

Cerebral Air Embolism Secondary to Lung Laceration

Ashok Kumar Singh, Jayant Verma, Surendra Kumar

Department of Pulmonary and Critical Care Medicine, Regency Hospital, Kanpur, Uttar Pradesh, India

Abstract

Cerebral air embolism is a rare clinical entity in day-to-day practice. The introduction of air into the venous or the arterial system can cause cerebral air embolism leading to severe neurological deficits. The common causes reported in the literature are iatrogenic; it can be caused by positive pressure maneuvers performed during cardiac resuscitation, lung biopsy, and the placement of venous catheters in the presence of a patent foramen ovale. We report a case of cerebral air embolism which has occurred secondary to lung laceration. The patient underwent intercostal drainage for hydro-pneumothorax and developed forceful cough and suddenly changed in consciousness. Air embolism was diagnosed by computed tomography brain and was managed by high-concentration oxygen therapy and other supportive measures and is being discharged in satisfactory condition.

Keywords: Air embolism, lung laceration, pneumocephalus

INTRODUCTION

Air embolism is a rare clinical entity, as has been said by Morgagni in 1769, who described the consequences of pulmonary overinflation leading to arterial gas embolism. Its increasing frequency is found in medicine due to indwelling central venous catheters, use of positive pressure ventilations with high level of positive end-expiratory pressure, bronchoscopic procedures, and in various forms of cerebral and thoracic trauma. Cerebral air embolism secondary to isolated chest injury in the form of lung laceration is not reported in the literature. We report a case of cerebral air embolism due to isolated chest trauma in the form of lung laceration. He was managed successfully by oxygen therapy, ventilation, and other supportive measures.

CASE REPORT

A 26-year-old male patient had road traffic accident. He was immediately transferred to the hospital, where he was assessed by an emergency medical officer who found that he was having difficulty in breathing, conscious, and following command, the findings observed were SpO₂ 85% with O₂ 4 L/mt by face mask, heart rate 124/mt, and RR 26/mt. There was significant subcutaneous emphysema all over the chest and face. He was not moving his right upper limb and there was swelling over the right scapula and shoulder.

Movement on the right side of the chest was significantly reduced and breath sound was absent. Abdomen was soft and there was no evidence of intra-abdominal trauma or bleed. He was resuscitated by intravenous fluid, oxygen therapy, and other conservative measures. Routine investigations were sent; chest X-ray revealed hydropneumothorax. Ultrasound abdomen suggested collection in the right pleural space and no evidence of abdominal injury. It was confirmed by computed tomography (CT) thorax which revealed large lung parenchymal tear, hydropneumothorax, multiple rib fracture on the right side, and associated subcutaneous emphysema [Figure 1]. Immediately, chest tube was placed on the right side and about 1200 ml blood came out. Gradually, consciousness level started deteriorating and he had an episode of convulsion, after which he was intubated and put on ventilator. CT scan brain was done to find any cause related to change in consciousness and convulsion. CT scan brain revealed pneumocephalus in the multiple sites of cerebrum [Figure 2], which was suggestive of brain air embolism due to systemic embolization of air secondary to lung tear as there was no evidence of direct brain injury. Two-dimensional echocardiogram was done and

Address for correspondence: Dr. Ashok Kumar Singh,
Regency Hospital, A 2, Sarvodaya Nagar, Kanpur - 208 005,
Uttar Pradesh, India.
E-mail: dr_ashok_rhl@yahoo.com

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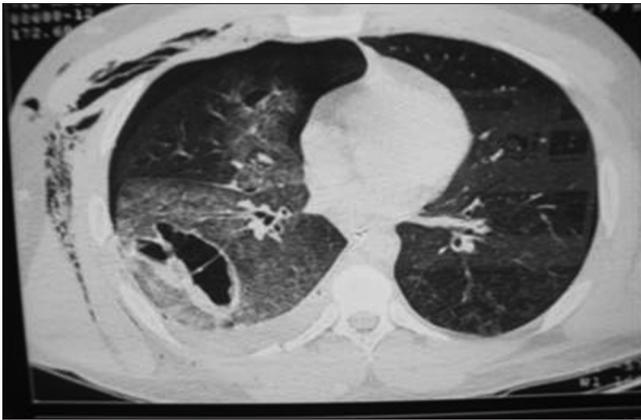


