due to septic shock secondary to nosocomial pneumonia. This case describes a possible link between cerebral venous air embolism and peripheral venous catheter insertion. However, further reporting is needed to estimate the incidence and the risk.

Keywords: Cerebral Venous Air Embolism; Cerebral Air Embolism; Hyperbaric Oxygen Therapy.

Introduction

Cerebral venous air embolism (CVAE) is a rare and potentially fatal condition that is often underrecognized and poorly understood.[1] Its exact incidence is unknown. CVAE involves air in the cerebral venous system, likely due to retrograde air migration through the valveless jugular veins, disrupting venous drainage and potentially causing venous infarction.[2,3]

The symptoms of CAVE are often nonspecific and may include headache, vomiting, altered sensorium, motor or sensory neurological deficits, and encephalopathy, among others.[1] Reported causes of CVAE include neurosurgical procedures, central venous catheter (CVC) placement, trauma, infections, and intracardiac or intrapulmonary left-to-right shunts. Air embolism resulting from peripheral intravenous catheter insertion is exceedingly rare.[4]

The diagnosis of CVAE is typically established through brain imaging, such as computed tomography (CT) or magnetic resonance imaging (MRI). There is no definitive treatment for CVAE, and management remains largely supportive. Similar to cerebral arterial air embolism (CAAE), therapeutic measures may include placing the patient in the Trendelenburg position, administering high-concentration oxygen, intravascular volume expansion, and, in select cases, hyperbaric oxygen therapy (HBOT).[1]

Verbal consent was taken from the relatives for publication purposes. There was no conflict of interest.

Case Report

A 72-year-old right-handed man with diabetes mellitus presented to Sultan Qaboos University Hospital with a two-week history of mild hemoptysis. He was an ex-smoker with a 55 pack-year history and reported unintentional weight loss of 5 kg. He denied fever, night sweats, appetite loss, connective tissue disease symptoms, sensory deficits, incontinence, or prior tuberculosis exposure.

On presentation to the emergency department, the patient was alert and oriented. Vital signs were normal except for elevated blood pressure (164/93 mmHg). He had mild digital clubbing. No lymphadenopathy was noted. Chest exam revealed fine basal crepitations on the right. Cardiovascular and ophthalmic exams were unremarkable. Neurologically, there were no cranial nerve deficits, and motor strength, tone, sensation, coordination, and gait were all normal.

Laboratory findings are presented in Table 1. The results demonstrated normal hemoglobin at 13.8 g/dL (11–14.5 g/dL), platelet count of 329 $\times 10^9$/L (150–450 $\times 10^9$/L), coagulation profile, estimated glomerular filtration rate (eGFR) >90 mL/min/1.73 m^2. Mild leukocytosis was noted, with a white blood cell (WBC) count of 10.7 $\times 10^9$/L (2.4–9.5 $\times 10^9$/L), and a mildly raised C-reactive protein (CRP) of 7 mg/L (0–5 mg/L). The electrocardiogram showed a normal sinus rhythm. The chest x-ray did not reveal any abnormalities.

Table 1: Summary of laboratory test results.

Test	**Result**	**Normal range**
Hb (g/L)	13.8	11-14.5
Haematocrit (L/L)	0.41	0.34-0.43
Platelet count (10^9 /L)	329	150-450
White blood count (10^9 /L)	10.7	2.4-9.5
PT (sec)	10.4	9.2-12
APTT (sec)	26.9	25-36.4
Fibrinogen (g/l)	4.3	1.7-3.6
D-dimer (mg.L FEU)	0.5	0.2-0.7
CRP (mg/L)	7	0-5
eGFR (ml/min/1.73 m^2)	> 90	> 90
CK (U/L)	144	39-308

Hb - hemoglobin, PT - prothrombin time, APTT – activated partial thromboplastin time, CRP – C reactive protein, eGFR – estimated glomerular filtration time, CK – creatinine kinase.

A CT scan of the chest, performed as part of the workup for hemoptysis and to exclude secondary malignancy, revealed features consistent with usual interstitial pneumonia (UIP) and a small 8 mm nodule in the left upper lobe. A CT scan of the abdomen and pelvis showed no suspicious masses or abnormal lymphadenopathy.

The patient was scheduled for bronchoscopy for hemoptysis evaluation but developed sudden left-sided hemiplegia on day three of admission. He remained conscious with stable vitals and no cranial nerve palsies, aside from mild flattening of the left nasolabial fold. Motor exam showed weakness in the left upper (Medical Research Council [MRC] grade 4) and lower limbs (MRC grade 3), without sensory loss.

An urgent CT scan of the head with cerebral angiography was normal. He was diagnosed with a right-sided ischemic stroke within the thrombolysis time window. However, thrombolysis was deferred due to ongoing hemoptysis, and he was managed with aspirin, clopidogrel, and atorvastatin.

Eight hours later, the patient became unconscious, with a Glasgow Coma Scale (GCS) score of 3. His pupils were pinpoint and non-reactive. He remained hemodynamically stable, with an oxygen saturation of 99% on room air. He had an uneventful peripheral intravenous catheter insertion in his left hand two hours before clinical deterioration.

