

CASE REPORT

A case of cerebral and coronary air embolism after flushing of a pleural drain

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Abstract

This report describes the case of a 64-year-old woman with loss of consciousness, forced deviation of the head and eyes, and signs of myocardial infarction immediately after flushing of a pleural drain. A CT scan of the brain demonstrated extensive bifrontal air configurations and her ECG showed ST-segment elevation. She was diagnosed with cerebral and coronary air embolism. She responded favourably to two sessions of hyperbaric oxygen therapy. We conclude that arterial gas embolism can develop, even in the absence of a right-to-left shunt, if air enters directly

into the pulmonary veins.

Introduction

Vascular gas embolism is predominantly an iatrogenic medical condition.^[1] It can occur in any procedure that includes the risk of air or another gas entering the bloodstream. Procedures that carry a high risk of gas embolism include neurosurgical procedures in the sitting position, central venous line placements, cardiac surgery^[1] and other procedures where the venous circulation is exposed at a level above the heart.^[2] The

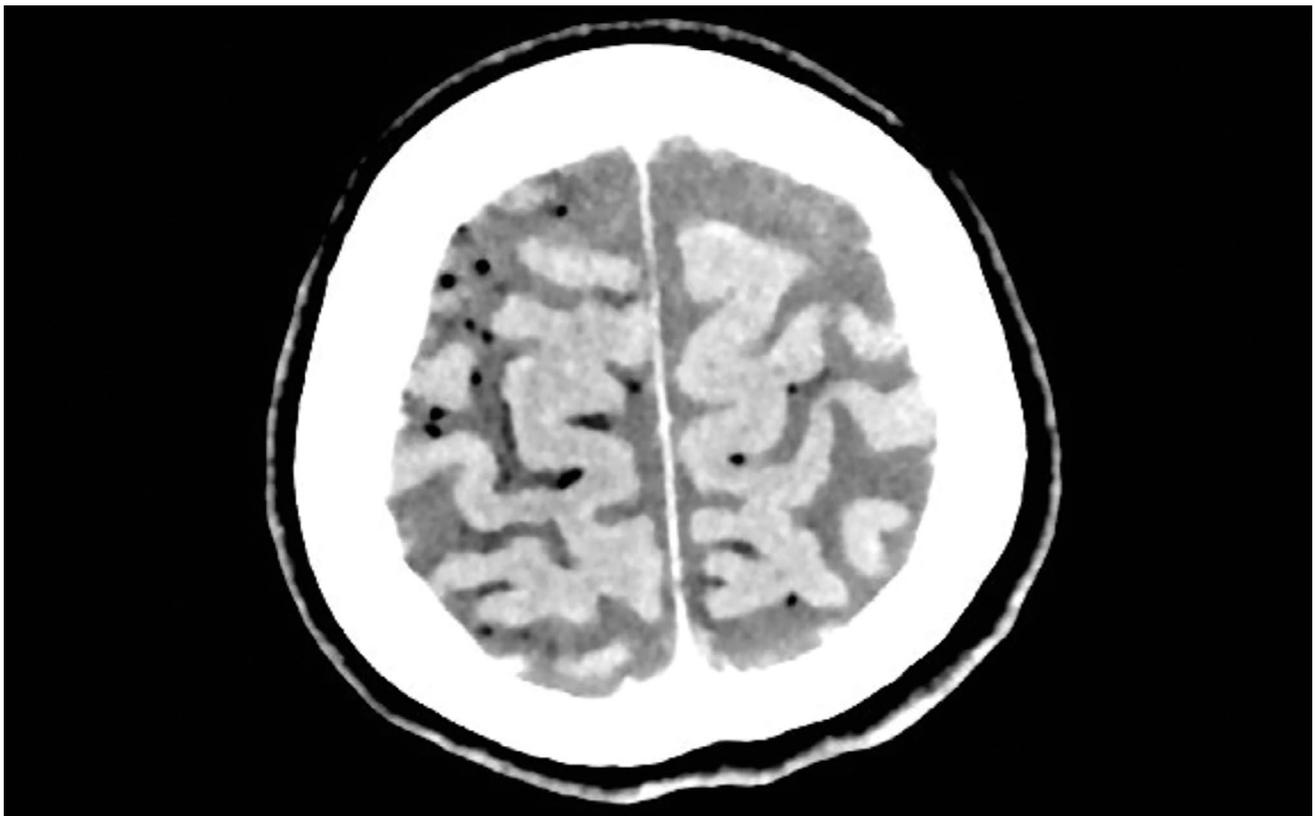


Figure 1. Air configurations in the subarachnoid space, surrounding the gyri, most probably in vascular structures



Figure 2. ST-segment elevations predominantly in leads I, II, III, aVF and V3-5

severity of symptoms is dependent on the size and location of the gas embolism.

