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Massive Ischemic Stroke Due to Pulmonary Barotrauma and Cerebral Artery Air Embolism During Commercial Air Travel

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Conflict of interest: None declared

Patient: Male, 65
Final Diagnosis: Air emboli
Symptoms: Short of breath
Medication: —
Clinical Procedure: —
Specialty: Anesthesiology

Objective: Unusual setting of medical care

Background: Air embolism into the systemic arterial circulation secondary to pulmonary barotrauma has rarely been reported. Herein, we report the clinical course of an extremely rare presentation of cerebral air embolism likely due to ruptured pulmonary bullae during commercial air travel.

Case Report: A 65-year-old man suddenly became unconscious during an airplane descent. Upon landing, he was immediately transferred to the nearest emergency department where he was intubated for airway protection. His head CT angiogram showed multiple air pockets in the right parietal lobe suspicious for multiple air emboli. His chest CT scan showed multiple large bullae in the left upper and lower lobes as well as diffusely emphysematous lung tissue. After initial stabilization, he underwent emergent hyperbaric oxygen treatment (HBOT) in the multiplace chamber at 2.8 atmospheres. The patient tolerated HBOT well with no complications. However, his neurologic status deteriorated in the following 24 hours due to progression of his cerebral edema and mass effects. The patient's clinical status was discussed with his family and the decision was made to withdraw life-sustaining measures. He died shortly after withdrawal of life support. Post-mortem examination confirmed the presence of very large bullae in the lungs bilaterally.

Conclusions: Spontaneous cerebral air embolism is a possible complication of ruptured pulmonary bullae during air travel. HBOT is well-tolerated and may be used with caution even in the presence of emphysematous bullae.

MeSH Keywords: Barotrauma • Embolism, Air • Stroke

Full-text PDF: http://www.amjcaserep.com/abstract/index/idArt/903354

Background

Introduction of air into the arterial system (arterial air embolism) is a rare but potentially catastrophic event that can occur following trauma (particularly head and neck injuries), vascular interventions (insertion of arterial catheters or other arterial procedures), or secondary to pulmonary barotrauma (as a rare complication of positive-pressure mechanical ventilation and SCUBA diving) [1,2]. Air embolism into the systemic arterial circulation due to pulmonary barotrauma during air travel is, however, extremely rare [3–5].

Here, we report a case of massive ischemic stroke as a result of cerebral artery air embolism that occurred during a commercial air travel, possibly as a consequence of ruptured pulmonary bullae. In addition to supportive care, our patient received hyperbaric oxygen treatment (HBOT) in an attempt to reduce the size of the air bubbles. The therapy was administered with caution given the presence of emphysematous bullae and was tolerated well with no complications. However, our patient's neurologic status deteriorated 24 hours after the initial presentation due to progression of cerebral edema, which, unfortunately, resulted in his death.

Case Report

A 65-year-old man, an ex-smoker, with a past medical history of hypertension, hyperlipidemia, and recent intentional weight loss of approximately 14 kg, but with no previous known history of lung disease, suddenly became unconscious during an airplane descent at the end of a 3-hour flight. The event, which was observed by his family, was not associated with any acute neurological or cardiac symptoms and the patient was noted to be completely well and asymptomatic on the preceding days.

A few minutes after the onset of symptoms, the plane landed in Toronto and Emergency Medical Services (EMS) were activated. Upon EMS arrival, the patient was found to be unconscious and was immediately transferred to the nearest emergency department. He remained hemodynamically stable during this time and no CPR was performed by bystanders or EMS. In hospital, initial assessments indicated the following: He was hemodynamically stable but unconscious, with a Glasgow Coma Scale of 8. His pupils were equal-sized and reactive to light. His best motor response on initial assessment was localization of painful stimuli with his right arm. He was intubated for airway protection and the emergency stroke protocol was activated. His unenhanced head CT scan and, subsequently, his head CT angiogram (Figure 1A) showed multiple air pockets in the right parietal lobe suspicious for multiple air emboli or trauma but no occlusion of large intra-cranial or extra-cranial vessels. Immediately after the CT scans, a brain MRI was done, which showed extensive ischemic changes in the right hemisphere with multiple defects involving anterior, middle, and posterior cerebral artery territories (Figure 1B, 1C). He also had a chest CT scan that showed multiple large bullae in the left upper and lower lobes, as well as diffusely emphysematous lung tissue, but no evidence of pneumothorax, pneumomediastinum, or intravascular air (Figure 1D).

