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Review Article

Venous Air Embolism: A Case Report

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Abstract

Vascular air embolism is the entrainment of air (or exogenously delivered gas) from the operative field or other communication with the environment into the venous or arterial vasculature, producing systemic effects. The true incidence of VAE may be never known, much depending on the sensitivity of detection methods used during the procedure. In addition, many cases of VAE are subclinical, resulting in no untoward outcome, and thus go unreported. That's why a high index of suspicion is necessary to establish the diagnosis and institute the appropriate treatment. The number of procedures that place patients at risk for VAE has increased, and these procedures occur across almost all clinical specialties. Venous air emboli pose a risk anytime the surgical wound is elevated more than 5 cm above the right atrium. The presence of numerous, large, non-compressed, venous channels in the surgical field (especially during cervical procedures and craniotomies that breach the Dural sinuses) also increase the risk of VAE.

The Objective of the following report is to present the case of a posterior fossa surgery, complicated by a VAE at the scalp incision and at the scalp closure. With appropriate patient selection and preparation, also the use of prudent intraoperative monitoring and anesthetic techniques, selected patients should still benefit from the optimum access to mid-line lesions, improved cerebral venous decompression, and lower intracranial pressure and enhanced gravity drainage of blood and CSF associated with the sitting position.

Keywords: vascular air embolism ; arterial vasculature ; right atrium

Introduction

The introduction of atmospheric gas into the systemic venous system, is defined as Venous Air Embolism (VAE). This medical condition was mostly associated with neurosurgical procedures conducted in the sitting position, central venous catheterization, blunt chest trauma, high-pressure mechanical ventilation, thoracocentesis, hemodialysis, and several other invasive vascular procedures. Many cases of VAE are subclinical with no adverse outcome and thus go unreported. Any possible suspicion in the occurence of venous air embolism is referred to prompt investigations because the non-specificity of the symptoms. The potentially life-threatening and catastrophic consequences of venous air embolism are directly related to its effects on the affected organ system where the embolus lodges. Venous air embolism has been a challenging factor due to its fatal effects on patients and high respiratory, neurologic, and cardiovascular morbidity rates. Generally, if the patient is in a sitting position, gas will travel retrograde via the internal jugular vein to the cerebral circulation, leading to neurologic symptoms secondary to increased intracranial pressure. Hypertension and systemic hypotension are noticed when patients are placed in a recumbent position, which affects the flow of gas into the right ventricle and pulmonary circulation. The above hemodynamic, pulmonary, and neurologic complications primarily result from gas gaining entry into the systemic circulation, occluding the microcirculation and causing ischemic damage to these end organs. There is a 10% estimation in the incidence of VAE during cervical laminectomy surgeries where the patients are in the prone position, and 80% incidence during posterior fossa surgeries (eg, repair of cranial synostosis) where patients are placed in the Fowler's position. A direct communication between source of air and the vasculature and a pressure gradient favoring the passage of air into the circulation, are two conditions that indicate the presence of VAE case. 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. There is a slight amount air that is broken up and absorbed in the systemic circulation without causing any symptom. Traditionally, it has been estimated that more than 5 mL/kg of air displaced into the intravenous space is required for significant injury (shock or cardiac arrest) to occur. However, complications have been reported with as little as 20 mL of air that was injected intravenously.

Rapid entry or large volumes of air entering the systemic venous circulation puts a substantial strain on the right ventricle, especially if this results in a significant rise in pulmonary artery (PA) pressures. Pulmonary venous return to the left heart and right ventricular outflow obstruction are linked to an increase in the PA pressure. The diminished pulmonary venous return will lead to decreased left ventricular preload with resultant decreased cardiac output and eventual systemic cardiovascular collapse. Myriad cellular changes are linked to a rapid ingress of air (>0.30 mL/kg/min) into the circulatory system, where it can overwhelm the air-filtering capacity of the pulmonary vessels. Thus, it can lead to serious inflammatory changes in the pulmonary vessels; these include direct endothelial damage and accumulation of platelets, fibrin, neutrophils, and lipid droplets. The release of mediators and free radicals lead to capillary leakage and no cardiogenic pulmonary edema which are a main source of secondary injury. Alteration in the resistance of the lung vessels and ventilation-perfusion mismatching can lead to intra-pulmonary right-to-left shunting and increased alveolar dead space with subsequent arterial hypoxia and hypercapnea. Arterial embolism is one the known complications of VAE. It occurs through the direct passage of air via anomalous structures (i.e. atrial ventricular septal defect, a patent foramen ovale, or pulmonary arterial-venous malformations) and into the arterial system. The incidence of paradoxical embolus has shown an increase during procedures that are done in the sitting position.