A repeat CT scan of the head revealed interval development of pneumocephalus, characterized by serpiginous air densities throughout the brain, predominantly along the sulci and gyri of the right cerebral hemisphere. Air lucencies were also noted within the superior sagittal sinus and left sigmoid sinus, findings consistent with CVAE. Additionally, there was new hypodensity in the periventricular region of the right parietal lobe, suggestive of acute infarction. There was no evidence of skull fracture [Figure 1A–E].

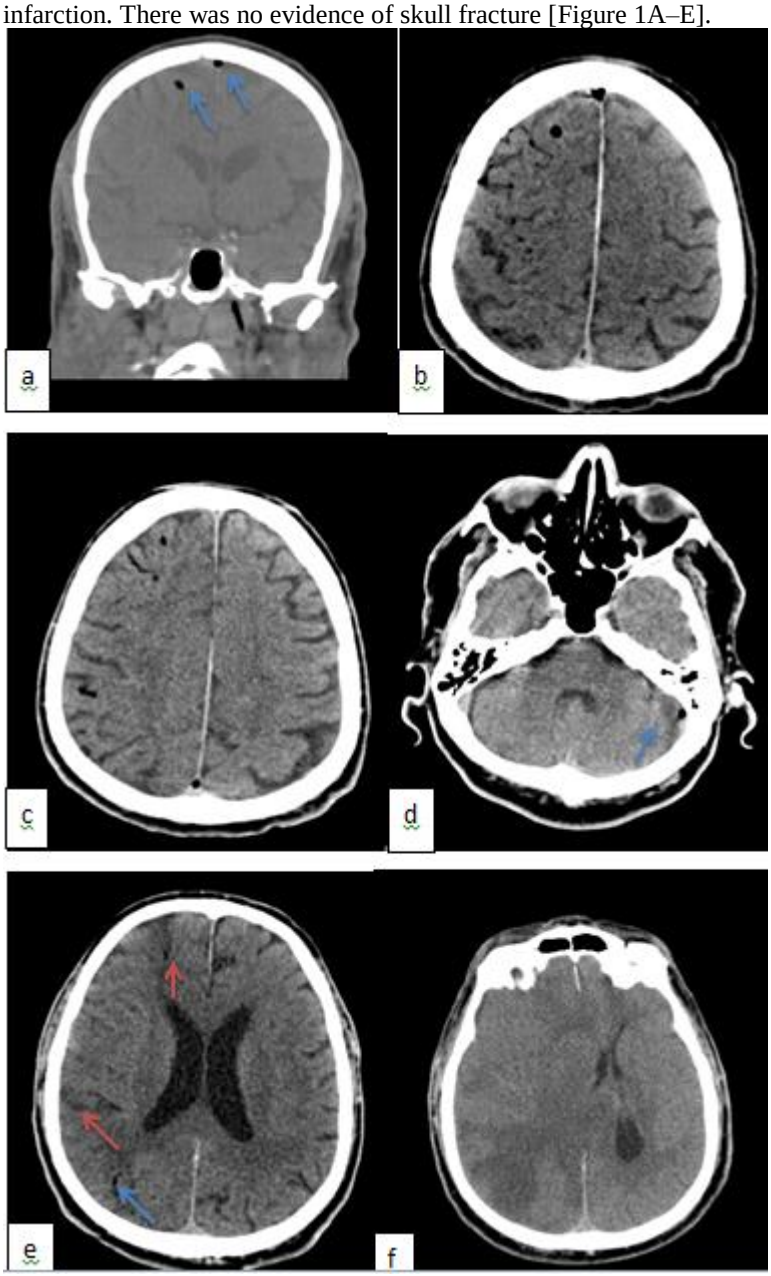

Figure 1: Non-contrast CT of the head after deterioration in consciousness A) Coronal reformation in soft tissue windows show better resolution of air in the superior sagittal sinus and right cerebral hemisphere B)& C) Axial section demonstrating diffuse pneumocephalus along with the areas of lucencies in the superior sagittal sinus and left sigmoid sinus in keeping with cerebral venous air embolism D) Axial image of posterior fossa showing air speck in left transverse sinus E) Axial section demonstrating hypo-density in periventricular area of right parietal lobe [blue arrow] and pneumocephalus [red arrow]. F) Axial section demonstrating brain edema and midline shift.

The patient was intubated and transferred to the intensive care unit (ICU). He was placed in the Trendelenburg position and ventilated with a fraction of inspired oxygen (FiO_2) of 100%. Based on neuroimaging findings, a CVAE was established, although no obvious source of the air embolism was identified at that time.

A diagnostic workup for air embolism was initiated, and the patient was referred for HBOT. Imaging studies showed no evidence of pulmonary arteriovenous malformations (AVM), pneumothorax, pneumomediastinum, or subcutaneous emphysema. A transthoracic echocardiogram with bubble study revealed no intracardiac thrombus or septal defects.

