CASE REPORT

# A patient with massive cerebral arterial air embolism

**Chelsia Chan Bao Tyng, MRCP, Loo Lay Khoon, MRCP, Anita Bhajan Manocha, MIntMed**

Department of Medicine, Seberang Jaya Hospital, Pulau Pinang, Malaysia

**SUMMARY**

**Cerebral air embolism is potentially a catastrophic event that occurs as a consequence of air entry into the vasculature. We report a mechanically ventilated 72-year-old woman who underwent multiple procedures during intensive care stay with few possible sources of emboli postulated. We also discuss regarding the preventive measures to minimise the risk of air embolism.**

## BACKGROUND

Cerebral air embolism is an uncommon event that occurs because of the entry of air into the vasculature. It is estimated that 300 to 500mL of gas introduced at a rate of 100mL/sec can acutely be fatal to humans.[1] In this report we describe of a rare occurrence, the possible aetiology of which might have contributed to a fatal case of cerebral air embolism.

## CASE PRESENTATION

A 72-year-old lady with underlying chronic renal disease due to diabetes and hypertension presented with shortness of breath, lethargy and reduced oral intake for three days. On physical examination, she was pale and tachypnoeic with bibasal lung crepitations. She was intubated at the emergency department for impending respiratory failure secondary to fluid overload and severe metabolic acidosis. She was then admitted to Intensive Care Unit and started on broad spectrum antibiotic for possible sepsis. Acute haemodialysis was initiated on the same day for severe metabolic acidosis, oliguria and uraemia, via a right femoral catheter. She had a triple lumen central venous catheter inserted in the left femoral vein and an arterial line in the right radial artery. All procedures were done in the supine position. On day two of admission, she was noted to have unequal pupils with right and left pupil measuring 5mm and 4mm respectively. CT brain was not done at that time due to haemodynamic instability. She underwent an uneventful second session of haemodialysis the following day. Despite cessation of sedation for more than 12 hours, her GCS remained E1VtM1. CT brain done on day-4 of admission showed massive cerebral arterial air embolism (Figure 1) with no involvement of the venous system. In an attempt to look for the source of air embolism, a CT angiogram of aorta, neck, abdomen and lower limb was done (Figure 2). Splenic and bilateral renal infarctions were reported. Transthoracic echocardiogram revealed ejection fraction of 75% with no evidence of patent foramen ovale. Electrocardiography showed sinus tachycardia. Thus, she was given FiO2 of 1.0 and remained on the supine position. However, she succumbed shortly after. The cause of death was acute ischaemic stroke secondary to massive cerebral arterial air embolism.

## DISCUSSION

The more common iatrogenic causes of cerebral air embolism are major interventions such as neurosurgery and cardiovascular operations, endoscopy, haemodialysis and minor interventions for peripheral and central venous access. Our patient underwent endotracheal intubation and positive pressure ventilation, arterial line and central line insertion, and haemodialysis, any of which could have caused the cerebral air embolism.

Few postulations arise about the source of air embolism. If small amounts of air were inadvertently infused through the femoral catheter and central line (in the femoral veins), it can be removed from the pulmonary vascular bed by gas diffusion across the arteriolar wall and into the alveolar spaces. However, when the capacity of the lung to remove gas is exceed ≥50mL, pulmonary outflow tract obstruction with or without concomitant arterial embolisation can occur.[2]

Iatrogenic cerebral air embolism related to central venous catheter insertion, manipulation or removal has been widely reported. The median time to the onset of symptom reported by the review being as fast as one minute.

The other possible source of air emboli for our patient was haemodialysis. Cerebral air embolism with a demonstrable air column in the haemodialysis circuit has been reported. Use of a haemodialysis catheter and declotting of haemodialysis access are the other possible causes.[3] Microbubbles in haemodialysis circuits may originate in arterial lock connector at negative pressure or from remnant bubbles due to insufficient priming. These may not be picked up as air by the dialysis circuit. Blood flow rate and negative arterial pressure correlate with the microbubble rate as per a recent observational study.[4] The microbubbles can be lodged in the capillaries and cause tissue ischemia, inflammatory response, and complement activation. Obstruction of microcirculation and tissue damage can also occur due to platelet aggregation and clot formation.

In our patient transthoracic echocardiogram was normal, therefore ruling out patent foramen ovale which provides a right-to-left shunt for venous air to embolise to the cerebral arteries. CT angiogram did not reveal pulmonary arteriovenous malformation, pulmonary embolism or any

*This article was accepted: 4 April 2019*
*Corresponding Author: Dr. Chelsia Chan Bao Tyng*
*Email: chelsia_cbt@hotmail.com*