Approximately 3 hours after the onset of symptoms, the patient was transferred to our center for emergency hyperbaric oxygen treatment (HBOT). He underwent HBOT in a multiplace chamber at 2.8 atmospheres according to treatment table 6 of the Undersea and Hyperbaric Medical Society (285 minutes) [6]). Bilateral prophylactic chest tubes were considered but not inserted before HBOT; a thoracic surgeon was on standby in case of a newly developed pneumothorax during HBOT. The patient tolerated HBOT well with no complications.

He was then transferred to the intensive care unit for ongoing supportive care including mechanical ventilation. He remained hemodynamically stable during his ICU stay but, unfortunately, his neurologic status deteriorated in the following 24 hours. He had a repeat brain CT scan (Figure 2A) that showed interval progression of edema and mass effect related to the previously described areas of diffuse cerebral and cerebellar infarcts. A repeat chest CT scan showed interval development of pneumomediastinum but no evidence of pneumothorax (Figure 2B).

The patient's neurological status and the CT scan findings were discussed with his family. It was clear, based on his previously stated wishes, that he would not want to continue with life-sustaining measures in this context. He died shortly after withdrawal of life support.

Post-mortem examination confirmed the presence of very large bullae on the left lung, measuring approximately 12×12×6 cm in composite. In addition, the left lung contained a single large tense air-filled bulla measuring 9.0×8.0×7.0 cm in size. Multiple bullae were also present in the right lung, measuring 8.0×8.0×6.0 in composite. Histologically, the lungs demonstrated dilated air spaces and loss of alveolar walls consistent with centriacinar emphysema, likely secondary to smoking. Cardiac examination on autopsy showed presence of patent foramen ovale and neuropathology confirmed a large acute infarct in the right middle cerebral artery territory. Death was attributed to brain ischemia from air emboli released from emphysematous bullae in the lung secondary to pulmonary barotrauma.

Discussion

Cerebral air embolism secondary to pulmonary barotrauma during air travel is extremely rare, with only 3 cases being

