Air Embolism - A Case Series and Review

S. SVIRI*, W. P. D. WOODS*, P. V. VAN HEERDEN*†‡
*Department of Intensive Care, Sir Charles Gairdner Hospital, Nedlands, WESTERN AUSTRALIA
†Medical Intensive Care Unit, Hadassah Hospital, Jerusalem, ISRAEL
‡School of Medicine and Pharmacology, University of Western Australia, Crawley, WESTERN AUSTRALIA

ABSTRACT
Venous or arterial air embolism may be a life threatening event. The condition is seen in many fields of medicine, including intensive care. We present a series of three cases of air embolism encountered in the intensive care unit, which demonstrate different pathophysologies for air embolism in critically ill patients. We also review the literature with respect to aetiology, incidence, pathophysiology, diagnosis and treatment options for venous and arterial embolism. (Critical Care and Resuscitation 2004; 6: 271-276)

Key words: Air embolism, cardiopulmonary resuscitation, aetiology, management

Air embolism can be a serious and even fatal event in the intensive care unit (ICU). The ICU is a setting where most patients have central venous catheters or undergo procedures which may put them at risk.¹ Patients may also present with post operative air embolism as a complication.¹² A high level of vigilance should be maintained, to allow for prompt diagnosis and rapid treatment of this condition.

We present three cases of air embolism in the ICU and review the literature regarding aetiology, incidence, pathophysiology and treatment of air embolism.

CASE REPORTS
Patient 1. A 45-year-old man with a past history of intravenous drug abuse, heavy ethanol intake and positive serology for Hepatitis C virus, was transferred to our ICU from another hospital. His presenting problems were sepsis and cardiorespiratory failure.

He presented initially to the peripheral hospital complaining of transient right sided weakness. On examination a right molar tooth abscess was found and a computed tomography (CT) scan of his cranium showed an old left frontal infarct. The patient underwent molar extraction in the emergency department as he refused admission.

The patient returned to the peripheral hospital three days later with a high fever, headache, rigors and abdominal pain. Initial examination failed to reveal a source of his sepsis. He was admitted, and intravenous amoxicillin and flucloxacillin were commenced. Subsequently, the patient became tachypnoeic and developed septic shock with a blood pressure of 80/60 mmHg, temperature of 39ºC and arterial oxygen saturation (SaO₂) of 90% (breathing oxygen at 10 L/min through a non-rebreathing mask). The antibiotics were changed to ticarcillin and gentamicin. On further questioning, the patient claimed he had undergone rectal trauma with a broom handle, prior to his first presentation. As his condition continued to deteriorate he was transferred to our hospital.

In the emergency department he was agitated with a blood pressure 116/80 mmHg, heart rate 120 beats per min, temperature 37.2ºC, tachypnoeic and hypoxic with bibasal crepitations heard on auscultation. There was no cardiac murmur. His abdomen was soft, without organomegaly and altered blood was found on rectal examination. Intravenous ‘track’ marks were noted between his toes. Neurological examination was unremarkable. A chest X-ray demonstrated bilateral alveolar infiltrates.

Due to progressive respiratory distress the patient was intubated and mechanically ventilated. A cranial CT was performed which showed an acute left cerebellar infarct and an old left frontal infarct. An abdominal CT showed gas in the middle hepatic vein, an absent left kidney, with compensatory hypertrophy of the right kidney, and a normal peri-rectal area.

Correspondence to: Dr. P. van Heerden, Department of Intensive Care, Sir Charles Gairdner Hospital, Nedlands, Western Australia 6009 (e-mail: peter.vanheerden@health.wa.gov.au)
The patient was transferred to the ICU, where he was sedated and ventilated with an inspired oxygen concentration of 70%. He required intravenous adrenaline to maintain his blood pressure. He remained oliguric with rising plasma urea and creatinine levels.

A bedside sigmoidoscopy was performed to exclude a rectal or sigmoid perforation which revealed blood in the lower rectum, although the mucosa, up to 15 cm, appeared to be normal. Shortly after the sigmoidoscopy the patient became cyanosed, bradycardic and subsequently became asystolic. Cardiopulmonary resuscitation was commenced and the patient was given adrenaline, atropine, isoprenaline, intravenous colloids and placed in the head down, right side up position, as an air embolism from the sigmoidoscopy was suspected. Aspiration of blood from the central venous catheter did not reveal air. External pacing was initiated but no pulse could be felt. He subsequently died.

A chest x-ray was taken during the resuscitation which showed a large amount of air within the cardiac silhouette (figure 1). A post mortem chest CT demonstrated a large amount of air in the right ventricular outflow tract (figure 2) and a post mortem abdominal CT showed extensive air within the hepatic veins.

