CASE REPORT

Systemic arterial air embolism: positive pressure ventilation can be fatal in a patient with blunt trauma

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SUMMARY

Systemic air embolism is a potentially fatal condition. Although venous embolism is commonly reported after deep sea diving or neurosurgical procedures, arterial embolism is rare. It usually occurs because of lung trauma after biopsy or lung resection but can rarely affect patients of blunt or penetrating trauma to chest managed on positive pressure ventilation. We report a case of road traffic accident with head injury, with normal primary survey of chest and abdomen who developed fatal systemic arterial air embolism immediately after intubation. Postmortem CT scan revealed huge amounts of air in left side of the heart, ascending aorta, arch of aorta, bilateral internal carotids and all right-sided intracranial arteries. In emergency departments of non-specialised centres, such complications are universally fatal. Thus, extreme caution needs to be exercised while managing patients of blunt trauma on mechanical ventilation even if the chest and abdominal examinations are normal.

Figure 1 Non-contrast CT head showing epidural haematoma in left frontotemporoparietal region with midline shift and free air in anterior and middle cerebral arteries.

BACKGROUND

Systemic arterial air embolism (SAAE) occasionally affects the patients with massive or clinically apparent blunt or penetrating injuries to the chest. However, SAAE can rarely affect a patient with minimal injuries to the chest managed on mechanical ventilation for head injury. Sudden hypotension, bradycardia and hypoxia in such a patient should arouse the suspicion of arterial air embolism. Immediate transoesophageal echocardiography is diagnostic and hyperbaric oxygen therapy can be life saving.

CASE PRESENTATION

A 19-year-old male pedestrian met with a road traffic accident and was brought to emergency department in an unconscious state. His blood pressure was 126/94 mm Hg, pulse 122/min, respiratory rate was 22/min and Glasgow Coma Scale was E1M4V1 with pupils semidilated bilaterally. On primary survey, there was suspicion of head injury with laceration and haematoma over right parietal region of scalp. Chest examination revealed negative chest compression test, no subcutaneous emphysema and breath sounds were normal bilaterally. Abdominal examination was normal. A closed fracture of left femur was also noted and with a provisional diagnosis of road traffic accident with a head injury, with left femur fracture, the patient was resuscitated. Gradually the patient became breathless and pO₂ started falling. He was intubated and put on artificial manual breathing unit (AMBU)

ventilation and was transferred to intensive care unit (ICU). In ICU, patient was started on mechanical ventilation, but within minutes, there was severe hypotension and hypoxia. Despite resuscitation and ionotropic support, hypotension progressed,

Figure 2 Non-contrast CT head showing epidural haematoma in left frontotemporoparietal region with midline shift and free air in anterior and middle cerebral arteries.

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Figure 3 Non-contrast CT chest showing free air in left ventricle, left atrium and right ventricle.

cardiopulmonary resuscitation had to be started and patient expired within 20 min after intubation. Relatives of the patient did not give consent for a formal autopsy, thus to elicit the cause of death, postmortem CT was planned and was done within 20 min after death with the consent of relatives.

INVESTIGATIONS

Postmortem non-contrast CT scan of head, chest and abdomen were carried out. Non-contrast CT NCCT head (figures 1 and 2) revealed a left-sided extradural haemorrhage in frontoparietotemporal region with brain parenchymal oedema and midline shift towards right side. Air was noted in right anterior and middle cerebral arteries and their branches. A right-sided scalp haematoma was also noted. NCCT scan of the chest revealed striking amounts of air in left side of the heart (atrium and ventricle) (figure 3) along with small amounts of air in the right ventricle and the right pulmonary vein (figure 4), arch of aorta and its branches (right brachiocephalic, left common carotid and left subclavian arteries) (figure 5), along with bilateral internal carotid and right vertebral artery also contained air

column (figure 6). There was a small contusion in the middle lobe of right lung (figure 4). Endotracheal tube was visible in the right bronchus (figure 7). NCCT scan of the abdomen was normal (figure 8). Thus, air was confined to aforementioned arterial system and superior vena cava, inferior vena cava and descending aorta were normally filled with blood.

