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Cerebral air embolism after indwelling pleural catheter insertion in a chronic hydropneumothorax secondary to epithelioid mesothelioma

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SUMMARY

A 75-year-old man with a history of epithelioid mesothelioma and a right-sided indwelling pleural catheter (IPC) presented with a history of a purulent fluid drainage via the IPC. The pleural fluid cultured *Klebsiella oxytoca* and *Enterococcus faecalis*. He was treated with a course of oral fluoroquinolone followed by uneventful IPC replacement. One and half hours postprocedure, the patient had a witnessed drop in conscious level accompanied by seizure like activity. Acute stroke was suspected and a CT head was performed. CT head revealed multiple serpiginous pockets of air along the cerebral fissure, with features that were highly suggestive of cerebral air embolism and multiple wedge-shaped areas of infarction involving the cerebral hemispheres. Further imaging revealed satisfactory position of the replaced IPC. The patient was admitted to the intensive care unit for high flow oxygen therapy and head down ventilation. However, his condition deteriorated and he died later.

BACKGROUND

Indwelling pleural catheters (IPC) are used in the management of benign and malignant pleural effusions. The procedure is usually carried out as a day case under local anaesthesia. IPC insertion may be associated with complications such as pleural infection, bleeding, pneumothorax, chest pain, blockage and leakage but the procedure may be associated with more serious complications.

Cerebral air embolism (CAE) is a rare but important complication of pleural procedures that has not been previously reported after IPC insertion. CAE is induced by barotrauma, chest injuries and iatrogenic causes such as cardiovascular surgery and invasive procedures. Pleural procedures such as pleural fluid aspiration, chest drain insertion and thoracoscopy have also been recognised as causes of CAE.

We report a case of CAE that occurred after the routine exchange of an IPC in a patient with a chronic complex hydropneumothorax secondary to epithelioid malignant mesothelioma

CASE PRESENTATION

A 75-year-old man with a 7-year history of epithelioid mesothelioma and a right sided IPC presented to the pleural clinic with a history of 2–3 weeks of a malodourous, purulent fluid drainage via the IPC.

The patient was Eastern Cooperative Oncology Group performance status 0 and was under the ambulatory pleural service but managed his pleural collections independently in the community. The IPC had been inserted 3½ years ago for a right-sided loculated hydropneumothorax that had developed after a radical extended pleurectomy, decortication and diaphragmatic patch surgery for mesothelioma. The rationale for the IPC was recurrent effusions and associated infections requiring repeat chest drains in the area of the postoperative hydropneumothorax.

The patient was asymptomatic and afebrile. The pleural fluid cultured *Klebsiella oxytoca* and *Enterococcus faecalis*. He was treated with the oral fluoroquinolone, ciprofloxacin and given advice and guidance on infection prevention strategies when handling the IPC and pleural drainage apparatus. A couple of weeks later it was decided to replace the IPC as an elective procedure due to the long duration of the IPC, a persistent low-grade purulent discharge, concerns about bacterial colonisation and history of a similar infection episode a year ago.

On the day of the procedure, the patient reported that the IPC had partially migrated out of the pleural cavity and the tunnelled subcutaneous tract due to loss of integrity of the IPC cuff and thus came out easily without any intervention. The IPC tip was sent for culture.

On clinical examination, blood pressure was 133/77 mm Hg, pulse rate was 77 beats per minute, respiratory rate was 18 breaths/min and the body temperature was 36.6°C with oxygen saturation 98% on room air. Lung auscultation revealed inspiratory crackles at the right lower lung zone. The laboratory test revealed white cell count (WCC) count $11.3 \times 10^9/L$, haemoglobin 108 g/L, platelets $430 \times 10^9/L$ and C reactive protein 71 mg/L. Bedside thoracic ultrasound scan was consistent with a hydropneumothorax.

The same day the patient underwent an uneventful IPC insertion under local anaesthesia and mild sedation with 1 mg of intravenous midazolam. Satisfactory IPC placement was confirmed on a post procedure chest radiograph (figure 1).

