

## Non-Accidental Cerebral Air Embolism Secondary to Pulmonary Fibrosis

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### Abstract

Cerebral air embolism (CAE) is a rare cause of stroke. Most cerebral air emboli are caused by iatrogenic factors, such as invasive cardiac and pulmonary procedures. Here, we report an unusual case of CAE not related to any medical intervention. We are reporting a case of 75-year-old man who has recent diagnosis of idiopathic pulmonary fibrosis, suddenly became unresponsive after straining in the toilet. A computed tomography (CT) scan of the brain done immediately in the hospital which revealed multiple dark foci (gas foci) bilaterally more in the right side. Patient was aphasic with bilateral weakness worse in upper limbs, MRI of the brain confirmed acute infarction in bilateral frontoparietal lobe, chest x ray done which showed no evidence of air in thorax. On Second day patient has echocardiogram which did not reveal significant cardiac shunt or PFO (patent foramen ovale). In this case, the increased intra-thoracic pressure may have been a trigger for the CAE especially in a patient with pulmonary vulnerability. This case suggests that CAE can occur in a non-accidental situation.

**Keywords:** Cerebral Air Embolism; Straining, Pulmonary Fibrosis; Cerebral Infarction

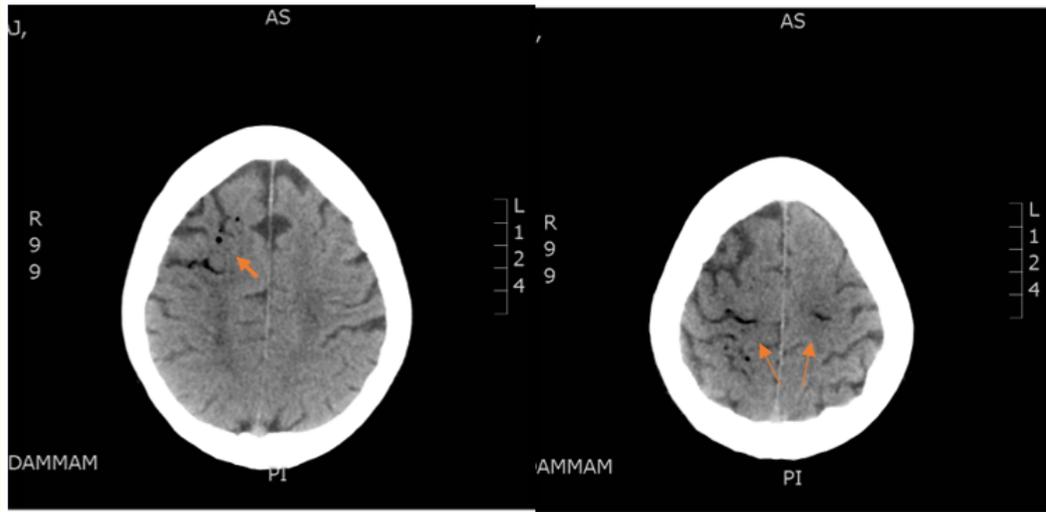
### Introduction

Cerebral air embolism (CAE) is a cerebral infarction caused by the entry of air into the vascular structures. CAE is largely recognized as an iatrogenic clinical problem [1]. On the other hand, there are few reports on CAE occurring in non-iatrogenic situations. Here, we report a rare case of CAE not related to an invasive medical procedure.

