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Vascular Air Embolism-A Review

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Abstract
Air embolism was reported as early as the 19th century in pediatric and adult practice. Vascular air embolism is the entrainment of air from the operative field or other communication with the environment into the venous or arterial vasculature, producing systemic effects. It is a rare but potentially life threatening event. It may occur in various procedures and surgeries but mostly associated as an iatrogenic complications. It can be venous or arterial, both condition can be differentiated by mechanism of air entry as well as site of embolization. Air embolism are no longer limited to neurosurgical procedures conducted in the "sitting position".The physiologic effects that result depend on the volume of air that has entered the system. It affects cardiovascular, pulmonary and central nervous system. This article reviews the etiology, pathophysiology, symptoms, diagnosis, treatment of the vascular air embolism.

INTRODUCTION

Vascular air embolism (VAE) is known since early 19th Century but the interest and reporting of VAE significantly increased in last three decade. Most of the episodes of VAE are preventable or at least detected early and managed properly[1].Air or gas is admitted into vascular system which is known as vascular air embolism. Usually an embolus lodging in the brain from either the heart or a carotid artery will most likely be the cause of a stroke due to ischemia. An arterial embolus might originate in the heart[2].It is potentially life threatening as it can lead to circulatory deficiency in the body organ with poor collateral circulation[3].It can occur iatrogenically via interventional procedures but has also been described as a complication from a variety of circumstances ranging from blunt and penetrating trauma to diving and child birth. The physiologic effects that result depend on the volume of air that has entered the system. A patient's symptoms may range from asymptomatic to cardiovascular collapse and death[4].Here we will review the VAE in the following sub-headings.

ETIOLOGY

Air embolism is a rare but potentially fatal occurrence. It can occur during various surgical interventions, especially neurosurgical, vascular, obstetric, gynecological, or orthopedic procedures[5].Conditions for the entry of gas into the venous system are the access of veins during the presence of negative pressure in these vessels. This is most commonly associated with central venous catheterization, as the potential for negative pressure exists in the thoracic vessels due to respiration. Also can occur in incorrect execution of procedures for pressure infusion and not properly filled and completely vented infusion line [6]. For air to enter a closed system, a connection must occur between the gas and the vessel and a pressure gradient must exist that enables flow of the air into the vessel[7].If a connection between the two systems exists,a venous air embolism always has the potential to become an arterial embolism. If a right to left pressure gradient exists, the gas can then travel from the venous to the arterial circulation[8].

PATHOPHYSIOLOGY

The embolism is propelled into the heart, creating an intracardiac air lock at the pulmonary valve and preventing the ejection of blood from the right ventricle of the heart. The right side of the heart overfills with blood because less blood is ejected from the right ventricle[9]. Smaller amount of air in the circulation does not cause any clinical manifestations but moderate amount of air causes pulmonary vascular injury leading to pulmonary hypertension and permeability pulmonary edema.Large bolus of air in venous system can cause an air lock in right side of the heart leading to right ventricular flow obstruction and death[10]. Air passes from right side to the left side of the heart and leading to systemic air embolization due to the presence of patent foramen ovale this is termed as paradoxical embolism[11]. The physiologic effects of vascular air embolism are evidenced by (1) elevated pulmonary artery and right ventricular pressures; (2) increased ventilation/perfusion mismatch; (3) intrapulmonary shunting; and (4) increased alveolar dead space. The degree of physiologic impairment depends on the volume of air, rate of air embolism, the type of gas (room air, carbon dioxide or nitrous oxide), and the position of the patient when the embolism occurs[12]. Multiple additional clinical settings have reported the occurrence of air embolism. These include but are not limited to disconnected central venous catheters, airline travel, ERCP, hemodialysis, trauma, laparoscopic insufflations, open heart surgery, lung biopsy, radiologic procedures, [2,6,14,15] childbirth, head and neck surgery, and diving.

SYMPTOMS

The symptoms and clinical signs of air embolism are related to the degree of air entry into the circulation system. They usually develop immediately after embolization. It may have cardiovascular, pulmonary, and neurologic sequelae. Any amount of air that might enter the patient must be considered critical. The impact is directly correlated with the patient's condition, the volume of air and the rate of accumulation. If a patient is conscious during the event, chest pain, dyspnea, headache, and confusion can all be symptoms of air emboli. Clinical

complications are diminished cardiac output, shock and death[13].

Cardiovascularly, tachyarrhythmias are common, Blood pressure decreases,Pulmonary artery pressures increase and decrease cardiac output.

Pulmonary symptoms are acute dyspnea, continuous coughing, urgent complaints of breathlessness, lightheadedness, chest pain and a sense of “impending doom.” Pulmonary signs are rales, wheezing, and tachypnea.

If embolization occurs to the cerebral arteries patients can have symptoms of confusion, stroke, cerebral hypo perfusion,cerebral edema, cerebral ischamia , cerebral hyperaemia , altered mental status [14].

DIAGNOSIS

Patient's history is the most important diagnostic criterion because embolism is based on the neurological symptoms The detection of VAE is taking into consideration the circumstances under which clinical alterations occur. There are specific circumstances where the diagnosis of VAE should be considered immediately in the differential diagnosis.

Any unexplained hypotension or sudden decrease in end-tidal CO2 level occurs intraoperatively in high risk cases.

If patient complains of short of breath during or immediately after insertion or removal of central venous catheter[15].

Patients undergoing cesarean delivery who have sustained hypotension and or hypoxia.

VAE should be differentiated from acute coronary syndrome, cardiogenic shock, cerebrovascular accidents, and pulmonary embolism. Various monitoring devices can help in early detection of air in the vascular system which includes

Transesophageal echocardiography
Precordial doppler ultrasound
End tidal nitrogen (ETN2)
End-tidal carbon dioxide (ETCO2)
Transcranial doppler and Esophageal Stethoscope
Electrocardiogram (ECG)[16].

TREATMENT

The treatment of VAE is primarily to prevent the air entry and reduction in volume of air entrained and aspiration of air from right atrium. Trendelenburg's position is a favorable placement which allows the entrapped air in the heart to be stabilized within the apex of the ventricle.To maximize the patient's oxygenation administrate 100% oxygen and it will also reduces the embolus volume by eliminating nitrogen[17].

Cardiac standstill can occur due to rapid cardiopulmonary resuscitation with chest compression. Hyperbaric oxygen therapy can be used as it causes compression of existing air bubbles by improving oxygenation in the ischemic tissues. In decompression syndrome, patients should be transported in supine position to minimize risk of cerebral embolization[18].

Anticoagulation therapy with heparin in patients with air embolism decreases the severity of the disease, if treated with heparin before air embolization. Steroid is controversial because it do not have any effect on cytotoxic brain edema which occurs in the patients with air emboilsm. Prophylactic lidocain is effective in reducing the gas embolism effect on brain[19].

CONCLUSION

Vascular air embolism (VAE) is preventable critical medical emergency. VAE is also common in obstetric and laproscopic surgeries.Clinical manifestation of VAE is mainly due to the involvement of respiratory, cardiovascular, and central nervous system.In the case of severe multiple complications which require full ICU treatment.A high degree of suspicion when doing high risk procedures should be present in order to promote early recognition and potentially life-saving therapy.

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