The post mortem examination revealed an intestinal perforation 15 cm from the ano-rectal margin extending 5 cm along the circumference of the bowel wall. The perforation was adjacent to iliac veins and probably provided a passage for air to travel into the inferior vena cava at the time of the sigmoidoscopy. Within the aorta and ventricles frothy blood was noted. The lungs were consolidated, consistent with acute respiratory distress syndrome (ARDS). A 15 mm right upper lobe abscess was noted. A recent haemorrhagic infarction in the left cerebellum and multiple small cerebral haemorrhages were seen and small air bubbles noted within the cerebral vessels.

Patient 2. An 85-year-old man underwent urgent femoral-popliteal bypass surgery at another hospital to manage a left gangrenous foot. His past history included severe peripheral vascular disease, chronic ischaemic cardiac disease with congestive heart failure and type 2 diabetes mellitus. Post operatively he was transferred to the ICU, where he was found to be hypotensive.

As he remained hypotensive in spite of crystalloid infusions, Haemaccell® (Hoechst Marion Roussel, Lane Cove, NSW) was prescribed and a needle was inserted into the top of the bottle to increase the administration rate. As the rate of fluid flow was still deemed to be inadequate, a pressure bag was placed on the bottle and the needle hole sealed with tape. Several minutes later, the patient became profoundly shocked and bradycardic. An air embolism was suspected and CPR was initiated. As there was no central venous access, air could not be aspirated. Resuscitation attempts failed and the patient died. A post-mortem chest X-ray revealed a large amount of intra-cardiac air (figure 3) and at post mortem examination, air was found in the cerebral vessels as well as the cardiac chambers.

Patient 3. A 26-year-old female was admitted to hospital for biopsy of a recurrent pineal lesion and underwent posterior fossa craniotomy in the sitting position. During the procedure the anaesthetist noted three episodes of profound hypotension and decreased end tidal carbon dioxide concentrations. For each episode the operative field was flooded with saline solution, the patient lowered to a supine position and air was aspirated from a central venous catheter.
Figure 3 Lateral chest X-ray demonstrating intracardiac air.

Postoperatively the patient was transferred to the ICU, where she was hypotensive, tachycardic, confused and hypoxic. A chest x-ray was consistent with pulmonary oedema and an ECG showed diffuse ST and T wave changes. She was reintubated, ventilated with 100% oxygen, positioned in the left lateral decubitus position and intravenous adrenaline was infused. A cranial CT performed the following morning showed extensive bilateral cortical and subcortical infarcts with diffuse cerebral oedema. Her best neurological response a week later consisted of spontaneous eye opening but no response to voice or pain. A transthoracic echocardiogram demonstrated normal ventricular function and no evidence of an atrioseptal or ventriculoseptal defect.

One month later good cognitive function and memory returned. An MRI of the brain before discharge demonstrated a small residual pineal tumour and an extensive anterior and posterior watershed ischaemic injury in keeping with global cerebral hypoperfusion. Four months later she was wheelchair bound but was communicating adequately with her family.

DISCUSSION and REVIEW

Air embolism, or the entry of air into the circulation, is mostly an iatrogenic problem but may also occur with trauma.1,2 Air commonly enters the venous system but may also enter the arterial system. The disorder may include gases other than air including carbon dioxide and nitrous oxide, which are used in medical procedures, and nitrogen, usually found in diving accidents.1,3 Air embolism may have disastrous cardiac, pulmonary or neurological effects and is associated with a high morbidity and mortality.1

The entry of air into the blood stream requires a pressure gradient favouring the passage of gas into the blood vessel.2 This occurs when venous pressure is negative relative to atmospheric pressure (e.g. case 3) or when air is forced under pressure directly or indirectly into the bloodstream1,2 (e.g. cases 1 and 2).

Aetiology

The causes of air embolism include entry of air through intravascular catheters such as peripheral and central venous canulae, pulmonary artery catheters, haemodialysis catheters, pressurised infusion systems and long term central catheters such as Hickman catheters.1,4

Entry of air during surgery may be common, especially during neurosurgery, laparoscopic surgery and cardiothoracic surgery.1,3,5,6 The risk of air embolism is particularly high in neurological procedures performed in patients in the sitting position. The incidence of air embolism is 10% for cervical laminectomies and up to 80% in posterior fossa surgery.1 This is due to the pressure gradient between ambient air and the exposed venous circulation in the head during the sitting position where the venous pressure is negative with respect to the heart. In the ICU, special care must be taken to avoid air embolism via intravenous and arterial catheters,4 pulmonary artery catheters and intra-aortic balloon catheters. During positive pressure ventilation, barotrauma has also been described as causing air embolism.1 Insufflation of air into a body cavity during endoscopy can also cause air to enter the circulation (e.g. case 1).

