

Cause Of Air Embolism: Injury Or Treatment

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ABSTRACT:

A 15 years old male was assaulted by a few people near his home. He suffered with few abrasions, a contusion and minor fracture of skull bone. He survived for approximately 22 hours while on conservative treatment. On postmortem examination, frothy blood was seen coming out from the right ventricle, along with plenty of air bubbles in jugular veins and in heart as well eliciting the diagnosis of air embolism. The case is being presented here for drawing attention towards the fact that simple head injury, augmented by lapses in treatment due to lack of suspicion, can lead to fatal degrees of air embolism.

Key-words: Venous air embolism, Gas embolism, Petrous temporal sinus, Head up position.

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INTRODUCTION:

An air embolism, or more generally gas embolism, is a condition caused by air/gas bubbles in the vascular system. It can be either arterial or venous air embolism. Venous air embolism (VAE), a subset of gas embolism, is an entity with the potential for severe morbidity and mortality. Venous air embolism is a predominantly iatrogenic complication that occurs when atmospheric gas is introduced into the systemic venous system.^{1,2,3} In the past, this medical condition was mostly associated with neurosurgical procedures conducted in the sitting position.^{4,5} More recently, venous air embolism has been associated with central venous catheterization,^{3,6,7} penetrating and blunt chest trauma,^{8,9} high-pressure mechanical ventilation,³ thoracocentesis,¹ hemodialysis,^{3,7} and several other invasive vascular procedures.

Many cases of VAE are subclinical with no adverse outcome and thus go unreported. Usually, when symptoms are present, they are nonspecific, and a high index of clinical suspicion of possible venous air embolism is required to prompt investigations and initiate appropriate therapy. Two preconditions must exist for venous air embolism to occur: (1) a direct communication between a source of air and the vasculature and (2) a pressure gradient favoring the passage of air into the circulation.⁴ The key factors determining the degree of morbidity and mortality in venous air emboli are related to the volume of gas entrainment, the rate of accumulation, and the patient's position at the time of the event.^{1,6} Generally, small amounts of air are broken up in the capillary bed and absorbed from the circulation without producing symptoms. Traditionally, it has been estimated that more

than 5 ml/kg of air displaced into the intravenous space is required for significant pathology (shock or cardiac arrest) to occur.¹ However, complications have been reported with as little as 20 ml of air⁷ (the length of an unprimed IV infusion tubing) that was injected intravenously. The injection of 2 or 3 ml of air into the cerebral circulation can be fatal.

Rapid entry of air or large volumes of air entering the systemic venous circulation put a substantial strain on the right ventricle, especially if this results in a significant rise in pulmonary artery (PA) pressures. This increase in PA pressure can lead to right ventricular outflow obstruction and further compromise pulmonary venous return to the left heart. The diminished pulmonary venous return will lead to decreased left ventricular preload with resultant decreased cardiac output and eventual systemic cardiovascular collapse.^{1,4,6}

With venous air embolism (VAE), resultant tachyarrhythmias are frequent, but bradyarrhythmias can also occur.^{4,2} Secondary pathology as a result of the activation of complement and the release of mediators and free radicals can lead to capillary leakage and eventual noncardiogenic pulmonary edema.^{1,7,3}

The potentially life-threatening and catastrophic consequences of venous air embolism (VAE) are directly related to its effects on the affected organ system where the embolus lodges. VAE may be fatal and frequently carries high neurologic, respiratory, and cardiovascular morbidity. Catheter-associated VAE mortality rates have reached 30%.² In a case series of 61 patients with severe lung trauma, the mortality rate associated with concomitant VAE was 80% in the blunt trauma group and 48% in the penetrating trauma group.^{8,14,15} The morbidity and mortality associated with traumatic VAE, as with nontraumatic VAE, depends not only on associated injuries but also on the volume and rate of air entry, underlying cardiac condition, and the patient's position.