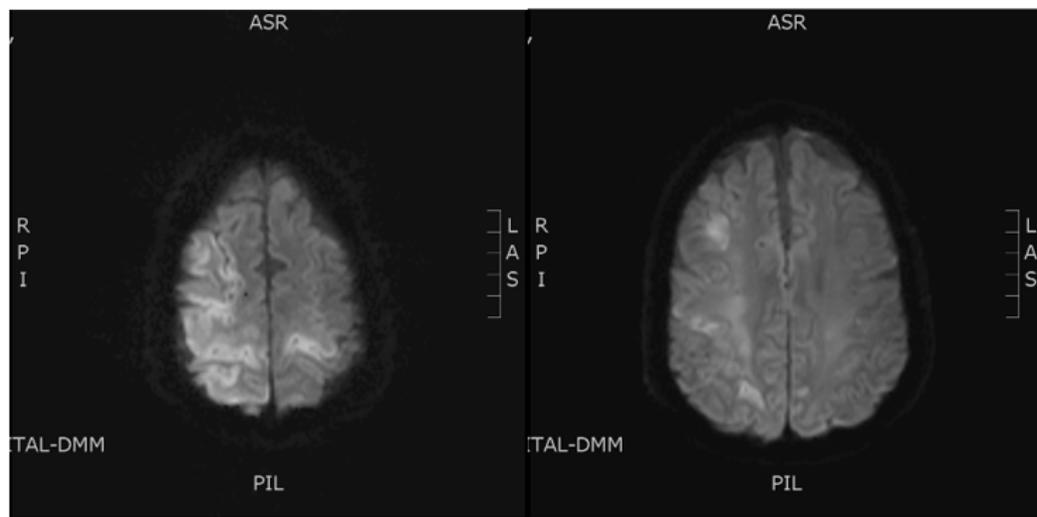
### Case Presentation

A 75-year-old man was admitted to the hospital under pulmonary team for further investigations for idiopathic pulmonary fibrosis. The patient had a history of hypertension, idiopathic pulmonary fibrosis and he is EX-smoker. Two days after admission the patient become unresponsiveness after straining in the toilet, initial assessments indicated the following he was hemodynamically stable but unresponsive, with a Glasgow Coma Scale of 9. His pupils were equal-sized and reactive to light, opening eyes spontaneously with no verbal output. His best motor response on initial assessment was localization of painful stimuli within Upper limbs more than lower limbs, Oculocephalic, corneal reflex, Cough and gag reflex were intact.

Then he was intubated and code stroke protocol was activated. Metabolic screen on same day showed no significant abnormality, his unenhanced head computed tomography (CT) scan (Figure 1) showed multiple dark foci which represent air in the subarachnoid spaces in the right frontal lobe and parietal lobe. Minimal gas in the contralateral side at the same level seen as well. Immediately after the CT scans, a brain MRI was done, which showed bilateral asymmetric frontoparietal diffusion restriction. Findings are likely representing ischemia secondary to air embolism (Figure 2). He also had repeated CT scan for brain after 24 hours which revealed near complete resolution of the foci of gas seen in the right frontoparietal lobe along the sulci. Chest x ray done which showed no evidence of air in thorax, CT for chest was not done as the patient was hemodynamically unstable. Echocardiogram on second day did not reveal significant cardiac shunt or PFO (patent foramen ovale).



**Figure 1:** Non-contrast computed tomography (CT), axial view, showed Multiple dark foci (gas foci) in the right frontal lobe and parietal lobe with minimal gas in the contralateral side at the same level (arrows).



**Figure 2:** Diffusion-weighted imaging sequences of non-contrast magnetic resonance imaging, axial view, showed Bilateral asymmetric frontoparietal lobe cortical/subcortical swelling right greater than left with diffusion restriction.

We recommended to start him on hyperbaric O<sub>2</sub> therapy but it not available in our facility, unfortunately, his neurologic status did not improve and he died shortly after withdrawal of life support.

### Discussion

We found in the literature review that most cases of CAE are secondary to iatrogenic causes such as central catheter placement, endoscopic procedure and others invasive procedure as well as non-invasive one (e.g. BiPAP or CPAP) which are associated with increase intra-thoracic pressure, on the other hand there are few cases of CEA are secondary to non-iatrogenic causes and in all these cases there is an underlying pulmonary vulnerability, we found 3 published cases of non-iatrogenic CAE which were associated with Pneumothorax and Pneumomediastinum (Matsuura, *et al* [2], Tabata, *et al* [3], Brunelli, *et al* [4]). However, in our case, there was no evidence of pneumothorax.

