Journal of Critical Care 42 (2017) 255–263

Contents lists available at ScienceDirect

Journal of Critical Care

journal homepage: www.jccjournal.org

Vascular air embolism: A silent hazard to patient safety

Sorin J. Brull [a,*], Richard C. Prielipp [b]

[a] Department of Anesthesiology and Perioperative Medicine, Mayo Clinic College of Medicine, Jacksonville, FL 32224, USA
[b] Department of Anesthesiology, University of Minnesota, Minneapolis, MN 55455, USA

ARTICLE INFO

Keywords:
Cerebrovascular circulation
Central venous catheters
Air embolism
Intravenous infusion
Pulmonary circulation

ABSTRACT

Purpose: To narratively review published information on prevention, detection, pathophysiology, and appropriate treatment of vascular air embolism (VAE).
Materials and methods: MEDLINE, SCOPUS, Cochrane Central Register and Google Scholar databases were searched for data published through October 2016. The Manufacturer and User Facility Device Experience (MAUDE) database was queried for "air embolism" reports (years 2011–2016).
Results: VAE may be introduced through disruption in the integrity of the venous circulation that occurs during insertion, maintenance, or removal of intravenous or central venous catheters. VAE impacts pulmonary circulation, respiratory and cardiac function, systemic inflammation and coagulation, often with serious or fatal consequences. When VAE enters arterial circulation, air emboli affect cerebral blood flow and the central nervous system. New medical devices remove air from intravenous infusions. Early recognition and treatment reduce the clinical sequelae of VAE. An organized team approach to treatment including clinical simulation can facilitate preparedness for VAE. The MAUDE database included 416 injuries and 95 fatalities from VAE. Data from the American Society of Anesthesiologists Closed Claims Project showed 100% of claims for VAE resulted in a median payment of $325,000.
Conclusions: VAE is an important and underappreciated complication of surgery, anesthesia and medical procedures.

Contents

* Corresponding author at: Department of Anesthesiology and Perioperative Medicine, Mayo Clinic Florida, 4500 San Pablo Rd S, Jacksonville, FL 32224, USA.
E-mail address: sjbrull@me.com (S.J. Brull).

http://dx.doi.org/10.1016/j.jcrc.2017.08.010
0883-9441/

1. Introduction

Vascular air embolism (VAE) occurs when air or medical gas enters a patient's venous or arterial circulation. Depending on the volume and rapidity of gas entry into the patient's vasculature, the physiologic effects of VAE may be trivial or catastrophic. Symptoms are cardiovascular, neurological and pulmonary. VAE occurs during surgery, traumatic events, endovascular or other interventional procedures, labor and delivery, or may be a complication of mechanical ventilation. Occurrence of VAE has been widely published in case reports and animal studies evaluating the etiology of VAE, alongside methods of prevention, detection, and treatment. With the advent of new medical technologies, the purpose of this narrative review is to provide updates on this classic clinical phenomenon. Occupational hazards of VAE (e.g., scuba diving) will not be reviewed.

2. Material and methods

The authors searched MEDLINE, SCOPUS, Cochrane Central Register and Google Scholar databases for articles providing preclinical and clinical information on the topic of VAE published through October 2016. Search terms included 'air embolism' or 'air emboli', 'vascular embolism', 'venous embolism', 'arterial embolism', and 'gas embolism'. Two independent searches were performed to establish a comprehensive data set; one search by the Mayo Clinic library, and a second search by an independent medical researcher/writer. References were downloaded into reference manager software, duplicates removed, and an initial review for relevance was performed. Descriptive, methodological, and outcome data from the remaining 605 abstracts were reviewed by the authors and refined for inclusion. Fig. 1 includes details of the search methodology. Additionally, the FDA Manufacturer and User Facility Device Experience (MAUDE) database was searched for all reports of "air embolisms" from 2011 to 2016 [1].

3. Results

3.1. Pathophysiology of the VAE

The pathophysiology of VAE typically requires both a negative pressure gradient and a communication channel between the vasculature and the atmosphere. Classically this happens in procedures where the surgical site is above the level of the heart, creating a gravitational gradient for air entry (e.g., sitting craniotomy), and when non-compressible venous channels (e.g., dural veins) are invaded, creating an open portal for air to enter the circulation [2]. VAE may occur during intravenous catheterization, surgical procedures, radiologic procedures, trauma, or positive pressure ventilation [3,4]. Less common situations include airline travel, lung biopsy, childbirth and diving [3,5–9]. Common entry conduits include central venous catheters (CVCs), peripheral catheters, and during intravenous infusions when residual air remains in the closed system, or when the intravenous tubing is compromised [10–18]. In most cases VAE is composed of atmospheric air, but may include oxygen mixtures and medical gases such as carbon dioxide, nitrous oxide, helium or nitrogen [19–22].