A 65 years old male patient, with no past medical or surgical history, heavy smoker was admitted for severe right sided headache associated with left eye vision disturbances. An MRI was done and showed a right parieto-occipital tumor at the posterior horn of the ventricle. The preoperative investigations were normal so the patient was scheduled for craniotomy (posterior fossa) in sitting position.

The induction of anesthesia was with propofol and fentanyl, maintenance with propofol (4mg/kg/hr) isoflurane 1% and remifentanil (0.1 micg/kg/min). A central catheter was placed with an arterial line, before incision the patient received 2 liters of normal saline. At the scalp incision we were surprised by a decrease of the end tidal CO2 from 31 to 10, with a decrease of the blood pressure from 110/70 to 70/45. Surgeon was informed; surgical field was covered, with aspiration of 40 cc of air from the central catheter. The diagnosis of venous air embolism was established. The surgery was stopped due to cardiovascular instability and rescheduled for another day.

One week later, the same patient was scheduled for the same procedure. A cardiac ultrasound was done confirming a normal heart with no patent foramen oval. The induction of anesthesia was with propofol and fentanyl, maintenance with propofol (4mg/kg/hr) and remifentanil (0.1micg/kg/min). An internal jugular was placed with an arterial line, before incision the patient received 2 liters of normal saline and 2 liters of Ringer's solution. Surgery went smoothly with no complications. However at the end of the surgery at the closure of the scalp the end tidal CO2 dropped from 29 to 15 and 10cc of air were aspirated from the central line, patient was stable. Surgery finally was done, patient transferred to ICU after 24 hours he was transferred to the floor, 7 days later he was discharged from the hospital.

Discussion

Anesthesiologists find it challenging when it comes to avoiding physiological challenges while performing posterior fossa surgeries in a sitting position. The sitting position provides optimum access to midline lesions, improves cerebral venous decompression, lowers intracranial pressure and promotes gravity drainage of blood and CSF. Complications related to the use of this position are numerous and it includes: hemodynamic instability, VAE, pneumocephalus, quadriplegia and compressive peripheral neuropathy.

The two fundamental factors determining the morbidity and mortality of VAE are directly related to the volume of air entrainment and rate of accumulation. Thus, the position of the patient and the height of the vein are considered when dealing with air being suctioned according to a graviatational gradient. From case reports of accidental intravascular delivery of air, the adult lethal volume has been described as between 200 and 300 ml, or 3–5 ml/kg. The closer the vein of entrainment is to the right of the heart, the less the lethal volume is. Thus, the rate of air entrainment is also of importance, because the pulmonary circulation and alveolar interface provide a reservoir for dissipation of the intravascular gas. Thus, both volume and rate of air accumulation are dependent on the size of the vascular lumen as well as the pressure gradient. Moreover, the heart may withstand large quantities of air if the entrainment is slow. There may be complete outflow obstruction from the right ventricle which will rapidly leads to right-sided heart failure and immediate cardiovascular collapse, for example if the embolism is 5ml/kg, a gas-air lock scenario occurs. Effects of embolism are decrease in cardiac output, hypotension, myocardial and cerebral ischemia, and death. Air entrainment into the pulmonary circulation may lead to pulmonary vasoconstriction, release of inflammatory mediators, bronchoconstriction, and an increase in ventilation/ perfusion mismatch.