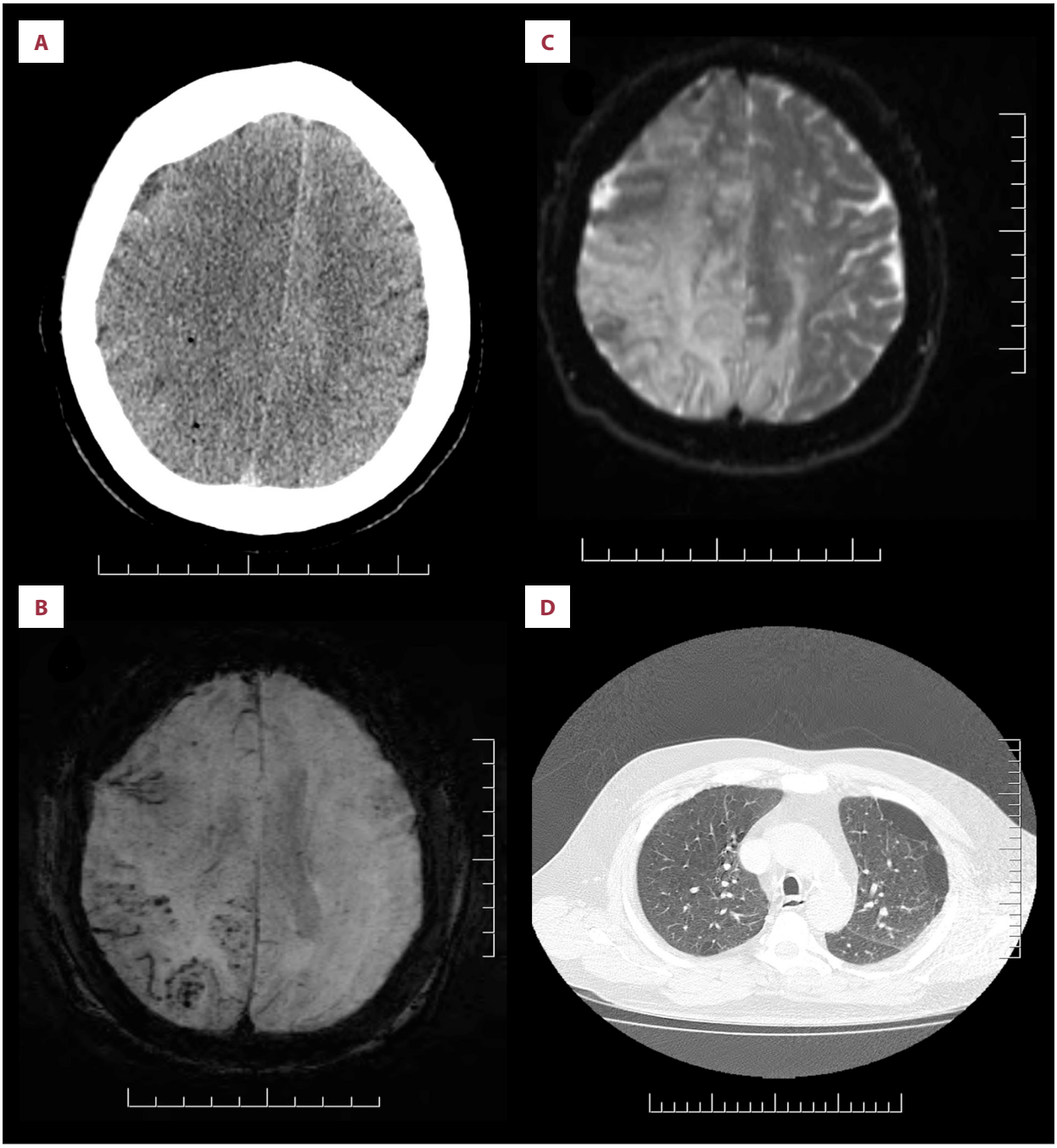

Figure 1. (**A**) Unenhanced head CT scan showing multiple pockets of air. (**B, C**) MRI images showing air pockets as well as extensive ischemic changes involving multiple vascular territories. (**D**) Chest CT scan showing large pulmonary bullae and emphysematous changes.

previously reported in literature [3–5]. The clinical presentation of cerebral air embolism in these cases range from acute focal neurologic symptoms such as hemiparesis and aphasia in a conscious patient to acute loss of consciousness and coma. Severity of the clinical syndrome and the clinical outcome appear to correlate with the extent of the intra-parenchymal gas and the associated cerebral edema. Our patient presented with acute loss of consciousness. He was found to have extensive areas of ischemic infarcts within cerebral and cerebellar hemispheres associated with multiple small air pockets in distal arteries but without occlusion of large extra-cranial or intra-cranial vasculature. He was therefore not a candidate for mechanical interventions aimed at reducing the bubble size, including procedural aspiration of the air pockets.