Figure 1: Computed tomography scan thorax revealing lung laceration with hydropneumothorax and subcutaneous emphysema on the right side

it ruled out intracardiac shunt. He was managed by ventilation with high FiO_2 , anticonvulsant drugs, antibiotics, and other supportive measures. After 72 h of ventilation, CT scan of brain was repeated and it was found that all hypodense lesions due to air embolism disappeared and were replaced by multiple infarctions. The patient remained on ventilator for 2 weeks with the tracheotomy tube and then gradually weaned off from ventilator. His chest tube was removed and was discharged in the 4th week with complete recovery.

DISCUSSION

Air embolism refers to entry of air into vascular structure. It is mainly due to iatrogenic clinical entity responsible for serious morbidity and mortality. This is the most serious problem in diving medicine.^[1]

There are two types of air embolism: venous and arterial. Venous embolism is more common as compared to arterial embolism. In venous embolism, air bubbles enter the systemic venous circulation^[2] and then were transported to lung via pulmonary arteries causing gas exchange abnormality, cardiac arrhythmias, right heart strain, acute cardiac failure, and finally death. Predisposition that allows entry of air into the venous system includes noncollapsed veins and presence of subatmospheric pressure in these blood vessels. This condition also occurs when surgical field is above the heart like neurosurgery done in sitting posture.^[3] Another condition is hemodialysis catheters where air also can enter into the veins in myometrium in peripartum period.^[4]

In arterial air embolism, air enters into pulmonary veins or directly into the systemic circulation. The mechanism includes the overexpansion of lung through decompression barotraumas in diving or pulmonary barotraumas in ventilator therapy in critical care patients. Arterial embolism can also occur as paradoxical embolism and in cardiac surgery.^[5] After that, air reaches to arterial circulation leading to embolization of air in various parts of the body and manifestation occurs accordingly depending on the site of maximum insults. Common clinical presentations are breathlessness, chest

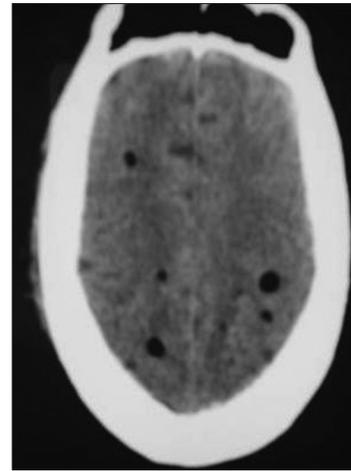


Figure 2: Computed tomography scan brain revealing evidence of pneumocephalus

pain, hypotension, and change in consciousness. The most serious consequences result from coronary and cerebral air embolism. This condition is termed as cerebral arterial gas embolism, as in our case embolization has occurred due to chest trauma leading to cerebral embolization. Embolic air also affects functions of other body organs such as muscle and kidney,^[6] leading to rhabdomyolysis and renal failure. Cerebral vessels show circulatory response in the form of inflammatory reaction to air and perfusion defect to distal part of obstruction leading to ischemic changes in the form of cerebral infarction.^[7] The same clinical presentation was present in our case.

Treatment consists of early clinical suspicion and use of 100% oxygen therapy or hyperbaric oxygen therapy.^[8] Hyperbaric oxygen therapy is the first-line therapy. In comatose patients, airway protection is mandatory and sometimes ventilator support is also needed depending on patient's conditions. Anticonvulsant drugs may be needed because cerebral embolism may be associated with generalized convulsions. The use of corticosteroids remains controversial.

CONCLUSION

This case report highlights the need of higher degree of suspicion in early recognition of cerebral air embolism in a chest trauma patient who developed sudden change in central nervous system status without any obvious evidence of head injury. If this entity is detected early, then appropriate and adequate treatment can be offered to the patient in need.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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