Laboratory Studies

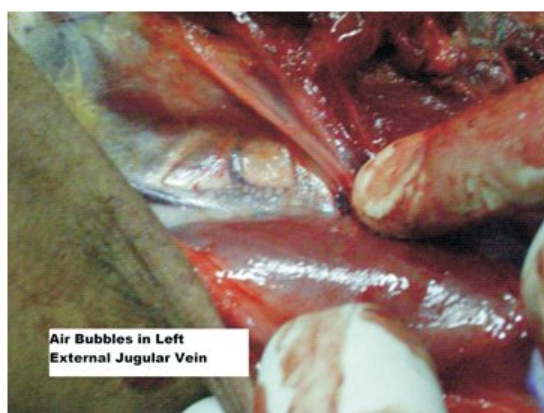
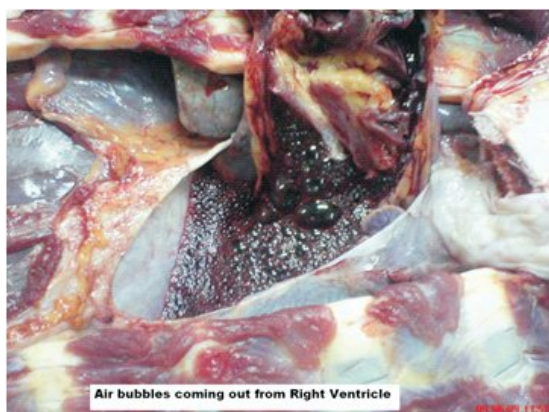
- Laboratory tests are neither sensitive nor specific for the diagnosis of venous air embolism. The only indication for obtaining routine laboratory tests is to evaluate the associated end-organ injury resulting from air embolism.
- Extravasation of fluid into inflamed tissue may result in laboratory findings consistent with intravascular depletion.
- Arterial blood gas samples often show hypoxemia, hypercapnia, and metabolic acidosis secondary to right-to-left pulmonary shunting.

Patients may develop a clinical picture similar to that of classic pulmonary embolism, with hypoxia, decreased PCO₂ levels, and respiratory alkalosis.

CASE HISTORY:

In the second week of June – 2009, dead body of an identified male aged about 15 years, was brought to the Dept. of Forensic Medicine and Toxicology, Govt. Medical College, Surat, Gujarat, for conducting post-mortem examinations. According to police papers, this was a case of assault—a head injury, being inflicted on the deceased and his father, in the early morning of a day before the post mortem examination was started. Father of the deceased had died immediately, while the subject survived for approximately 22 hours after infliction of injury. During this time, investigations,

Subdural and subarachnoid hemorrhage as described in CT was also seen. On opening the neck, extensive air bubbles was founded in the external jugular veins bilaterally. Then the body of the sternum was removed along with the ribs while the manubrium was kept in place, the pericardium was opened, heart was lifted and an incision was kept on the anterior surface which revealed huge bubbles of air and frothy blood coming out from the right ventricle. The cause of death was hence revealed to be “air embolism due to head injury”.



DISCUSSION:

Most of the cases of air embolism occur either due to rapid ascent from a high pressure zone to a low pressure one or due to free passage of air across the pressure gradient, in an open vascular channel, mostly a vein. In the second scenario, most cases are iatrogenic in nature, i.e. either because of

some cranial surgeries in sitting position or through central lines, venous catheters, and air insufflations in the uterus or in positive pressure ventilation where the pressure maintained is much more than the resistance offered by the lungs.

In this case also, the second factor played an important role, i.e. a pressure gradient favoring the entry of air into an open vascular channel petrous temporal sinus. The condition was aggravated by the prescription of “head up” position of the patient, probably to control cerebral edema. This, along with sufficient time for air to enter the venous circulation through the fracture in the air cells and temporal bone, resulted in the accumulation of air in the right ventricle – ultimately leading to right heart failure.

CONCLUSION:

- Adequate scientific examination and interpretation during autopsy enables the autopsy surgeon to find out the exact cause of death in doubtful cases.
- A thorough study and knowledge of the circumstances of death may also reveal significant clues regarding the mode and cause of death and hence a sincere attempt should be made to study the variables surrounding death – including the site and the size of the injury, its possible complications, the treatment given and its possible side effects.
- A high degree of suspicion needs to be maintained to diagnose air embolism and hence to prevent its complications.

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