Chapter 8

Air Embolism

Bojan Biocina, Mate Petricevic and Ivica Safradin

Additional information is available at the end of the chapter

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Abstract

Air embolism is one of the serious causes of morbidity and mortality in medicine and surgery, especially in cardiac surgery. Various medical and surgical procedures have been associated with the risk of air embolism. In the chapter, all procedures and pathologic conditions will be described, paying special attention to the root cause analysis of the events in any given circumstance. Special attention is to be paid to techniques of risk minimization of this serious complication. The chapter will give an in-depth insight to the anatomical, physiological and other preconditions of air embolism, thus helping the reader to implement preventive measures and to increase patient safety

Keywords: embolism, air, gas

1. Introduction

Air embolism is, although uncommon, a potentially catastrophic event that occurs as a consequence of the entry of air into either arteries or veins. Put briefly, air embolism occurs when atmospheric gas is introduced into the systemic vasculature. Here, it would be prudent to clarify that the most appropriate name for this entity would actually be "gas embolism". In most cases, gas embolism is in fact air embolism, although the medical use of other gases such as carbon dioxide, nitrous oxide and nitrogen can also result in the condition [1].

Air embolism in the vasculature is the clinical entity with the great potential for severe morbidity and mortality. Venous air embolism is more prevalent when compared to arterial gas embolism. Even though the etiology of air embolism will be discussed in more detail later on, it is worth to mention that air embolism is actually predominantly an iatrogenic complication of both diagnostic and therapeutic procedures in different medical specialties [1–6].

2. Etiology

As previously mentioned, gas embolism can be either venous or arterial. The most common causes of air embolism are surgery, trauma, vascular interventions and barotrauma from mechanical ventilation and diving [7–10].

Gas embolism most commonly occurs not only in an anterograde venous course, as is most typical, but also may occur via epidural spaces and/or via tissue planes [2]. Medical specialties with documented cases of gas embolism were comprehensively reviewed by Muth et al. [1]. According to the literature, gas embolism may occur in cardiac surgery, cardiology, critical care and pulmonology, diving and hyperbaric medicine, endoscopic and laparoscopic surgery, gastroenterology, nephrology, neurosurgery, obstetrics and gynecology, otolaryngology, orthopedics, urology, vascular surgery, etc. [9–23]. Among these, air embolism occurs more frequently in neurosurgical and otolaryngological procedures when compared to surgical procedures in other specialties. An air embolism incident during neurosurgical procedures ranges from 10 to 80%.

There are numerous surgical or other non-surgical invasive procedures where gas embolism has been reported as a complication: (1) needle biopsy of the lung (bronchoscopic or percutaneous), lung resection [15–17, 24] and radiofrequency ablation of lung cancer [25], (2) arthroscopy and arthroplasty [18, 26], (3) gynecological procedures (hysteroscopy [19, 27, 28], C-section [29]), (4) gastrointestinal procedures (laparoscopy [30], colonoscopy [21], endoscopic retrograde cholangiopancreatography (ERCP) [20]) and (5) cardiac procedures (heart surgeries performed with cardiopulmonary bypass [22], cardiac implantable electronic devices implantation [23, 31], cardiac ablation procedures of cardiac arrhythmias) [32–34]. Gas embolism has also been described in ophthalmological [35] and dental procedures [36]. Mechanisms for gas embolism differ widely among the specialties. For example, in cardiac surgery procedures, possible mechanisms are the entry of air into extracorporeal bypass pump circuit and incomplete removal of air from the heart following weaning from cardiopulmonary bypass [22]. In neurosurgery procedures, the possible mechanism of gas embolism is entry of air through incised veins and calvarial bone, especially during craniotomy with the patient in a sitting position. What remains common for all the surgical procedures is the intraoperative use of hydrogen peroxide which may cause formation of arterial and venous oxygen emboli [1].

Gas embolism may certainly occur when handling intravascular catheters. Gas emboli can occur at the time of catheter insertion, while catheter is in place, or at the time of catheter removal [37]. Handling different types of catheters, be it venous or arterial (i.e., central venous catheters [10, 38], hemodialysis catheters [39, 40], pulmonary artery catheters [41] and angioplasty catheters [42]) may result in gas embolism. When handling intravascular catheters, one should keep in mind the factors that contribute to gas embolism occurrence (fracture or detachment of catheter connections, failure to occlude the needle hub, dysfunction of self-sealing valves in plastic introducer sheaths, the presence of a persistent catheter tract following the catheter removal, deep inspiration during catheter insertion or removal, hypovolemia that reduces central venous pressure and upright positioning of the patient).