The patient underwent bronchoscopy, which revealed no endobronchial lesions or identifiable source of bleeding. The tuberculosis (TB) GeneXpert test was negative. Connective tissue disease workup showed an antinuclear antibody (ANA) titer of 1:160 and weakly positive antineutrophil cytoplasmic antibodies (ANCA). However, both proteinase 3 (PR3) and myeloperoxidase (MPO) antibodies were negative.

A repeat CT scan of the brain on day 4 of admission revealed vasogenic edema involving the right cerebral hemisphere, accompanied by mass effect and midline shift [Figure 1F]. Notably, no air lucencies were observed on the follow-up scan. The patient subsequently underwent urgent decompressive craniotomy.

Unfortunately, his clinical condition deteriorated on day 5, and he died from septic shock, secondary to Extended-Spectrum Beta-Lactamase (ESBL) *Escherichia coli* pneumonia.

Discussion

Cerebral air embolism is a rare but serious condition caused by air entering the vascular system, affecting either arteries or veins.[1] It can occur via paradoxical embolism through intracardiac shunts like a patent foramen ovale (PFO) or atrial septal defect, allowing air to reach cerebral arteries. Retrograde venous embolism may also happen when air rises against blood flow, especially in upright patients.[2]

Air can also enter the arterial system through pulmonary AVMs or inadequate filtration by pulmonary capillaries, reaching the brain. The exact incidence of cerebral air embolism is unknown.[2,3] Its severity depends on factors like patient position, gas solubility, and the volume and rate of air entry.[5] Symptoms vary from headache to coma or death. Air in cerebral vessels may trigger vasoconstriction and platelet aggregation, leading to stroke.[3] Mortality risk depends on the volume and speed of air entry and the affected vascular territory.[6]

Trauma is the leading cause of cerebral air embolism, responsible for about 75% of cases.[7] Infections like chronic otitis media and mastoiditis account for 9% of pneumocephalus cases.[7] Iatrogenic causes include neurosurgery, endoscopy, cardiovascular procedures, and central line manipulation.[8]

In our case, CVAE was diagnosed based on the presence of air in the superior sagittal sinus and a decline in GCS. Trauma was excluded due to no history or imaging evidence of fractures. There was no recent surgery, endoscopy, tube insertion, or CVC use. Bronchoscopy occurred after the diagnosis, and infections such as otitis media and mastoiditis were ruled out.

CVAE can occur via a right-to-left shunt, allowing air to bypass the lungs and enter the arterial system (paradoxical embolism). In this case, a PFO was excluded with a negative bubble study, and a bronchopleural fistula was unlikely due to the absence of pneumothorax, pneumomediastinum, or subcutaneous emphysema.

Although pulmonary AVM was considered due to hemoptysis, CT pulmonary angiography (CTPA) showed no evidence of AVM. The only potential cause of CVAE in this case was peripheral intravenous catheter insertion two hours before deterioration, a rare but recognized risk factor. Few cases link CVAE to peripheral access, likely due to retrograde air migration, though the exact mechanism remains unclear.[1,4,9]

Management of CVAE is mainly supportive. The left lateral Trendelenburg position (Durant's maneuver) is often used to redirect air to central veins,[2] though it hasn't shown hemodynamic benefit in animal studies.[10]

Administering 100% FiO_2 reduces air bubble volume. In severe cases, HBOT can compress bubbles, improve gas exchange, and lower intracranial pressure. However, its mortality benefit is proven only in cerebral arterial air embolism.[1,3]

Early HBOT within six hours significantly improves neurological outcomes in gas embolism.[11] Time to first HBOT is the strongest predictor of recovery, while initial presentation with hemiplegia indicates poor prognosis.[12] In our case, the patient did not require HBOT, as the air embolism resolved with high-concentration oxygen therapy alone.

A systematic review on cerebral gas embolism from CVCs reported a 21.7% mortality rate, with a higher risk in older patients, those with coma, or early cardiac arrest.[13] Prompt and appropriate treatment can greatly reduce CVAE-related morbidity and mortality.[14] In our case, the patient's death was attributed to septic shock rather than CVAE.

To reduce the risk of CVAE, CVC insertion and removal should follow key precautions. Elevating central venous pressure (CVP) via Trendelenburg positioning and adequate hydration helps prevent air entry. Similarly, CVS should be removed with the patient supine or in Trendelenburg to minimize venous air aspiration.[15] Unfortunately, there are no established guidelines for preventing this complication during peripheral venous cannula insertion, as occurred in our case.

Air embolism, though rare, can be life-threatening during invasive procedures. Clinicians should remain vigilant, ensure early recognition, and initiate prompt, targeted management. Early consideration of advanced therapies like HBOT is crucial for optimal outcomes.[15]

Conclusion

This case demonstrates a rare occurrence of an extensive CVAE in a 72-year-old male who presented with hemoptysis. Thorough investigations ruled out all possible common causes of CVAE. The only considered explanation for CVAE is the placement of a peripheral venous catheter. However, further reporting is needed to estimate the incidence and risk of CVAE after peripheral venous catheter insertion.

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