Radiological procedures such as angiography and during the injection of air as a contrast agent have also been implicated in venous and arterial air embolism,1 as have cardiac catheterisations6 and cardiac ablation procedures.9 Other high risk procedures include radical neck surgery, obstetric and gynaecological procedures including Cesarean sections and laparoscopic surgery,10 vascular surgery (for example endarterectomies) and orthopaedic surgery (such as hip replacement, spine surgery and arthroscopy).1,5

Cases of air embolism have also been described in trauma such as blunt and penetrating chest trauma, abdominal trauma, neck and craniofacial injuries and decompression injury1. Air embolism has also been described as a result of orogenital sexual activity during pregnancy.

Pathophysiology

In 1947 Durant studied air embolism in dogs and reported that the most important factors determining mortality were the amount of air entering the bloodstream, the speed with which it enters and the body position at the time of embolism.11 Rapid entry of air into the circulation may cause severe haemodynamic instability. A fatal dose is considered to be 300 – 500 mL of air at a rate of 100 mL/sec; a rate which is
possible with a 14 gauge needle and a pressure gradient of only 5 cm H$_2$O between air and venous blood.\textsuperscript{2,5} In the critically ill, unstable patient, smaller volumes of air may also be fatal.

When a large bolus of air rapidly enters the venous system, it causes an air lock in the right atrium and ventricle causing right ventricular outflow obstruction and death. With slow entry of air into the right ventricle, obstruction occurs at the level of the pulmonary vasculature, causing vasoconstriction and pulmonary hypertension. Small amounts of air may be tolerated, as air is absorbed from the circulation, but large amounts of air may result in right ventricular strain, decreased cardiac output, shock and death.\textsuperscript{1,3}

**Signs and symptoms**

Cardiac manifestations of air embolism include chest pain and light-headedness.\textsuperscript{2} The typical “mill-wheel” murmur that has a splashing sound may be heard with a precordial or oesophageal stethoscope, but is generally a late sign. Electrocardiographic changes include nonspecific ST and T wave changes and evidence of right ventricular strain.\textsuperscript{2}

The patient may also develop bradycardrhythmias or tachyarrhythmias. Pulmonary artery pressures are decreased in a “bolus” air embolus and increased in “slow” air embolism.\textsuperscript{2} Central venous pressures are increased due to right ventricular failure and the patient may become haemodynamically compromised. In severe cases, asystole or pulseless electrical activity may occur leading to death.

Respiratory manifestations include dyspnoea or tachypnoea and the classic “gasp” reflex due to acute hypoxaemia.\textsuperscript{2} Neutrophils release cytokines in the lung causing tissue damage and increased permeability that is similar to ARDS.\textsuperscript{2} Patients may develop a deterioration in lung function, with reduced compliance, increased dead space and shunting leading to hypoxaemia and hypercarbia.\textsuperscript{12}

The term “paradoxical embolism” is used to describe situations in which gas crosses into the left atrium through a patent foramen ovale or atrial septal defect, thus causing air embolism within the systemic circulation.\textsuperscript{13} This may cause cardiac and neurological manifestations, although neurological deficits may develop as a result of prolonged hypoxaemia and shock as well as direct air embolism. There have been several cases of systemic air embolism due to venous embolism where a cardiac defect could not be found. It is presumed that air can also enter the systemic circulation through physiological pulmonary right to left shunts or due to passage of air into the left atrium via the pulmonary veins.\textsuperscript{14}

**Central venous catheters**

The frequency of venous air embolism reported with central catheters has ranged from 1 in 47 to 1 in 3000 catheters.\textsuperscript{2} The mortality associated with this event reaches 30%. As air may enter whenever venous pressure decreases below ambient air pressure, the risk is increased during deep inspiration, hypovolaemia, strained respirations and when the patient is in the upright position. Most cases occur during catheter manipulation, disconnection, hub fracture or removal, and not only during central venous catheter insertion.\textsuperscript{15,16} Catheter removal should be performed with the patient supine or in the Trendelenburg position, while holding his or her breath at the end of inspiration or during a Valsalva maneuver.\textsuperscript{17} In mechanically ventilated patients the catheter should be removed at end inspiration, when the pressure in the chest is more than atmospheric pressure. Direct pressure should be applied on the catheter site for several minutes after bleeding has stopped\textsuperscript{18} and an occlusive dressing should be placed over the exit site.\textsuperscript{19}