Diagnosis of an acute episode of VAE should be made immediately with no delay for the institution of the proper management. For that, anesthesiologist's need a thorough understanding of the clinical signs. Monitors in the operating rooms will aid in the detection of VAE. We will have tachyarrhythmias, and the electrocardiogram demonstrates a right heart strain pattern as well as ST–T changes. Myocardial ischemia may be observed. Blood pressure decreases. Pulmonary artery pressures increases as a consequence of increased filling pressures, reduction of cardiac output, jugular venous distention, and increase in central venous pressure. Respiratory monitoring will show a decrease in end-tidal carbon dioxide (ETCO2), and in arterial oxygen saturation (SaO2). These will lead to cerebral hypo perfusion. It is known and recommended by anesthesiologists due to its convenience and criticality when dealing with high-risky populations. A change of 2 mmHg ETCO2 can be an indicator of VAE. Therefore, the "low"-level alarm should be adjusted to detect even this small decrement, especially in high-risk procedures. Unfortunately, ETCO2 monitoring is not very specific. Anticipated vascular bleeding, or vascular abdominal cases (cesarean delivery), precordial Doppler ultrasound should be strongly considered in the anesthetic plan. It is the most cost effective, most easy to use, and least invasive of the sensitive monitoring devices. There is still no sufficient information about the clinical benefits of trans cranial Doppler or TEE.

The diagnosis and treatment of VAE requires more advanced monitoring devices, that provide early diagnosis and prognosis before any critical cardiovascular collapse occurs. Management modalities of VAE are through the prevention of further air entry; the reduction in the volume of air entrained, and hemodynamic support.

Air Entrainment

Saline soaked dressing and stopping further entrainment of air both prevent air entrainment. The surgeon should then be asked to assess and to eliminate any entry site. The tilt of the operating table should be adjusted to lower the likely source of air entry and eliminate the negative air pressure gradient. Transient jugular venous compression reduces air entrainment during cranial surgeries. In which retrograde may result due to increasing venous pressures that open the sinuses.

High flow of Oxygen

While facing cardiovascular instability, discontinuation of nitrous oxide should be considered and placement under 100% oxygen should be done. This process eliminates nitrogen N and reduce embolus volume. Air

might not clear rapidly after VAE and may be still susceptible to augmentation.

Embolic Obstruction

Placing the patient in a partial left lateral decubitus position (Durant maneuver), relieves air-lock in the right side of the heart, or simply by placing the patient in the Trendelenburg position if the patient is hemodynamically unstable. Recent literatures didn't give any conclusion about the advantages of neither position on patient hemodynamics when VAE occurs.

Aspiration of Air from the Right Atrium

Although intuitive, the success rates of appreciable aspiration of air during VAE are far from ideal. In one of the earliest case reports, Stallworth *et al.* reported that withdrawing 15 ml of air from the right heart percutaneously in a case of venous air embolism, resulting in prompt hemodynamic improvement. The average of aspirated volumes of air are 15-20 ml as measured by a variety of devices.

Hemodynamic Support

Acute right ventricular failure and subsequent decrease in left ventricular output occur after the increase in clinical VAE. Optimizing myocardial perfusion is a management option that provides inotropic support of the right ventricle. Patients with a low central venous pressure have increased incidences of VAE, because of the negative pressure gradient at the wound site compared to the right atrium. Thus, hydration reduces the risk of VAE. Respiratory variations in systolic blood pressure and urine, measurement of central venous pressure, and optimization of volume status are some parameters that should be considered in order to prevent wide gradients between the right atrium and the entraining vein.

Prevention is the key to manage VAE cases. Even after significant VAE, the greatest risk to the patient is continued entrainment of air. Preventive measures such as reducing the pressure gradient through repositioning, irrigating the field with fluid, intravascular volume loading, and use of moderate levels of PEEP remain important. Management algorithms such as recognizing procedures at risk of VAE and proper monitoring are highly important for patient safety. For catastrophic VAE with cardiovascular collapse, use of inotropic support and, if necessary, cardiopulmonary resuscitation are standard measures that also may have a beneficial action in clearing residual air embolism. There is little evidence to support special patient positioning as a means to enhance air dispersion.

Concerning the case presented above, we realized that preoperative hydration didn't help in the prevention of the occurrence of VAE. Also, we realize now that VAE occurs in a perfectly healthy patient without the presence of a patent foramen oval. In the second setting, isoflurane was not used as a maintenance for anesthesia due to its cardioplegic effects but there is no elaborative studies showing the relationship between isoflurane and the incidence of VAE. Once the patient is stabilized there was no need to call off the surgery, as it occurred the second time. Early detection and supportive measures are sufficient to stabilize the patient in order to proceed with the surgery.