One and half hours postprocedure, the patient had a witnessed sudden drop in conscious level with associated seizure like activity. The patient was immediately assessed by the cardiac arrest team where blood pressure, heart rate and oxygen



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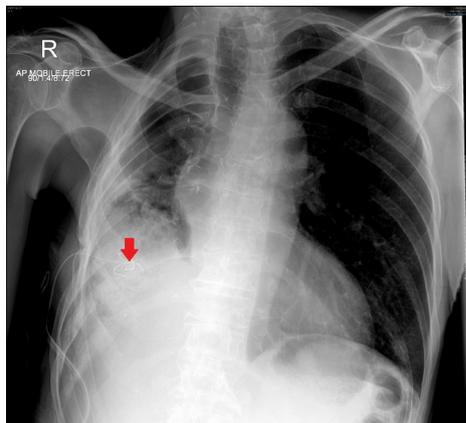


Figure 1 Postprocedure chest radiograph showing satisfactory IPC placement in chronic right hydropneumothorax (marked by red arrow). IPC, indwelling pleural catheter.

saturation were maintained within the reference range throughout. Glasgow Coma Scale (GCS) was 4 (eyes opening 1, verbal response 1 and motor response 2). General examination did not demonstrate any peripheral stigmata of systemic air embolism and no retinal air bubbles were seen in fundoscopic examination. An acute stroke was suspected and an urgent CT head was performed. Repeat blood tests revealed WCC $9.4 \times 10^9/L$, haemoglobin 107 g/L, platelet $408 \times 10^9/L$, blood urea 4.0 mmol/L, serum creatinine 73 $\mu\text{mol/L}$, estimated glomerular filtration rate >90 , C reactive protein 70 mg/L, alanine transaminase 9 iu/L. ECG was in sinus rhythm and no ischaemic changes were noted. CT head revealed multiple serpiginous pockets of air along the cerebral fissure, with features that were highly suggestive of an embolism and multiple wedge-shaped areas of infarction involving the cerebral hemispheres (figure 2). CT chest revealed satisfactory positioning of the IPC with no aetiology for the acute deterioration (figure 3). The patient was admitted to the intensive care unit for high flow oxygen therapy and head down ventilation.

OUTCOME AND FOLLOW-UP

Our patient's condition slowly deteriorated and his GCS was not improved. Following a discussion with the multidisciplinary team and family, a previous decision to manage him conservatively in

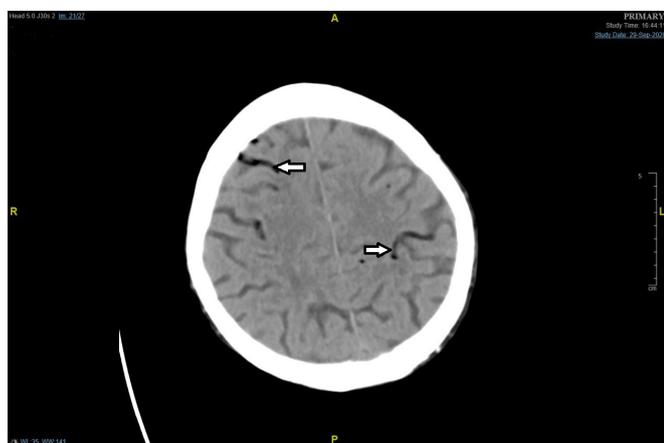


Figure 2 CT head showing multiple serpiginous pockets of air along the cerebral fissure (white arrows) suggestive of air embolism.



Figure 3 CT chest showing satisfactory placement of IPC (white arrow) and old diaphragmatic construction (black arrow). IPC, indwelling pleural catheter.

the event of any deterioration was respected and he was palliated. The patient died 6 days later.

DISCUSSION

Air embolism is defined as entry of air into the arterial or venous system. It may result from invasive procedures, trauma and barotrauma to the lungs.¹ Arterial air has direct access to the cerebral circulation and venous air is thought to be shunted through a patent foramen ovale (PFO). In the absence of a PFO, systemic venous air needs to traverse the pulmonary vasculature. Pulmonary arterioles and capillaries act as a physical barrier for various substances entering in to the arterial circulation, but some particles can still get through pulmonary capillary filter.² CAE of venous origin has occurred in the absence of intracardiac septal defects.³ Air directly entering into pulmonary veins, which drain into the left atrium can result in CAE without any of the mechanisms described above.

Pleural procedures can result in lung puncture and introduce gas bubbles into a pulmonary vein. Air bubbles can flow in when a needle tip enters a pulmonary vein while its base is opened to the atmosphere. The atmospheric pressure exceeds the pulmonary venous pressure during inspiration and air gets sucked into the pulmonary venous system. It may also occur when a needle penetrates both an air-containing space and a nearby pulmonary vein, causing a broncho-venous fistula. Coughing or a Valsalva manoeuvre may increase airway pressure and air enters the pulmonary vein.^{4,5}

One cannot be sure of the exact incidence of iatrogenic air embolism but it has been reported to be as low as 2.65/100 000 and is therefore not routinely consented for at the time of pleural procedures.⁶ The fatality rate in CAE has been reported to be as high as 21% although there are very few case reports of CAE in the literature.⁷ Knowledge of CAE is important for patients and operators given the large volume of commonly performed thoracic procedures in which CAE could occur such as percutaneous transthoracic needle lung biopsy, thoracentesis, chest drain insertion, intrapleural fibrinolytic therapy and pleural lavage.¹ CAE occurring after IPC insertion has not been reported.