3. Detection of gas embolism

In order to diagnose air embolism, a clinician should first set the suspicion and should assess clinical findings. Many cases of gas embolism are subclinical with no adverse outcomes. Usually, even when symptoms are present, they are non-specific, and a high index of clinical suspicion for possible gas embolism is required to prompt investigations and initiate appropriate therapy. A splashing auscultatory sign indicating the presence of gas in cardiac chambers can be auscultated using stethoscope [1]. Doppler ultrasonography is a sensitive and a practical means of detecting intracardiac air [43, 44]. Transesophageal echocardiography remains an even more sensitive and definitive method for detecting intracardiac gas [45].

Transesophageal echocardiography is currently the most sensitive monitoring device for detection of air presence, detecting as little as 0.02 ml/kg of air administered by bolus injection [46, 47]. The major deterrents to transesophageal echocardiography are that it is invasive, is expensive and requires expertise and constant vigilance that may limit its use to just a well-trained cardiac anesthesiologist or cardiologist [2].

Noteworthy, a decrease in the end-tidal carbon dioxide levels, as determined by capnometry, may be suggestive of gas embolism as well.

4. Management

Early diagnosis and treatment before catastrophic cardiovascular collapse are of utmost importance. In general, there are three principle goals in air embolism management: (1) prevention of further air entry, (2) a reduction in the volume of air entrapped and (3) hemodynamic support [2]. In case of gas embolism, clinician should institute high-flow oxygen to maximize patient oxygenation during the period of hemodynamic instability. Nitrous oxide should be discontinued, and the patient should be placed on 100% oxygen. Administration of oxygen is important not only to treat hypoxia and hypoxemia but also to eliminate the gas in the bubbles by establishing a diffusion gradient that favors the egress of gas from bubbles [1, 48]. In certain cases, therapy with catecholamines is required, as well as aggressive cardiopulmonary resuscitation, if needed. Rapid volume expansion is recommended to elevate venous pressure, thus preventing the continued entry of gas into intravascular space. Normovolemia should be achieved to optimize microcirculation. Colloid solutions are preferable to crystalloid solutions for hemodilution as crystalloid solutions may promote cerebral edema.

Hyperbaric oxygen therapy decreases the size of the gas bubble both by rising the ambient pressure and by causing hyperoxia [1]. There is emerging evidence suggesting that all patients with clinical symptoms of arterial gas embolism should receive recompression treatment with hyperbaric oxygen, which is in fact considered the first line treatment of choice for arterial gas embolism [1, 49–51].

As Muth et al. discussed in their paper [1], there is evidence that heparin may be beneficial in the treatment of gas embolism [52]. The possible disadvantage would be the risk of hemorrhage

into the infarcted tissue. Whereas the use of corticosteroid therapy remains controversial and to date is not recommended, the lidocaine therapy has been shown to provide cerebral protection during cardiac surgery [53]. Even with lidocaine, the evidence is controversial [1, 54] and further research is needed to shed a light into its neuroprotective role.

In conclusion, gas embolism is a risk associated with different diagnostic and/or therapeutic procedures in virtually all medical specialties [1]. Arterial gas emboli may be particularly dangerous if they occlude cardiac or cerebral vessels [1]. Whereas hyperbaric oxygen remains the first choice of arteria gas emboli treatment, the mainstays of treatment for venous gas embolism are volume expansion, targeting 12 mm of mercury of central venous pressure, the administration of 100% oxygen, often with ventilatory support [1]. Finally, prevention of air embolism and prevention of further entry of gas in cases of present air embolism remain the Cornerstone Treatment in management of patients at risk for such a clinical entity.

Author details

Bojan Biocina[1]*, Mate Petricevic[2] and Ivica Safradin[2]

*Address all correspondence to: bbiocina@kbc-zagreb.hr

1 Department of Cardiac Surgery, School of Medicine, University of Zagreb, Zagreb, Croatia

2 Department of Cardiac Surgery, UHC Zagreb, Zagreb, Croatia

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