Case Report

Massive Cerebral Air Embolism on Ante Mortem CT Head Following Pneumothorax in a Child with Pneumonia

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Summary

Cerebral air embolism (CAE) is a rarely reported complication of a common condition like pneumothorax, presenting with deterioration of sensorium and cardiovascular instability. We report a case of 3-year-old male who developed pneumothorax after positive pressure ventilation followed by deterioration of sensorium. CT head revealed massive CAE.

Key words: pneumothorax, positive pressure ventilation, cerebral air embolism.

Introduction

Introduction of air into venous or arterial circulation can cause cerebral air embolism (CAE), leading to severe neurological deficit or death [1].

CAE can occur secondary to iatrogenic complications (procedures like cardiopulmonary resuscitation, cardiovascular surgeries, neurosurgery, upper gastrointestinal endoscopy, arterial cannulation, central venous pressure line insertion, tooth extraction, positive pressure ventilation, etc.), trauma, certain recreational activities (scuba diving) or even air travel [2–4].

In cases with pneumonia, pneumothorax is a well-documented complication but occurrence of CAE following pneumothorax is quite rare. Most of the reports in literature of pneumothorax leading to CAE are due to scuba diving, air travel, trauma, blast injuries or artificially created pneumothorax. Fatal cerebral air embolism related to positive pressure ventilation is a rare complication [5, 6].

Case Report

A 3-year-old boy presented in pediatric emergency with cough and fever for 2 weeks. He had several episodes of complex partial seizures 3 days prior to admission and for 2 days breathing difficulty was experienced. On the day of admission, he developed altered sensorium. There was history of complex partial seizures 1 month back.

At admission, patient had respiratory rate of 42 min⁻¹ with signs of respiratory distress in the form of subcostal and intercostal retractions; heart rate and blood pressure were within normal limits. Chest auscultation revealed crepitations bilaterally. Glasgow coma scale (GCS) score was E3M4V3. There was no cyanosis and oxygen saturation on pulse oximeter was more than 95% on room air. Provisional diagnosis of pneumonia with encephalopathy was kept. Tubercular meningitis and sepsis were thought as the probable differential diagnoses.

On investigations, hemoglobin was 10.2 g dl⁻¹, total leukocyte count 28.8 x 10⁹ / l with polymorphonuclear cells of 90% and lymphocytes 7%, and platelet count was 120 x 10⁹ / l. Renal function tests were within normal limits. Arterial blood gas analysis showed compensated metabolic acidosis with normal oxygenation. Chest X-ray (CXR) was suggestive of bilateral bronchopneumonia.

After 5 h of admission, child threw a generalized tonic clonic seizure and developed signs of raised intracranial pressure in the form of decerebrate posturing. Elective intubation was done and hyperventilation was given for half an hour. Posturing subsided after this. Patient was put on T-piece ventilation. Broad spectrum antibiotics and anticonvulsants were started. After 12 h, he was sent for computed tomography (CT) scan of the head, where while coming out of CT room he was found in cardiorespiratory arrest, and there was massive subcutaneous emphysema all over his chest wall and neck. He was resuscitated and suspecting pneumothorax, needle drainage was done. CXR showed bilateral drained pneumothorax and streak of air in
mediastinum (Fig. 1); CT head showed massive amount of air in all major arteries of the brain, suggestive of massive generalized cerebral arterial air embolism (Fig. 2). Patient was put on mechanical ventilation. Hyperbaric oxygen therapy was not available. For 12 h, he continued to have low GCS (E1V1M1) and dilated–fixed pupils, after which there was another cardiac arrest of which he could not be revived.

**Discussion**

CAE can be cerebral arterial air embolism (CAAE) or cerebral venous air embolism (CVAE) [7]. In our case, it was CAAE.

CAAE may occur as a result of introduction of air into systemic arterial or venous circulation [8]. Air can easily enter cerebral arterial circulation from pulmonary venous circulation through a bronchovenous fistula and/or damaged pulmonary vessels (which might be because of barotrauma or lung disease) [9]. In our patient, barotrauma occurred in diseased lung and so above mechanism is the probable cause of CAAE in our patient.

Further, air injected via peripheral venous circulation can cause CAAE via paradoxical embolism (via patent foramen ovale) or by overwhelming the natural mechanisms of lung to prevent arterial gas embolism [1, 4].

In newborns, CAE following ventilation is a known entity, because damaged pulmonary vascular integrity due to relatively higher pulmonary inflation pressure in respiratory distress syndrome is supposed to facilitate the entry of air into systemic circulation,
including the cerebral circulation [10]. In children, apart from newborns, CAE associated with ventilation is not a much reported entity [6]. Such massive antemortem pneumoencephalogram is also rarely seen.

The pneumothorax in our patient might be spontaneous or consequent to barotrauma caused by positive pressure ventilation in already diseased lung (underlying pneumonia). Although patient did not show any sign of increasing respiratory distress for nearly 12 h after hyperventilation, it is possible that either already existing pneumothorax caused by pressure ventilation was increasing in size during this time or patient developed sudden spontaneous pneumothorax in the CT room which resulted in CAE.

Neurological manifestations in systemic air embolism can be due to cardiovascular collapse causing reduced cardiac output, leading to cerebral hypoperfusion or cerebral air embolism [3].

The neurological symptoms associated with CAE may range from transient neurological symptoms to sudden unconsciousness and death. According to Murphy et al. [1], neurological symptoms may include focal motor deficit, alteration in sensorium, visual and sensory deficits. Our patient had deterioration in sensorium, which perhaps was because of massive CAE and cardiovascular collapse. Massive bilateral pneumothorax and CAE might have contributed for the sudden cardiorespiratory arrest.

To conclude, CAE should be suspected in all cases of pneumothorax with sudden deterioration of sensorium and/or development of neurological signs, so that proper management can be instituted at the earliest.

References