Conclusion

Vascular air embolism is a potentially life-threatening event that is now encountered routinely in the operating room and other patient care areas. The circumstances under which physicians may encounter air embolism are no longer limited to neurosurgical procedures conducted in the "sitting position" and occur in such diverse areas as the interventional radiology suite or laparoscopic surgical center. Advances in monitoring devices coupled with an understanding of the pathophysiology of vascular air embolism will enable the physician to successfully manage these potentially challenging clinical scenarios. A comprehensive review of the etiology and diagnosis of vascular air embolism, including approaches to prevention and management based on experimental and clinical data, is presented. This compendium of information will permit the healthcare professional to rapidly assess the relative risk of vascular air embolism and implement monitoring and treatment strategies appropriate for the planned invasive procedure. The anesthesiologist must be aware that surgery in the head-up position places the patient at risk for VAE. In such situations, the propensity of incurring a negative gradient between the open site veins and the right atrium can be decreased by increasing right atrial pressure *via* leg elevation and using the "flex" option on the operating table control. Vascular air embolism may be detected by ETCO2 monitoring, and precordial Doppler ultrasound should be used in moderate- to high-risk patients undergoing high risk procedures. Emphasis is given to the prevention (hydration, positioning) and prompt recognition of this event and to the use of all available tools (fluids, positive inotropic agents) in the management of cardiovascular complications. The use of invasive monitoring devices such as TEE and central venous catheters should be dictated by the presence of comorbidities, rather than as a primary tool to manage VAE.

With appropriate patient selection and preparation, also the use of prudent intraoperative monitoring and anesthetic techniques, selected patients should still benefit from the optimum access to mid-line lesions, improved cerebral venous decompression, and lower intracranial pressure and enhanced gravity drainage of blood and CSF associated with the sitting position. Echocardiography should be used as a screening technique to detect the population at risk of paradoxical air embolism caused by the presence of a patent foramen ovale.

References

1. Albin MS, Ritter RR, Reinhart R, Erickson D, Rockwood A. (1992). Venous air embolism during radical retropubic prostatectomy. Anesth Analg. 74:151-153.
2. Albin MS, Ritter RR, Pruett CE, Kalff K. (1991). Venous air embolism during lumbar laminectomy in the prone position: Report of three cases. Anesth Analg. 73:346-349.
3. Robinson DA, Albin MS. (1987). Venous air embolism and cesarean sections. Anesthesiology. 66:93-94.
4. Albin MS. (1978). Venous air embolism and lumbar disk surgery (letter). JAMA. 240:1713.
5. Oppenheimer MJ, Durant TM, Lynch P. (1953). Body position related to venous air embolism and associated cardiovascular-respiratory changes. Am J Med Sci. 225:362-373.
6. Alvaran SB, Toung JK, Graff TE, Benson DW. (1978). Venous air embolism: Comparative merits of external cardiac massage, intracardiac aspiration, and left lateral decubitus position. Anesth Analg. 57:166-170.
7. Munson E, Merrick HC. (1966). Effect of nitrous oxide on venous air embolism. Anesthesiology. 27:783-787.
8. Toung TJ, Rossberg MI, Hutchins GM. (2001). Volume of air in a lethal venous air embolism. Anesthesiology. 94:360-361.
9. Martland HS. (1945). Air embolism. Fatal air embolism due to powder insufflators used in gynecological treatments. Am J Surg. 68:164-169.
10. Flanagan JP, Gradisar IA, Gross RJ, Kelly TR. (1969). Air embolus: A lethal complication of subclavian venipuncture. N Engl J Med. 281:488-489.
11. Hybels RL. (1980). Venous air embolism in head and neck surgery. Laryngoscope. 90:946-954.
12. Takeoka M, Sakai A, Ueda G, Ge RL, Panos RJ, Taniguchi S. (1996). Influence of hypoxia and pulmonary air embolism on lung injury in perfused rat lungs. Respiration. 63:346-351.
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