**Diagnosis**

The diagnosis of venous air embolism relies on a high index of suspicion in high risk cases (e.g., neurosurgical procedures in the sitting position, etc). Sudden hypotension, hypoxia and bradycardia, aspiration of air bubbles through a central catheter and characteristic cardiac sounds all aid in the diagnosis. The precordial stethoscope may help in detecting a “mill-wheel” murmur, but this often difficult to hear and may develop late in the patient’s condition.\textsuperscript{2,5} End-tidal carbon dioxide monitoring is convenient and practical and with air embolism it decreases as a result of increased dead space caused by an obstruction of pulmonary arteries. Precordial Doppler ultrasound is a sensitive and noninvasive and may detect small amounts of air by converting changes in blood density into sound.\textsuperscript{2,5} Transcranial Doppler has also been used to detect cerebral microemboli during endovascular procedures.\textsuperscript{8}

The most sensitive monitoring device is the transoesophageal echocardiograph which may detect as little as 0.02 mL/kg of air and air bubbles as small as 5 - 10 microns. Transoesophageal echocardiography (TOE) may also identify the cardiac location of air bubbles and may demonstrate the passage of air into the left atrium.\textsuperscript{2,5,14,20}

**Treatment**

The source of air must be identified and further vascular entry of air stopped. Operative fields should be flushed with saline, central lines secured or removed etc.\textsuperscript{5} Air may be evacuated from the right atrium or
ventricle via a central venous catheter or a pulmonary artery catheter with up to 50% of air aspirated depending on catheter placement and patient positioning.\textsuperscript{2,5} It has been shown that optimal placement of the catheter tip for aspiration of air is 2 cm below the junction of the superior vena cava and right atrium.\textsuperscript{5}

Supportive therapy for the patient with air embolism includes intravascular volume to increase venous pressures and venous return, intravenous catecholamines and mechanical ventilation\textsuperscript{1,5,21}. Cardiopulmonary resuscitation may be used as a means of maintaining cardiac output but may also serve to break large air bubbles into smaller ones and force air out of the right ventricle into the pulmonary vessels, thus improving cardiac output.\textsuperscript{5} Oxygenation is important, not only to increase haemoglobin saturation and improve peripheral tissue oxygenation, but also to reduce bubble size, as 100% oxygen will reduce the nitrogen content and size of the air bubble.\textsuperscript{1,5}

Patient positioning has been shown to be important. The patient should be placed in the left lateral decubitus position with the head tilted downwards as this will place the right ventricular outflow tract below the right ventricular cavity,\textsuperscript{2,5} and allow air to migrate up and out of the ventricular outflow tract. If the patient requires CPR, then he or she should be placed in a supine and head down position.\textsuperscript{2,5} During anaesthesia it is important to discontinue nitrous oxide as this will diffuse into air bubbles and enlarges them.\textsuperscript{5}

After the resuscitation attempt, microbubbles may remain in the circulation for a further 10 - 30 minutes until absorbed. Prolonged resuscitation and hypoxaemia, or the development of systemic air embolism may have adverse long term effects.\textsuperscript{2}

\textbf{Arterial air embolism}

This may cause severe morbidity and mortality, and can be due to direct entry of air into the arterial circulation or paradoxical venous embolism.\textsuperscript{1} During arterial air embolism there is not only reduced perfusion distal to the obstruction but also an inflammatory response resulting in vasogenic oedema and surrounding cellular injury\textsuperscript{1} (e.g. neuronal injury in cerebral air embolism). Cardiac effects of arterial embolism include ischaemia, infarction, heart failure and dysrhythmias. Neurological manifestations include a wide range of clinical features, including confusion, loss of consciousness, coma, convulsion, motor deficits and impairment of vision.\textsuperscript{1,5}

Treatment of systemic embolism is largely supportive, including mechanical ventilation, control of seizures and fluid replacement. The use of hyperbaric oxygen is advocated by some as the treatment of choice for cerebral air embolism, as it decreases bubble size and increases oxygen solubility in plasma thereby improving oxygen tissue delivery.\textsuperscript{1,22} In a recent review,\textsuperscript{1} hyperbaric oxygen therapy was recommended in all patients with arterial air embolism. Although there have been many promising results, even with delayed treatment, the efficacy of this treatment has yet to be proven, especially when taking into consideration the logistic difficulties involved and the cardiovascular instability of these patients.\textsuperscript{1,23}

Intraoperative retrograde cerebral perfusion has been used with a good neurological outcome for a proven arterial air embolism in a 5-year-old girl undergoing repair of an atrial-septal defect together with post-operative barbiturate induced anaesthesia and hyperbaric oxygen therapy.\textsuperscript{22} Other treatments, such as intravenous lignocaine, corticosteroids and heparin\textsuperscript{1} have been suggested for the management of arterial air embolism but none of these have been shown to be effective.


