

Cerebral air embolism after pleural streptokinase instillation

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Abstract

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Iatrogenic pulmonary barotrauma and cerebral arterial gas embolism (CAGE) may complicate a variety of medical procedures, such as certain types of surgery, drug administration through thoracic drainage, pneumoperitoneum, cystoscopy, bronchoscopy, etc. Hyperbaric oxygen treatment following the guidelines for CAGE in diving is the treatment of choice. Pleural streptokinase instillation is a common treatment for parapneumonic pleural effusion and may lead to CAGE. We present such a complication in a 79-year-old woman with a left-sided empyema. Neurological recovery was reasonable, but a left hemiparesis persisted. Prompt treatment of CAGE is necessary to avoid permanent injury and severe disability.

Key words

Pulmonary barotrauma, cerebral arterial gas embolism (CAGE), hyperbaric oxygen therapy, case reports

Introduction

Iatrogenic pulmonary barotrauma and arterial gas embolism (AGE) may complicate a variety of medical procedures.¹⁻⁵ We present a case of cerebral AGE (CAGE) in a patient undergoing pleural streptokinase instillation.

Case report

A 79-year-old woman, with a medical history of arterial hypertension, hypothyroidism after radioiodine-therapy 30 years before, an untreated umbilical hernia, diverticulitis and fractures of the humerus and pelvis two months before, was admitted to hospital because of a decline in her general health. Chest X-ray showed left-sided pneumonia and empyema. Antibiotic therapy with tazobactam and piperacillin was started and the pleural effusion was relieved with a dual-sump drain. Next day, chest X-ray showed a persistent pleural effusion, so 250,000 i.u. streptokinase were instilled into the pleural space via the drain. Immediately after instillation, the patient showed a marked reduction of consciousness associated with a right lateral gaze.

She was intubated and mechanically ventilated. Cranial computer tomography (CT) showed multiple pockets of cerebral air (Figures 1 and 2), whilst a thoracic CT showed no pneumothorax (Figure 3). Because of the suspected diagnosis of CAGE with the need for hyperbaric oxygen treatment (HBOT), transfer to a hospital with a hyperbaric chamber was arranged. Organisation and transfer took about 24 hours. At the hyperbaric centre, the sedated, intubated and mechanically ventilated patient was treated with a US Navy Treatment Table 6 (USN TT6) with extensions (in total, 3 hours at 280 kPa and 4 hours at 190 kPa). After HBOT, repeat cranial CT showed no more cerebral air. Further HBOT was not undertaken because of the non-availability of hyperbaric staff. After reduction of sedation in the intensive care unit, the patient showed inadequate awaking, a left lateral gaze and focal seizures. An EEG showed status epilepticus,

which responded to phenytoin, topiramate and levetiracetam. The patient underwent tracheostomy because prolonged weaning from mechanical ventilation was anticipated. A further cranial CT showed ischaemic infarcts in the areas of the right anterior and middle cerebral arteries; chest CT showed no changes from the previous CT.

Over several weeks her neurological status improved and she was discharged to a neurological rehabilitation unit six weeks after the incident. At that time, she had a persistent left hemiparesis, predominantly of the arm, and could speak, sit and walk up to 150 metres with the assistance of a wheeled walker. She had no more seizures under anti-epileptic medication.

Discussion

AGE is a rare but well-recognised complication of certain types of surgery, intravascular radiological procedures, drug administration through thoracic drainage, pneumoperitoneum, cystoscopy, bronchoscopy, gunshot or penetrating injuries of the thorax, etc.¹⁻⁵ Clinical signs should lead to diagnosis. In some cases, the cranial CT shows no gas in the cerebral circulation as gas is usually pushed through the cerebral circulation. Sometimes persistent air bubbles may be too small to detect with routine CT.⁶

Whether the cause is iatrogenic or diving accidents, HBOT reduces bubble size and enhances elimination of any inert gas.³ Furthermore, the high oxygen partial pressure can maintain aerobic metabolism in poorly perfused tissue by diffusion and reduces cerebral oedema.⁷ Prompt HBOT could lead to a *restitutio ad integrum*.^{3-5,8} Similarly to the treatment of CAGE in divers, a second (or more) HBOT should follow the first within 24 hours if symptoms persist.⁹ In our case, an extended USN TT6 was delayed 24 h and further treatment was not possible for logistic reasons. The cerebral ischaemic infarcts reported on follow-up CT might have been avoided if treatment could have been commenced

Figure 1
CT of the brain, arrow shows air pocket

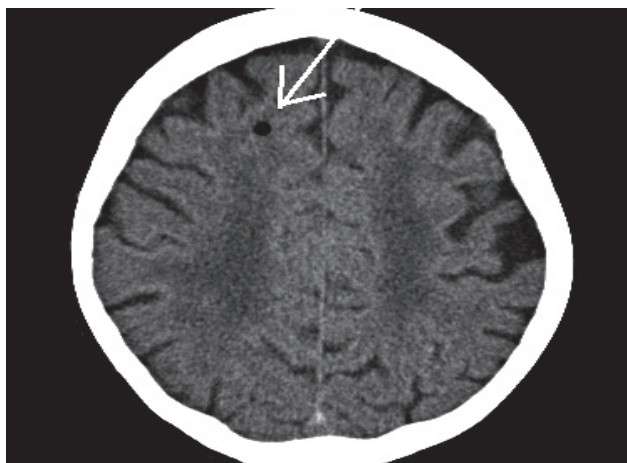


Figure 2
CT of the brain, arrows show air pockets



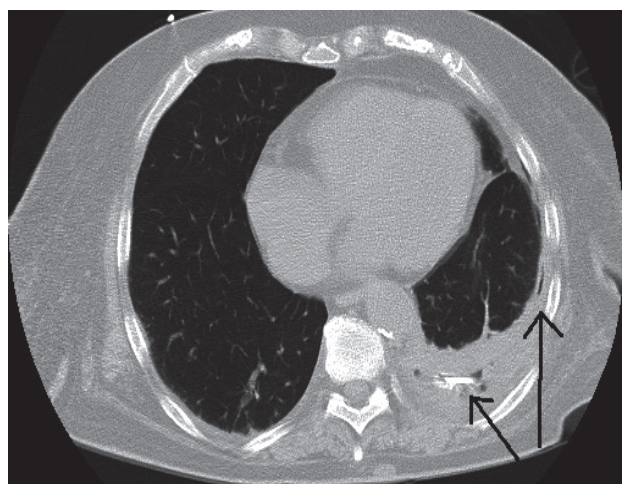
earlier. Unfortunately in Germany there is a lack of hyperbaric facilities with 24-hour emergency intensive care capability as a result of governmental policies. Nevertheless, and not withstanding that studies suggest that clinical outcome is better the shorter the delay to recompression following CAGE, HBOT is the only effective treatment for CAGE.⁹ The take-home message is that treatment of CAGE with HBOT has to happen as soon as possible, because “time is brain”.

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Figure 3

CT of the thorax, showing left-sided empyema, arrows show some air pockets and a chest drain



from: <http://www.gtuem.org/wcms/ftp//g/gtuem.org/uploads/leitlinietauchunfall2011awmftemplateleitlinienreport.pdf>

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