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Normal diffusion-weighted imaging in cerebral air embolism complicating angiography

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Abstract We report a case of cerebral air embolism resulting from accidental air injection during cerebral angiography. A 60-year-old man was accidentally injected with air via the left subclavian artery. Angiography demonstrated air within the basilar artery. The patient showed signs of posterior circulation ischaemia (confusion, blindness, gaze palsy and hemiparesis). However, MRI, including diffusion-weighted imaging, showed no abnormality 4 h later. The patient was treated with

hyperbaric oxygen within 5 h of the embolism. All symptoms and signs resolved completely within a week.

Key words Embolism, cerebral air · Magnetic resonance imaging, diffusion-weighted image · Hyperbaric oxygen therapy

Introduction

Cerebral air embolism occasionally occurs in diving accidents or as a complication of positive-pressure mechanical ventilation [1, 2]. Multiple brain infarcts can be seen on CT or MRI. Diffusion-weighted imaging (DWI) is particularly useful for detection of early brain lesions in stroke [3–6]. For cerebral air embolism, immediate hyperbaric oxygen therapy is recommended as specific, definitive treatment [7].

We report a case of cerebral air embolism due to accidental air injection during angiography, resulting in severe neurological manifestations. MRI, including a DWI sequence was performed, and immediate initiation of hyperbaric oxygen therapy (HBOT) was accompanied by resolution of symptoms.

Case report

A 60-year-old man underwent cerebral angiography for a recurrent cervical tumour. During angiography via the right subclavian artery, approximately 15 ml of air was accidentally injected due to a



Fig. 1 Approximately 15 ml of air was accidentally injected into the left subclavian artery. The angiogram shows air within the basilar (*arrow*) and occipital (*arrowhead*) arteries

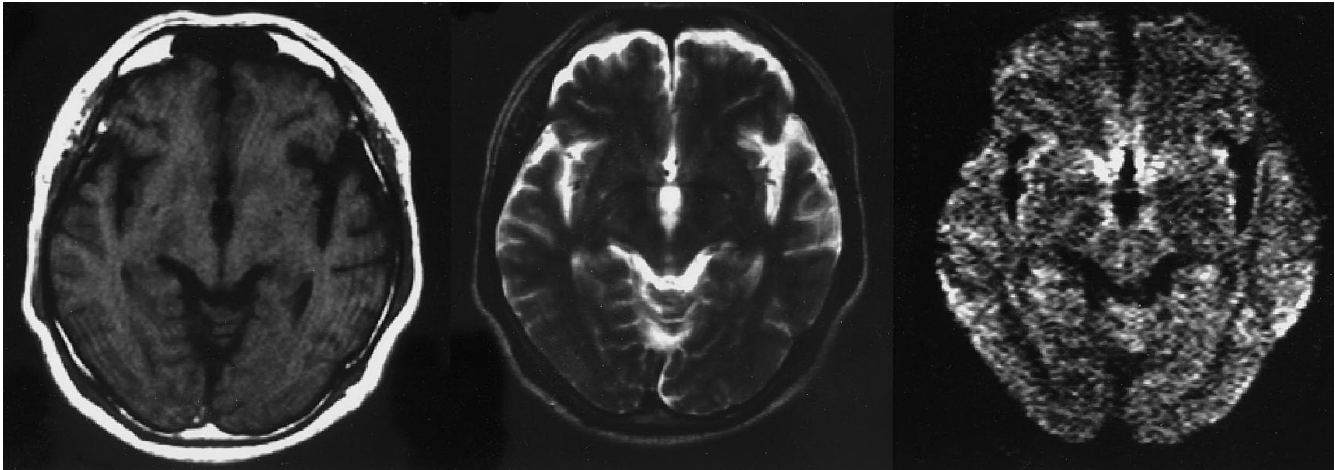
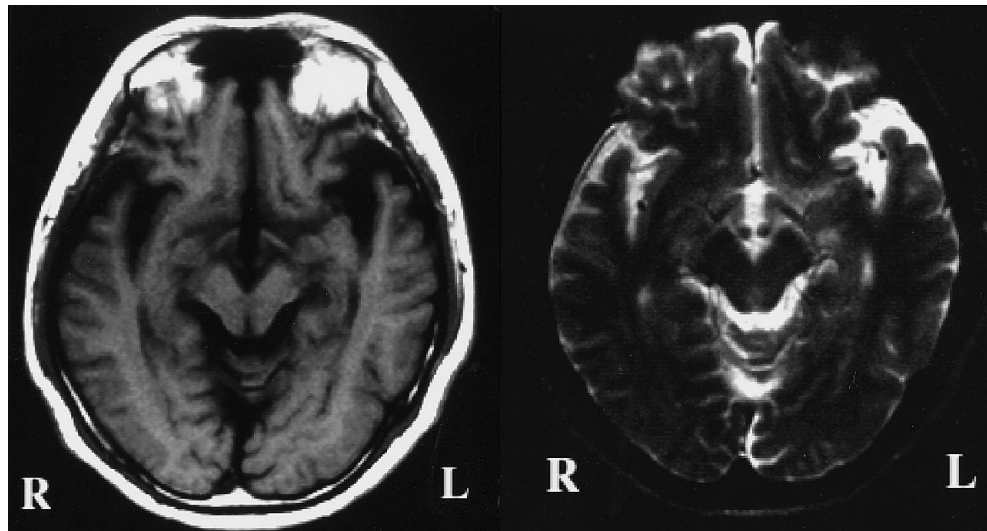