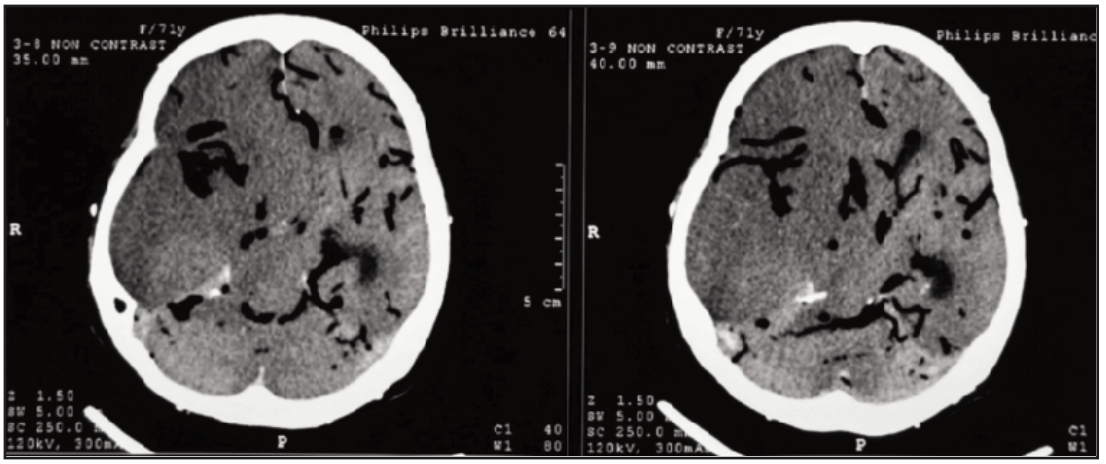


**Fig. 1:** CT brain showing large right middle cerebral artery territory infarct with mass effect and midline shift with generalised cerebral edema predominantly on the right side. Air embolism involving cerebral arteries with pneumoventricles and pneumocephalus were noted.

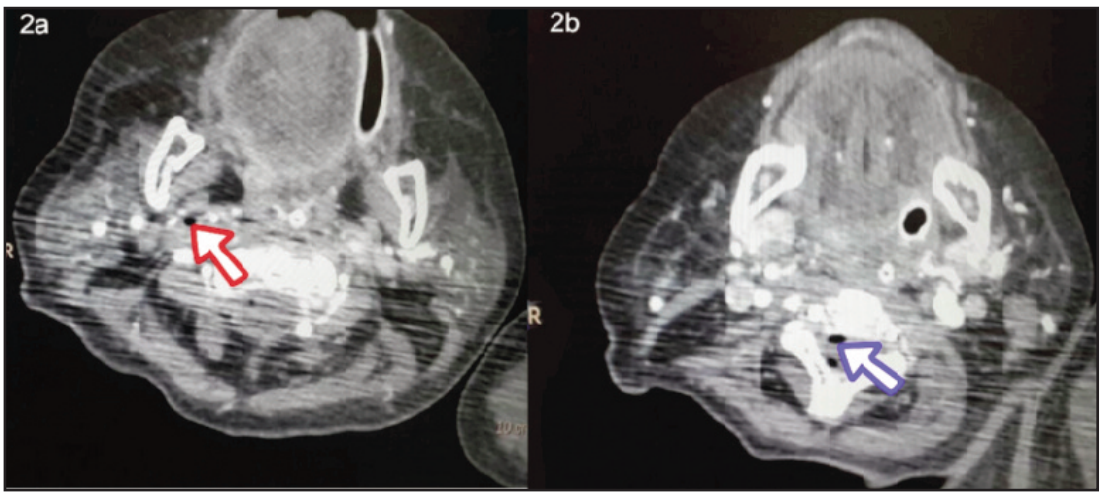


**Fig. 2:** Computed tomography (CT) angiogram of the level of neck showing air foci in the right common carotid artery (arrow in 2a) and epidural CSF spaces in the cervical cord level (arrow in 2b).

air foci in the mediastinal great vessels, heart chambers, thoracic and abdominal aorta, inferior vena cava and visualised vessels in the groin. Air embolism can theoretically be a complication of non-invasive positive pressure ventilation (NPPV). In this case, the portal of entry for air into the vasculature remains speculative.

When air embolism is suspected, simultaneous with repositioning, the airway and breathing should be stabilised with high flow oxygen and/or mechanical ventilation, when necessary. Intravenous access should be secured so that patients can be treated with intravenous fluids and/or vasopressors to restore adequate tissue perfusion. High-flow supplemental oxygen increases the rate with which the embolised air resorbs. A patient with arterial air embolism should be placed in the supine position.

Accurate reports of the prognosis of patients with air embolism are confined to case series that have selected patients for therapy with hyperbaric oxygen (HBO). While in the past mortality rates as high as 90 percent were reported, case series since then suggest rates that range from 12 to 30 percent.[5]

Preventive measures should be undertaken to reduce the risk of air embolism. This includes keeping all connections to a central line closed and locked when not in use, withdrawing blood and injecting medications with the patient supine (i.e., below the level of the heart), and asking the patient to perform Valsalva manoeuvre or exhale during removal. For patients who are mechanically ventilated, airway pressures should be minimised to prevent pulmonary barotrauma. For patients with central intravascular catheters, Trendelenburg position is preferred for central venous catheter insertion and removal from jugular and subclavian vein sites; the supine position is sufficient for the femoral site.

## CONCLUSION

Cerebral air embolism should be suspected in any patient with sudden neurological deterioration with risk factors especially in intensive care setting as it transfers high mortality. Preventive measures should be undertaken to minimise the risk of cerebral air embolism.

## REFERENCES


1. King MB, Harmon KR. Unusual forms of pulmonary embolism. Clin Chest Med 1994; 15(3): 561-80.
2. Palmon SC, Moore LE, Lundberg J, Toung T. Venous air embolism: a review. J Clin Anesth 1997; 9(3): 251-57.
3. Yu AS, Levy E. Paradoxical cerebral air embolism from a hemodialysis catheter. Am J Kidney Dis 1997; 29(3): 453-5.
4. Wagner S, Rode C, Wojke R, Canaud B. Observation of microbubbles during standard dialysis treatments. Clin Kidney J 2015; 8(4): 400-4.
5. Bessereau J, Genotelle N, Chabbaut C, Huon A, Tabah A, Aboab J et al. Long-term outcome of iatrogenic gas embolism. Intensive Care Med 2010; 36(7): 1180-7.