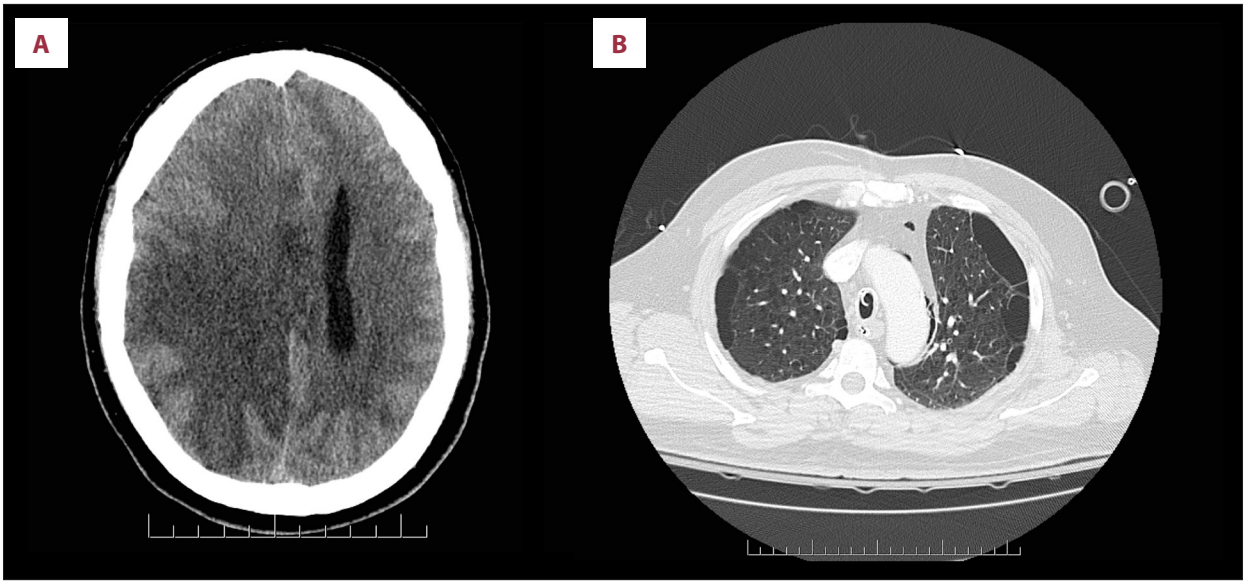

Figure 2. (**A**) Head CT scan showing interval progression of cerebral edema and mass effect. (**B**) Chest CT scan showing interval development of pneumomediastinum.

He underwent HBOT but, unfortunately, his neurological status did not improve following this treatment. Of the 3 other cases that have been reported in the literature, 2 presented with acute loss of consciousness. These patients were considered to be too critically ill to be transferred to a hyperbaric chamber and subsequently died [3,4]. In contrast, the case described by Gudmundsdottir et al. presented with focal neurologic deficit but did not lose consciousness [5]. He survived the acute event and underwent conventional oxygen therapy, which resulted in symptomatic improvement.

The pathophysiology of the arterial air embolism secondary to pulmonary barotrauma associated with air travel is not clearly understood [7,8]. It is hypothesized that previously asymptomatic pulmonary bullae may expand during air travel due to lower ambient pressures. As a result of this stretch injury, the pulmonary bullae may rupture and release its gas into the pleural space. Air embolism into the systemic circulation may then occur through any of the following mechanisms [9]:

1. Direct embolization of the gas bubble into the pulmonary veins and from there into the systemic circulation.
2. Gas embolization into the pulmonary arterial system with subsequent incomplete filtration of the gas bubbles by the pulmonary capillaries.
3. Paradoxical embolization through a functional right-to-left shunt, such as a patent foramen ovale.

We suggest that all these mechanisms may have played a role in our case. Of note, the patient was found to have a patent foramen ovale on autopsy. However, surprisingly, his initial chest CT scan did not show any evidence of pneumothorax, pneumomediastinum, or intravascular air.

HBOT has been used in severe cases of arterial air embolization to reduce bubble size. It is thought that the high oxygen tension promotes the resorption of nitrogen from the bubble and the elevated ambient pressure reduces the size of the bubbles in accordance with Boyle's law, such that bubble passage through the microcirculation and resolution of embolic phenomena may occur [6,10].

Given the severity of neurological status, the emergency nature of treatment, and lack of other therapeutic options, in the present case it was decided to proceed with HBOT despite the presence of emphysematous lung bullae, a relative contraindication to the treatment due to the barotrauma risk. The biggest risk related to bullous lung disease in HBOT is the bullae rupture due to changes in ambient pressure during decompression. The bullae rupture can cause tension pneumothorax with potential hemodynamic collapse and air gas embolism with neurological and hemodynamic compromise. This was minimized by exerting great caution in ensuring a slow decompression phase. The multiplace chamber attendants and hyperbaric physician were aware of the high risk of potential pneumothorax and were ready for thoracostomy if needed.

Conclusions

In summary, we present an unfortunate case of spontaneous cerebral air embolism during air travel in a patient with pulmonary bullae. Although the patient ultimately died due to ischemic neurological injury, HBOT was well-tolerated and should be considered for similar situations, even in the presence of emphysematous bullae.

Conflict of interests

No relevant conflict of interests.

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