Fig. 2 MRI demonstrated no abnormal signal 2 h after the embolism: *left* T1-weighted; *centre* T2-weighted; *right* diffusion-weighted

mechanical malfunction. The angiogram showed air within the basilar artery (Fig. 1). Premedication with sedatives prior to angiography hindered assessment of the level of consciousness. Impairment of consciousness was prominent 2 h later. Examination disclosed confusion, blindness with preserved light reflexes, a disturbance of horizontal eye movements, and left hemiparesis. MRI, including DWI, showed no abnormal signal intensity. Cerebral air embolism was diagnosed and the patient was transferred for HBOT 4 h after the arterial air injection. HBOT was initiated immediately at 2.5 atm for 90 min, and the patient became alert and showed improvement of the left hemiparesis the next day. His blindness ameliorated after three further sessions of HBOT. All neurological symptoms and signs resolved completely within a week. T2-weighted MRI showed only a small focus of high signal in the right frontal white matter, not seen on previous examinations.

Fig. 3 MRI showed no abnormal signal in area supplied by the posterior circulation on (*left*) T1- or T2-weighted images



Discussion

Cerebral air embolism can cause a variety of neurological disturbances: sudden loss of consciousness, nausea, dizziness, seizures, limb weakness, paraesthesiae, visual dysfunction and headache [1]. As imaging shows brain infarcts which are not aetiologically specific [8–10], the diagnosis should be made clinically when sudden neurological deficits follow events capable of introducing air into vessels. In the present case, the complex of impaired consciousness, left hemiparesis, horizontal eye-movement disturbances and cortical blindness suggested involvement of the posterior circulation, which corresponded well to the angiographic demonstration of air in the basilar artery.

Echo-planar DWI generates images related to diffusion of water molecules [11–12]; in stroke, early cerebral ischaemia can be detected even when T2-weighted imaging shows no abnormality [13–17]. Specifically,

DWI shows an increase in the amount of intracellular water in the early phase of ischaemic stroke. Reuter et al. [10] documented cerebral changes on MRI in six of eight patients with arterial gas embolism [10]. However, while our patient showed severe neurological deficits, MRI, including DWI, showed no abnormality. As all symptoms resolved within a week, any lesion caused by the air embolism may have been too small to be demonstrated by DWI, and the amount of air – 15 ml – might be considered relatively small.

In cerebral air embolism, biochemical actions at the blood-gas interface can lead to alterations in haemostasis, endothelial damage and activation of leukocytes [18, 19]. Immediate initiation of HBOT, typically at 2.5–3.0 atm for 2–4 h, has been the treatment of choice [18]. It reduces the size of bubbles and helps to correct hypoxia. In our patient, HBOT was given within 5 h of the embolism, and repeated HBOT was accompanied by complete resolution of all deficits, without abnormal signal on MRI in the affected arterial territory.

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