Case Report

Mind the bubbles - A patient presents with seizure after haemodialysis

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Abstract

A patient with patent ductus arteriosus presents with seizure after haemodialysis. Although conscious on arrival to the emergency department with spontaneous limb movement, he develops recurrent convulsion and left hemiparesis after admission. The approach to the haemodialysis patient presenting with seizure is discussed and the role of early hyperbaric oxygen therapy for an uncommon but important diagnosis is highlighted.

Keywords

Air embolism, Hyperbaric Oxygen Therapy, Haemodialysis, Seizure

Key points

- Cerebral arterial gas embolism is an uncommon but preventable cause of seizure after haemodialysis.
- This differential should be considered in patients with suggestive history.
- Presence of pneumocephaly on computed tomography is diagnostic.
- Early definitive treatment with hyperbaric oxygen therapy can improve patient outcomes.

Case presentation

A patient with history of patent ductus arteriosus presented with his first episode of generalized-tonic-clonic convulsion after haemodialysis. There was no fever or head injury. The seizure aborted upon intravenous administration of midazolam. At the resuscitation room, he was afebrile, with blood pressure 129/91mmHg, heart rate 140 beats per minute, respiratory rate 20 breaths per minute, oxygen saturation 100% on room air and Glasgow Coma Scale E4V4M6. His neck was soft and pupils equal in size and reactive to light. Spontaneous movement of four limbs was noted but power on his left side was diminished. Point-of-care blood glucose was 6.3 mmol/L. Electrocardiogram showed sinus rhythm with normal intervals and chest X-Ray was unremarkable. Computed tomography (CT) scan of the brain was performed (Figure 1).

Questions

1. Describe the findings of the CT brain performed on admission (Figures 1a-d).

2. What is the diagnosis?

3. What is the definitive management for his condition?

Answers

1. Scattered air pockets in the right posterior subarachnoid space. Reduced grey-white differentiation at the right occipital region. Intraparenchymal gas bubble at right frontal lobe. Gas in the central and post central sulci of the right frontal lobe.
2. Cerebral arterial gas embolism (CAGE).

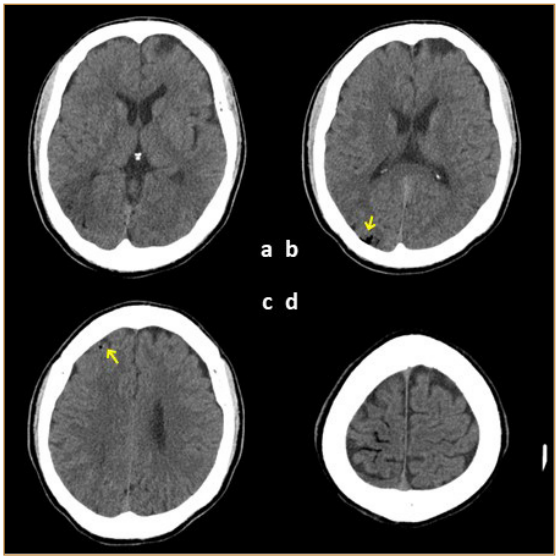

Figure 1.

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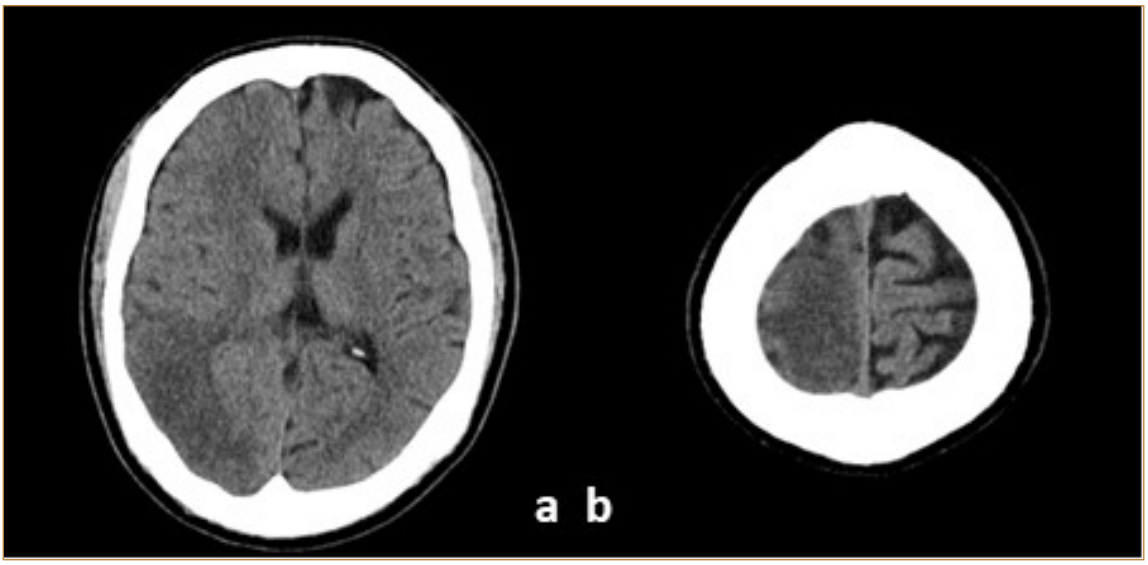

Figure 2.