Modern medical and surgical procedures have created a second, more insidious pathway for VAE to enter circulation. Contrast injection during radiographic imaging, laparoscopic abdominal insufflation, or intra-aortic balloon rupture inject air, gases, or other fluids into various body cavities under pressure. While the pressure of injections is typically closely monitored, the volume of injected gas is seldom measured or monitored, potentially forcing large amounts of air or gas into vessels or cavities [2].

3.2. Venous air embolism

The most common type of VAE occurs when air enters the venous circulation (venous air embolism). Venous air embolism may be completely asymptomatic, as small amounts of air are typically broken up in the capillary bed of the lungs and are absorbed without causing symptoms. Alternatively, some patients exhibit minor symptoms like coughing. However, larger volumes of venous air produce pulmonary vasoconstriction, increased pulmonary artery pressures, elevated resistance to right ventricular outflow and ultimately, right ventricle failure [23-24]. Diminished right ventricular performance and interventricular septal shift decrease left ventricular preload, decreasing left heart output and eventually leading to systemic cardiovascular collapse. Further, activated neutrophils in the pulmonary capillary beds release thromboxane and leukotrienes, increasing airway resistance and alveolar capillary permeability, and resulting in pulmonary edema and alveolar collapse [25]. Findings in animals suggest that repeated exposure to venous air embolism damages micro-vessels in the pulmonary arteries leading to pulmonary hypertension [26-27] and widespread edema [28-29]. Underlying pulmonary disease in the form of pulmonary calcification decreases tolerance for even small amounts of air [30]. In extreme cases, a large bolus of venous air may fill the right atrium and ventricular outflow track, causing complete obstruction of right ventricular flow, cardiac arrhythmias, right ventricular dilation, failure of cardiac output, and cardiovascular collapse. Myocardial ischemia leads to cardiac arrest [10,31].

The ultimate impact of VAE depends on a number of factors, including the intense inflammatory response initiated by microemboli trapped in the capillaries. The pathogenesis of these emboli can therefore be modulated – at least in the experimental laboratory – by establishing leukopenia prior to the embolic insult [32]. For instance, in five leukopenic rabbits, infusion of 400 μL of air in the cerebral circulation did not produce the anticipated decline in cerebral blood flow or the cortical somatosensory evoked response. The authors concluded that changes of cerebral air embolism, such as decreases in cerebral blood flow and brain function, requires the presence of activated leukocytes [32].

3.3. Arterial air embolism/paradoxical embolism

Venous air emboli may enter the arterial (systemic) circulation due to intra-cardiac and intra-pulmonary shunting. If the "filter capacity" of the pulmonary capillary bed is overwhelmed, translocation of air from the venous to arterial side of circulation occurs [33]. This phenomenon is termed "paradoxical air embolism" and may result in cerebral air embolism (CAE) producing a significant risk for stroke or death. Arterial air embolism results in ischemia of the organ where the air ultimately becomes trapped. In dogs, a volume of air of 0.02 mL/kg can be fatal [25]. Air in the arterial circulation can form microscopic spheres that can disrupt the microvascular circulation, and trigger release of plasminogen-activator inhibitor and induce platelet aggregation [34]. These changes may in turn trigger the release of cytokines (such as interleukin-1 and tumor necrosis factor) from the arterial endothelium, resulting in diffuse endovascular injury, microvascular thrombosis, organ ischemia, and multi-organ failure [34-36].

Arterial air embolism occurs at greater frequency in patients with an atrial-septal defect (ASD) or patent foramen ovale (PFO), which allow transcardiac passage of the air from the venous (right heart) to arterial (left heart) systems. Clinicians should recognize that one-quarter of the adult population has an asymptomatic and undiagnosed PFO [37-41]. Although rare, paradoxical air emboli have also been reported across atrial-esophageal fistulae and via intrapulmonary arterio-venous anastomoses [42-44].