Vascular gas embolism can occur in the venous or arterial system. Arterial gas embolism can result in neurological and myocardial damage, usually due to obstruction of the supplying vessels of these tissues. Venous gas emboli, however, are mostly filtered out by the lungs, except if they pass on to the arterial system through a right-to-left shunt, such as a patent foramen ovale which is present in 20-34% of the population.^[3] This type of embolism is referred to as paradoxical embolism.^[4] Here, we present a case of arterial gas embolism resulting in myocardial and cerebral symptoms treated with hyperbaric oxygen therapy.

Case report

A 64-year-old woman with a previous medical history of bronchial asthma was admitted to the hospital with a diagnosis of pneumonia with pleural effusion. After two weeks of in-hospital treatment that involved intravenous antibiotics and placement of a pleural drain, she was slowly recovering. On day 16 of her admission, immediately after flushing the pleural drain with saline, she reported general discomfort, followed by acute loss of consciousness with a Glasgow Coma Scale (GCS) of E1M1V1, forced deviation of her head and eyes to the right and a bilateral Babinski reflex. Notable was also a tachycardia (130 beats/min) with hypotension (80/40 mmHg).

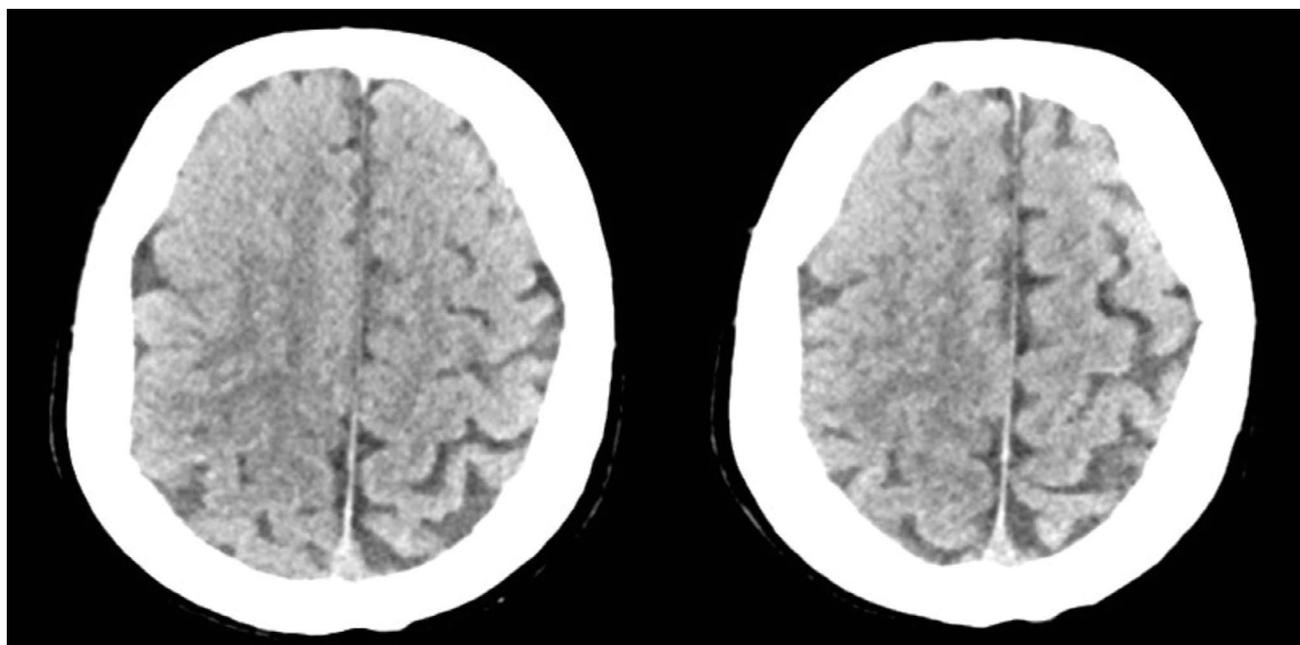


Figure 3. Right frontal lobe swelling

A CT scan of the brain showed extensive air configurations between the frontal sulci on both sides, highly suspect for air emboli in vascular structures (*figure 1*). CT scan of the thorax showed consolidation in the right lung and a correctly placed pleural drain. Her ECG showed signs of acute ischaemia in the anterolateral and inferior regions (*figure 2*), confirmed by an elevated troponin level. The patient was diagnosed with arterial gas embolism in the coronary as well as cerebral arteries due to air entry in the pulmonary veins after flushing of the pleural drain. Treatment with 100% oxygen through a non-rebreathing mask was started. After consultation with our hyperbaric department, she was urgently transported to our hospital for treatment.

After arrival in our hospital, vital parameters showed a normal blood pressure and 100% oxygen saturation on 15 litres per minute of supplemental oxygen through a non-rebreather mask. ST-elevations were still apparent on her ECG, cardiac ultrasound revealed normal cardiac function with the exception of apical hypokinesia. The neurological symptoms had improved; her GCS was now E3M5V2, her right arm was paretic. Babinski reflexes were still present.