Based on postmortem CT scan, a diagnosis of SAAE was considered to be the cause of death. Such unprecedented amounts of air in the arterial system of our patient must have caused massive cardiac ischaemia resulting in rapidly fatal cardiogenic shock. However, NCCT showed only a small region (as compared to free air in arterial system) of contusion in the right lung. This can be explained by the fact that inadvertent placement of endotracheal tube in right bronchus (corresponding to the side of lung contusion) and AMBU bag ventilation may have generated excessive positive pressure, causing massive air

Figure 4 Non-contrast CT chest showing right lung contusion and free air in right pulmonary vein.

Figure 6 Non-contrast CT neck showing free air in bilateral internal carotids and right vertebral artery.

Figure 5 Non-contrast CT chest showing free air in arch of aorta and its branches.

Figure 7 Non-contrast CT chest showing endotracheal tube in right bronchus.

embolism through a bronchopulmonary vein fistula that may have formed in the contused lung.

OUTCOME AND FOLLOW-UP

Patient expired 20 min after intubation.

DISCUSSION

SAAE is potentially life-threatening condition usually seen after pulmonary procedures (biopsy or resection) or penetrating trauma to lung but rarely may be associated with blunt trauma to the chest,¹ as we just reported. Even a minor injury to chest along with positive pressure ventilation is sufficient to cause fatal air embolism. An increased bronchopulmonary pressure gradient, due to positive pressure ventilation, favours transmission of air from injured bronchus to pulmonary vein.²

Even small amounts of air (0.5–1 ml) entering the pulmonary vein may be fatal.³ Thus, strong clinical suspicion in patients on positive pressure mechanical ventilation is corner stone of management of these cases. Small amounts of air may cause neurological or cardiac symptoms depending on the arteries affected, but massive embolism is usually fatal as a result of cardiovascular collapse.

Hyperbaric oxygen therapy is considered as the treatment of choice for arterial air embolism.⁴ But such equipment is usually not readily available and patient might not be haemodynamically stable for such a procedure. Isolated ventilation of normal lung or needle venting of air from left ventricle may also be tried.⁵ Immediate thoracotomy and cross-clamping of

pulmonary vasculature of affected side has been reported to prevent further embolisation.⁶ But all these therapies may not be readily available, thus prevention is deemed necessary in all cases. Besides monitoring, it is necessary to avoid excessive positive pressure ventilation and positive end expiratory pressure by low tidal volume and use of high-frequency jet ventilation.⁷ Transesophageal echocardiography may be used for intraoperative monitoring of high-risk cases.⁸

Thus, massive SAAE may result from minimal chest trauma and positive pressure ventilation, and if appropriate precautions are not observed, it may be universally fatal.

Learning points

- ▶ Systemic arterial air embolism can rarely affect patients with minimal injuries to chest managed on mechanical ventilation for head injury.
- ▶ Even small amounts of air is fatal.
- ▶ Sudden hypoxia, bradycardia and hypotension in a patient of blunt trauma on mechanical ventilation should arouse the suspicion.
- ▶ Universally fatal if not identified rapidly and treated.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Worth ER, Burton RJ Jr, Landreneau RJ, *et al.* Left atrial air embolism during intraoperative needle biopsy of a deep pulmonary lesion. *Anaesthesiology* 1990;73:342–5.
- 2 Graham JM, Beal AC, Mattox KL, *et al.* Systemic air embolism following penetrating trauma to the lung. *Chest* 1979;72:449.
- 3 Goldfarb B, Bahnson HT. Early and late effects on heart of small amounts of air in the coronary circulation. *J Thoracic Cardiovasc Surg* 1980;80:708–17.
- 4 El-Halabi DA, Bortros AR, Nowacka ED. Massive air embolism following a blunt chest injury. *Ann Saudi Med* 1998;24:425.
- 5 Hanowell LH. Perioperative management of thoracoabdominal trauma. In: Grande CM ed. *Textbook of trauma, anesthesia and critical care*. St Louis, MO: Mosby Year Book Inc, 1993:559–82.
- 6 Yee ES, Verrier ED, Thomas AN. Management of air embolism in blunt and penetrating trauma. *J Thoracic Cardiovasc Surg* 1983;85:661–8.
- 7 Shackdard SR. Blunt trauma chest, the intensivists perspective. *J Intensive Care Med* 1986;1:125.
- 8 Tingleff J, Joyce FS, Pettersson G. Intraoperative echocardiography study of air embolism during cardiac operation. *Ann Thorac Surg* 1995;60:673–7.

Figure 8 Non-contrast CT abdomen reveals normal descending aorta and superior vena cava.

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