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A patient with chronic obstructive pulmonary disease developed a cough, loss of consciousness, and convulsions during an air flight. Chest radiography showed a large lung bulla. Computed tomography of the brain showed intraparenchymal air and bilateral cerebral infarcts. The findings were compatible with cerebral air embolism, most likely predisposed to by lung bulla and an air flight. The underlying pathology and possible treatment are discussed.

Case report

A 69-year-old tourist from Hangzhou with a history of chronic obstructive pulmonary disease (COPD) travelled to Hong Kong by plane in March 2012. He developed a cough and loss of consciousness shortly after the plane took off and was given oxygen during the flight, and 2 hours later developed convulsions. He was sent to our hospital for medical attention and his convulsions were controlled by intravenous diazepam. He remained unresponsive with occasional limb and facial twitching, whereupon he was intubated for airway protection and ventilation. A chest radiograph (Fig 1) showed a giant bulla occupying the middle and lower zones of right lung. Urgent computed tomography (CT) of the brain showed several tiny gas pockets within the brain parenchyma, diffuse cerebral oedema, and hypodensity of both cerebral hemispheres with loss of grey-white differentiation (Fig 2). The findings were compatible with air embolism with bilateral cerebral infarcts. A repeat CT brain 6 hours later showed that the air had been resorbed, and the extensive bilateral acute cerebral infarcts were associated with more severe oedema. In view of the grave prognosis, the patient received supportive treatment. He developed a cardiac arrest about 18 hours after onset of the event and succumbed. Autopsy confirmed the presence of a 16-cm right lung bulla with no pneumothorax. The brain was heavy, weighing 1599 g, and oedematous. Bilateral microinfarcts and haemorrhages were evident in the thalamic regions and brainstem.

Discussion

Air embolism may be due to iatrogenic causes, diving, and trauma.¹ Air embolism occurring during an airplane flight is rare, but has been reported in the literature. Almeida and Schuller² reported a woman with cystic adenomatoid malformation who suffered a cerebral air embolism during a flight, and was able to recover partially and went on to rehabilitation. Moreover, fatal strokes due to cerebral air embolism in airplane passengers with pulmonary bronchogenic cysts have been reported by Edwardson et al³ and Zaugg et al.⁴

Air embolism in patients with pre-existing lung pathology during air flight is likely related to decompression during ascent. Commercial aircrafts are pressurised during flights to maintain a cabin pressure equivalent to an altitude of 8000 ft or below.⁴ The pressure drop inside the cabin during ascent from sea level to cruising level therefore amounts to around 200 mm Hg. According to the Boyle-Mariotte's law, an air-containing bulla can increase in volume by up to around 35% with this change in pressure. Expansion of the bulla can cause tears of its wall, and air leakage into the circulation.² A giant bulla (occupying more than 30% of the hemithorax) was present in the right lung of our patient, and was probably the predisposing cause of his air embolism.

Neurological deficits due to cerebral air embolism may be reversible.¹ Emergency treatment for air embolism includes administration of 100% oxygen and hyperbaric therapy.¹ Delivery of high levels of oxygen counteracts the ischaemia caused by air embolism, and reduces the size of the air bubbles by depleting the contained nitrogen.¹ Hyperbaric therapy compresses the bubbles, reducing the extent of inflammation secondary to their presence, whilst dexamethasone may also help prevention of cerebral oedema.¹

Hodics and Linfante⁵ reported a patient who suddenly developed decreased responsiveness during chest tube placement; CT brain showed air density in the cortical branches of the right middle cerebral artery and diffusion-weighted magnetic resonance

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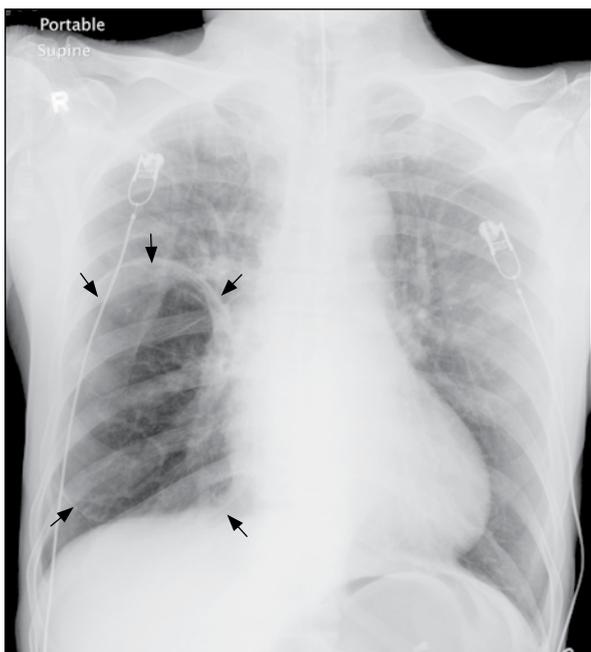


FIG 1. Chest radiograph shows a large bullae over the right middle and lower zones (arrows)

imaging showed increased signal intensity in the corresponding sites. The patient made a full recovery in 1 week with induced hypertension and hyperoxygen treatment. The good outcome was likely related to small areas of cortical involvement, early diagnosis, and early treatment. By contrast, our patient had already developed extensive bilateral infarcts on presentation to our hospital, which led to his inevitable fatal outcome.

Pre-flight screening may be considered in patients with respiratory symptoms. As pulmonary bullae are a common finding in patients with COPD, doctors may consider requesting chest radiographs to look for bullae when advising them on the safety of air travel.

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與乘搭飛機相關的致命腦空氣栓塞

一名有慢性阻塞性肺病的病人在乘搭飛機時出現咳嗽、意識喪失和抽搐。胸部影像顯示一個大的肺大疱，而腦部的電腦斷層掃描顯示腦實質內空氣和雙側腦梗塞，結果與腦空氣栓塞症吻合。有肺大疱的病人乘坐飛機有可能出現腦空氣栓塞。本文續討論此病潛在的病理和可行的治療方法。



FIG 2. Plain computed tomographic brain: bilateral cerebral hemispheres show diffuse swelling with sulcal effacement and decrease in attenuation with loss of grey-white differentiation (arrows). Tiny intraparenchymal gas pockets were present in both frontal lobes (arrowhead); such findings are compatible with air embolism-related cerebral infarcts in both hemispheres