Six and a half hours after development of her symptoms, treatment with hyperbaric oxygen therapy was started. The treatment was performed according to U.S. Navy Treatment Table 6, which lasts approximately five hours. The treatment table was completed without complications and it was noticed she moved both arms afterwards. She was transferred to the coronary care unit for haemodynamic monitoring.

The next morning, neurological examination showed slight improvement. GCS was E3M4V3 with flexion of both arms to a painful stimulus. However, she did stick out her tongue upon request. Because of this minor improvement, it was decided that the patient should undergo another session of hyperbaric oxygen therapy for two hours. At the end of this second session, she developed a self-limiting tonic-clonic seizure lasting one minute, which was thought to be the result of cerebral oxygen toxicity, a rare but well-known side effect of hyperbaric oxygen therapy.^[5] However, back on the ward she developed another two tonic-clonic seizures accompanied with hypotension and bradypnoea upon which she was intubated, antiepileptic medication was started and the patient was transferred to the ICU. CT scan of the brain (*figure 3*) showed swelling of the right frontal lobe, and no bleeding or signs of ischaemia. No further seizures developed and two days after ICU admission, she was successfully extubated. Neurological examination showed maximal GCS, but paralysis of her left arm and a severely paretic left leg, consistent with the abnormalities in the right frontal lobe seen on CT. Besides the development of atrial fibrillation, the haemodynamic parameters remained stable. On the third day of her stay in the ICU she was transferred back to the referring hospital. Remaining neurological abnormalities at that time were paralysis of her left arm and mild paresis of her left leg. At follow-

up, four months after discharge from our hospital, the paralysis of her left arm had disappeared. Paresis of her left leg had also improved except for a slight persisting foot drop.

Discussion

We present a case of coronary and cerebral arterial gas embolism after flushing of a pleural drain. In our patient, it was quickly recognised that the neurological abnormalities could very well be the result of arterial gas embolism. This resulted in timely transfer to our hospital and treatment with hyperbaric oxygen therapy. Her neurological situation greatly recovered after two courses of hyperbaric oxygen therapy. The tonic-clonic seizures that developed at the end of the second treatment session were in retrospect probably not a sign of cerebral oxygen toxicity, since they reoccurred hours after hyperbaric oxygen therapy. They were most likely a result of cerebral swelling due to capillary leakage caused by the arterial gas embolism.