In general, air embolism can be venous or arterial and most common causes are Surgery, trauma, vascular interventions, and barotrauma from mechanical ventilation and diving [5-8]. Less common causes such as central line, hemodialysis and intravenous contrast injection can also lead to entry of air to blood stream. The pathophysiology behind presence of air embolism in our blood stream can be associated with three distinct processes [9,10]:

- The incomplete filtration of venous air emboli by the pulmonary capillaries.
- The direct introduction of air into the arterial system, usually through a breach in an arterial vascular bed (e.g. trauma or surgery, barotrauma). Reduced alveolar permeability to gas is also thought to result in the increased risk of arterial air embolism that can worsen during anesthesia.
- The paradoxical embolization of air through a septal defect, patent foramen ovale, or pulmonary arteriovenous malformation (including hepatopulmonary syndrome). In patients with a left-to-right shunt, significant volumes of air in the right heart/pulmonary circulation can raise right heart pressures and reverse the direction of the shunt, also allowing paradoxical embolism to occur [11,12].

In our case, the patient's straining caused a rapid increase in the intra- pulmonary pressure, and may have caused a subsequent rupture of the alveoli and the nearby tissue. Thus, we hypothesize that the straining was a trigger not only to induce the pneumomediastinum but also to push the air into the pulmonary vein through the damaged vessel wall especially in patient with underlying pulmonary vulnerability. Which in order lead to entry of air to the heart and subsequently to the brain. As our hospital did not have hyperbaric oxygen, we couldn't offer this option to the patient in addition to his condition was rapidly deteriorating and unfortunately, he died shortly.

Hyperbaric oxygen therapy (HBO) is not routinely administered in patients with air embolism but is a useful adjunct in severe cases, which is often those with arterial air embolization. When available, HBO should be administered to patients with evidence of hemodynamic or cardiopulmonary compromise, as well as those with neurologic deficits, or other evidence of end-organ damage [13,14]. It's better to administered HBO within the first four to six hours after the symptom onset but continued benefit has been reported when HBO therapy is delayed for up to 30 hours [15,16].

The potential clinical benefits of HBO are mostly derived from small retrospective case series that examine patients with neurologic deficit from air embolism [17,18]. These studies are limited by small sample size, their retrospective design and lack of adjustment for severity of illness. Nonetheless, collectively they report benefit particularly when HBO is administered within the first four to six hours after symptom onset. As examples:

- In one retrospective cohort study of 86 patients with venous or arterial air embolism, patients treated with HBO within six hours of presentation were more likely to recover than patients who received HBO after six hours or more (68 versus 40 percent), with outcomes best in those who underwent HBO within three hours [19].
- In another case series of 17 patients treated with HBO for air embolism that developed during cardiac surgery, all five patients who were treated within three hours from the operation and half (two out of four) of those treated three to five hours from operation experienced a full neurologic recovery. With a delay of 9 to 20 hours, only one (out of eight) patients had a full neurologic recovery [20].
- HBO is of limited availability and only highly specialized centers have expertise in the provision of this therapy. It is an intermittent therapy that often requires physical transfer of patients which can infer risk of cardiac arrest or death during transfer. Thus, the benefits of HBO therapy should be weighed against the risk of death during transfer and discussed with the patients and caregivers in every case.

### Conclusion

Air embolism is an uncommon, but potentially catastrophic, event that occurs as consequence of the entry of air into the vasculature. Surgery, trauma, vascular interventions, and barotrauma from mechanical ventilation and diving are the most common causes of air embolism but also, we have put in our mind the possibility of having CAE without any trauma or intervention as well epically with underlying pulmonary vulnerability. Air embolism should always be suspected when patients experience sudden-onset respiratory distress and acute neurological event even without the setting of accidental factor (e.g. intravenous catheter insertion, trauma).

### Disclosure

Nothing to be disclosed.

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