3. Hyperbaric oxygen therapy (HBOT) is the definitive treatment for symptomatic cerebral arterial gas embolism (Type 1 recommendation, Level C evidence).[1] Where HBOT is not available, 100% oxygen should be administered immediately while arrangements are made for transferral.

Clinical progress

The patient was transferred to another hospital for hyperbaric oxygen therapy where he developed recurrent convulsions with fluctuating conscious level requiring intubation, phenytoin and propofol infusion. CT brain repeated 1 day after admission (Figure 2a, 2b) demonstrated hypodense infarct over right temporal-parietal-occipital area with compression of the occipital horn of the lateral ventricle. There was diffuse right cerebral oedema involving high parietal and frontal lobes with sulcal effacement. Pneumocephaly was no longer obvious. CT thorax showed no air density within pulmonary vasculature, cardiac chambers, the included aorta and branches from the aortic arch. He was extubated on day 3 and discharged to the stroke ward on day 6, fully conscious, but remained densely hemiplegic with hemineglect and hemisensory loss (National Institute of Health Stroke Scale of 15).

Discussion

The approach to the emergency management of the haemodialysis patient presenting with first episode of seizure is similar to that of the general population. Differential diagnoses to consider given the history of end stage renal failure and haemodialysis include: uraemic encephalopathy, dialysis disequilibrium syndrome, intradialytic haemodynamic instability and air embolism. These patients are also more susceptible to cerebrovascular accidents, electrolyte and metabolic disturbances, and drug- or toxin-induced seizures as a result of decreased clearance.[2]

Uraemic encephalopathy should be considered in patients with untreated severe uraemia, and is therefore likely to occur before haemodialysis. Dialysis disequilibrium syndrome should be considered in patients undergoing haemodialysis for the first time. Seizures related to air embolism may occur during placement or removal of the haemodialysis catheter, as a result of accidental disconnection or breakage of the arterial tubing or during administration of anticoagulant or saline.[3]

Immediate management should include stopping the dialysis, placing the patient in the recovery position, protecting the airway, administration of oxygen and IV fluids as indicated. IV benzodiazepine is the first-line drug of choice. If venous gas embolism is suspected to be the cause of seizure, the patient should be provided with 100% oxygen. Current recommendations prefer the supine position over the head down position. Aspiration of air from the catheter may be attempted. [3]

Pathophysiology of CAGE

Air embolism is estimated to occur in 8.5-33 per million haemodialysis sessions in North America. With the incorporation of safeguards designed to prevent air embolism in the modern haemodialysis machine, most incidences occur as a result of human error.[3]

The source may be venous, such as from placement, manipulation or removal of a central venous catheter or arterial, such as from

extracorporeal circuit. Depending on the volume, rate and site of air embolism, presentation can range from asymptomatic to respiratory failure to circulatory collapse to coma.

There are two ways for air to travel to the cerebral vasculature: either (i) retrograde flow from the jugular venous system resulting in venous embolism or (ii) "paradoxical" passage from the venous to arterial system through intracardiac, pulmonary or arteriovenous shunts resulting in arterial embolism.

Proposed mechanisms of neurological injury include: (i) mechanical obstruction leading to neuronal death and cytotoxic oedema, (ii) endothelial irritation resulting in disruption of the blood-brain barrier and (iii) immune response activation. Changes in cerebral blood flow, hypoxia, involvement of the cortex by haemorrhage or infarct and development of epileptogenic changes in cortical neurons and connections subsequently result in seizures.[4]

Role of HBOT

Hyperbaric oxygen therapy (i) diminishes the volume of intravascular gas bubbles to relieve obstruction and restore circulation, (ii) facilitates bubble resorption by de-nitrogenization, (iii) increases the partial pressure of dissolved oxygen in the blood to relieve tissue ischaemia, (iv) reduces the permeability of the blood-brain barrier, and (v) reduces the adherence of leucocytes to damaged endothelium. Although patients treated within 6 hours of embolism are reported to have better outcome, clinically significant improvement has been reported in cases treated with HBOT beyond 24 hours.[5]

Conclusion

Cerebral arterial gas embolism is an uncommon but important differential diagnosis in the patient presenting with seizure after haemodialysis. Negative prognostic factors include encephalopathy or hemiparesis at presentation, presence of gyriform air, cerebral infarct or oedema on initial imaging, retrograde mechanism of cerebral air embolism and old age.[5] Prompt recognition is important as early definitive treatment with hyperbaric oxygen therapy can improve patient outcomes.

Declaration of competing interests

Nothing to declare.

Authorship

Both authors contributed equally to the writing process.

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Conflict of interest

Nothing to declare.

Ethical approval & Human rights

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