Cerebral and coronary arterial gas embolism can develop in four ways. It can evolve from direct air entry into the pulmonary veins, as occurred in our patient and has been described previously.^[6] This is also the mechanism in divers holding their breath during ascent, or when arterial gas embolism occurs as a complication of mechanical ventilation.^[7] Another cause of arterial gas embolism is direct air entry in the left side of the heart, the aorta or carotid arteries, as can occur during cardiac, aortic or carotid surgery.^[8] A third cause is paradoxical embolism resulting from air entry into the systemic veins, for example during placement of a central venous line, followed by shunting into the arterial system through a cardiac right-to-left shunt or due to overflow of air from the pulmonary arteries to the veins.^[9] The last possibility is direct introduction of air into a systemic artery, for instance due to flushing of a radial artery catheter with retrograde ascent of bubbles into the aortic arch and then into the carotid arteries.^[10] Regarding our case, it is remarkable that arterial gas embolism developed after flushing the drain, especially because the drain had been flushed regularly in the days preceding the event without complications. Possibly, movement of the drain had occurred, damaging the pleural viscera, making the pulmonary veins vulnerable for air entry. Another way of damage to the visceral pleura could have been through excessive force during flushing.

In our patient, neurological deterioration immediately after flushing of the pleural drain supported the diagnosis of arterial gas embolism. Many cases, however, are less clear cut, and the possibility of arterial gas embolism may not be considered. This might be the result of the common belief that arterial gas embolism is a rare condition. Indeed, a large clinical study estimated the incidence of cerebral arterial gas embolism as being 5.7 in every 1,000,000 admitted patients.^[11] However, this number is likely an underestimation of the actual incidence. Contributing factors to this underestimation are the broad differential diagnosis of acute in-hospital neurological or

cardiac deterioration, the difficulty in diagnosing arterial gas embolism with imaging techniques^[4] and the lack of knowledge of the disorder. The presumptive clinical diagnosis of arterial gas embolism should be made in all cases of acute cerebral or coronary ischaemia after witnessed or possible vascular air introduction. CT scan of the cerebrum may be used to rule out other diagnoses, but should not be used to exclude cerebral arterial gas embolism, since air bubbles may not be visible.^[12] Cardiac ischaemia may be diagnosed with ECG and cardiac enzymes, bearing in mind that coronary angiography will not reveal a thrombus in cases of arterial gas embolism. In all cases of proven or suspected arterial gas embolism, a hyperbaric facility should be consulted immediately. Large case series show that a delay of more than six to seven hours until starting hyperbaric oxygen therapy is associated with a reduced chance of successful outcome and therefore all efforts should be made to transport the patient to a hyperbaric facility as soon as possible.^[11,13]

The mechanisms of action of hyperbaric oxygen therapy in arterial gas embolism are fourfold. Firstly, direct pressure on the bubble causes a decrease in size following Boyle's law (at a constant temperature, the volume of a fixed amount of a gas varies inversely with its pressure).^[14] Secondly, it promotes gas resorption by applying high concentrations of oxygen, reducing the partial pressure of nitrogen in the blood and tissues, thus providing a large gradient for nitrogen absorption from the air bubble.^[4,15,16] In the third place, the high partial oxygen tensions improve tissue oxygenation of marginally perfused tissue (the penumbra), promoting cellular survival. Lastly, hyperbaric oxygen therapy has immunomodulatory properties, for instance by decreasing inflammation by reducing leucocyte adherence.^[4] Multiple hyperbaric oxygen therapy regimens exist, but most experts advise primary treatment of arterial gas embolism with US Navy Treatment Table 6, which consists of initial compression to 2.8 atmospheres absolute (ATA), the equivalent of a dive to a depth of 18 meters, followed by decompression to 1.9 ATA (9 meters water depth). Total duration is almost five hours. Hyperbaric oxygen therapy can be repeated daily as long as there is stepwise improvement after each session, and is usually performed for two hours at 2.4-2.5 ATA (14-15 meters water depth).^[17] Our patient responded favourably to two courses of hyperbaric oxygen therapy and was then transferred back to the referring hospital.

Conclusion

In this case report we present a case of arterial gas embolism after flushing of a pleural drain, resulting in myocardial as well as cerebral hypoxia. The patient responded favourably to hyperbaric oxygen therapy. Arterial gas embolism is rare but the incidence is presumably underestimated. Prompt recognition of this condition is vital, since urgent administration of hyperbaric oxygen therapy is advantageous. Acknowledging the four possible ways in which arterial gas embolism can develop helps in making the right diagnosis.

Disclosures

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