At 7 years postdiagnosis, our patient had defied the odds in terms of his survival from mesothelioma. Mean 1-year survival

rate for mesothelioma in men aged above 75 years in UK is 29.8% and 5-year survival rate for the same group is 3%.⁸ Our patient's performance status was zero, despite his chronically distorted intra-thoracic anatomy due to previous mesothelioma surgery, a dehisced diaphragmatic patch and a loculated pneumothorax. The cause of CAE in our patient is not clear as there were no immediate complications at the time of the IPC procedure and subsequent radiology confirmed satisfactory position of the IPC with no trauma to the lung or any major blood vessels but it is likely that he had a pulmonary venous air embolism.

We hypothesise that the chronic anatomical and associated pathological changes may have resulted in inflammatory and friable intrathoracic tissue and (1) development of a breach or fistulae in a part of the collapsed lung and/or an adjacent vessel or (2) development of an arteriovenous malformation, either of which may have been aggravated by the replacement IPC and on Valsalva due to coughing lead to pressure changes and ingress of air into a branch pulmonary vein and subsequent embolisation with the catastrophe that ensued.

CAE is often a retrospective clinical diagnosis, based on a high suspicion of a patient having sudden onset and unexplained neurological symptoms following an invasive procedure as in our case. CT brain in the acute phase can demonstrate air bubbles in the cerebral arteries. In addition careful fundoscopic examination can reveal air bubbles in the retinal vasculature.^{4,9}

If CAE is suspected treatment should not be delayed while the diagnosis is established. Therapeutic measures includes administration of high-flow oxygen to reverse the cerebral ischaemia and placing the patient in a lateral decubitus position with head down or Trendelenburg position in order to facilitate retrograde flow of air bubbles from the cerebral arteries.¹⁰

Hyperbaric oxygen therapy (HBOT) is the most effective treatment for CAE and reduces the size of the air bubbles causing obstruction to the cerebral circulation. HBOT also increases the soluble oxygen availability for ischaemic tissues. According to Boyle's law, by increasing the pressure we can compress the size of air bubbles to release the obstruction and establish perfusion. Giving HBOT is also thought to reduce cerebral oedema by decreasing the permeability of the blood-brain barrier.⁹ Unmanaged pneumothorax is an absolute contraindication for HBOT but otherwise it is a simple and safe procedure, although not always readily available.¹¹

Early detection and thereafter prompt management is essential to reduce morbidity and mortality. In one series of 16 patients treated with hyperbaric oxygen, 50% patients had complete recovery, 31% had partial relief and 19% had no benefit with death in 12.5% patients.¹² In our case, the patient was given 100% oxygen therapy and not considered for mechanical ventilation or HBOT given his poor physiological reserve due to mesothelioma and the hydropneumothorax.

In conclusion, iatrogenic CAE is extremely rare in pleural interventions but it carries significant mortality and morbidity. A high degree of clinical suspicion is needed to diagnose CAE suggested by unexplained neurological symptoms following an invasive procedure. Prompt treatment of CAE is required. Giving 100% high flow oxygen therapy will increase the tissue oxygenation and reduce burden of air emboli by eliminating nitrogen. HBOT is the most effective treatment of CAE, but contraindication in unmanaged pneumothorax and accessibility may limit utilisation of HBOT. As pleural procedures are widely carried out in outpatient and inpatient settings, clinicians need to be competent in diagnosing and managing CAE.

Learning points

- ▶ Cerebral air embolism is a rare but important complication of pleural procedures, which carries significant mortality and morbidity.
- ▶ Diagnosis is based on a high degree of suspicion in a patient developing unexplained neurological symptoms following an invasive procedure.
- ▶ Administering high-flow oxygen and hyperbaric oxygen therapy with management in the Trendelenburg position is the main mode of treatment.
- ▶ Early detection and prompt treatment can reduce the morbidity and mortality.

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Contributors DMCJ Managed the patient and drafted the manuscript, RKP managed the patient as the leading respiratory consultant, supervised, reviewed and edited the manuscript. SA managed the patient as ITU consultant, reviewed and edited the manuscript. ID reviewed and edited images and provided radiological opinion.

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