International Journal of Anaesthesia and Research
IJAR, 4(2): 171-173
www.scitcentral.com

ISSN: 2641-399X

Mini Review: Open Access

Venous Air Embolism (VAE) During Craniotomy of Supratentorial Meningioma in Supine Position: A Mini Review

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Received June 10, 2021; Revised June 18, 2021; Accepted June 21, 2021

ABSTRACT

Venous air embolism (VAE) is a serious and dangerous neurosurgical complication with higher incidence in procedures requiring the patient to be in sitting or semi-sitting position. VAE may also occur in the supine position, for example during craniotomy of supratentorial tumor. VAE occurs in the presence of pressure differential at two different venous systems, in this case, causes by negative pressure between the right atrium and the cranial venous sinuses. The emboli may cause hypoxemia as the result of ventilation-perfusion (V/Q) mal-distribution. A sudden drop in end tidal CO_2 level associated to hypotension in capnography is highly suggestive of VAE. Diagnostic tools that can be used to detect VAE are precordial Doppler, transesophageal echocardiography, CT-scan, expired nitrogen and pulmonary artery catheter (PAC). The primary goal in treatment of VAE is the prevention of further air entry and, if possible, a reduction in the volume of air entrained.

Keywords: Venous air embolism, VAE, Supine position, Craniotomy, Supratentorial tumor, Neuroanesthesia

Abbreviations: VAE: Venous Air Embolism; V/Q: Ventilation-Perfusion; PAC: Pulmonary Artery Catheter; RAC: Right Atrial Catheter; RAP: Right Atrial Pressure

INTRODUCTION

Venous air embolism (VAE) is a serious and dangerous neurosurgical complication with incidence ranges between 16% and 86%. This incidence found to be higher for procedures requiring the patient to be in sitting or semi-sitting position. The incidence of air embolism in the sitting position is variable but has been described in up to 45% of the cases. But VAE may also occur in patient with lateral decubitus, prone or supine position. The procedures more commonly associated with VAE are craniotomy in the sitting position, surgery of the posterior fossa, and craniosynostosis repair [1].

VENOUS AIR EMBOLISM (VAE) DURING NEUROSURGERY

VAE occurs in the presence of pressure differential at two different venous systems, in this case, causes by negative pressure between the right atrium and the cranial venous sinuses. When the cranial venous sinuses or venous system of the central nervous system is exposed to environmental pressure and there is a difference of at least 5 cm of H2O between the two sites, there will be air in flow [2]. The lethal dose of VAE is 3-4 ml/kg. The volume of air in venous system that triggers clinical manifestation is around 100ml in adult. Both volume and rate of air accumulation are dependent on the size of the vascular lumen as well as the pressure gradient. When the air gets into the circulation, it may lodge inside the superior vena cava and right atrium in which some of that air volume pass through the tricuspid

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Citation: Sutawan IBKJ, Bisri T, Rahardjo S, Lalenoh D, Jehosua BT, et al. (2021) Venous Air Embolism (VAE) During Craniotomy of Supratentorial Meningioma in Supine Position: A Mini Review. Int J Anaesth Res, 4(2): 171-173.

valve to the pulmonary artery. When the air volume is large enough, it may block the pulmonary arteries causing vasoconstriction and impairment of ventilation and also pulmonary perfusion ratio. The mechanism of this hypoxemia is the result of ventilation-perfusion (V/Q) mal-distribution. If the air volume obstructs the pulmonary artery flow, it may cause a drop in cardiac output which due to acute right heart failure or reduced left ventricular filling caused by decreased of pulmonary veins blood flow [3]. Large emboli may cause paradoxical (arterial) embolization by acutely increasing RAP, facilitating a right to left shunting through a patent foramen ovale or across the pulmonary capillary bed.

The clinical manifestation depends on patient's consciousness. If the patient is awake during the surgical procedure, VAE may manifest as coughing with arterial oxygen desaturation, dyspnea, chest pain, nausea and arterial hypotension with heart murmur in auscultation. If the patient is unconscious, there may be a sudden drop in end tidal CO_2 level associated to hypotension in capnography. In the post-operative period, patients may develop neurological impairment and cardiovascular disorder such as acute right heart failure, pulmonary hypertension, myocardial ischemia and pulmonary edema. The air bubbles in pulmonary microvasculature may also trigger the release of inflammatory cytokine and platelet activation which result in the presence of coagulopathy and thrombocytopenia [4]. Another diagnostic tool that can be used to detect VAE is precordial Doppler, transesophageal echocardiography, CT-scan, expired nitrogen and pulmonary artery catheter (PAC). Precordial Doppler can detect the presence of air in the blood and highly sensitive as compared to capnography and conventional hemodynamic monitoring for the diagnosis of VAE. The transducer should be placed in the right atrium when the patient is already in the final surgical position. A routine test should be done prior to starting the procedure: an amount between 0.25 and 1 mL of air or 3-5 mL of stirred saline solution is injected through the central catheter. The anesthesiologist should be familiar with the sound of the precordial Doppler when the air flows in to the heart cavities in order to have a reference prior to starting the procedure [5]. TEE is the most sensitive invasive method for diagnosing VAE which allow the diagnosis of small air volumes in the heart - between 0.01 and 0.19 mL/kg [6]. In the CT-scan, the presence of air in the dural venous sinuses, in the cortical vein or in the pterygoid plexus is diagnostic [4]. In expired nitrogen test, the presence of nitrogen in the expired gas monitor when the patient is breathing 100% oxygen is highly suggestive of VAE [1]. Increase in PAP caused by small amount of air can be detected through PAC. PAC offers early detection of VAE and has the possible advantage of offering prognostic information as to whether the surgical procedure should be continued or not [7].

POSITIONING DURING CRANIOTOMY

Some factors that associated with VAE are air volume, patient's position and the type of surgical procedure. The procedure with the highest risk of VAE occurrence is when the patient is required to be in the sitting or semi-sitting position; however, it is important to note that the fact of being in the supine position does not rule out the probability of VAE. Therefore, the sitting or semi-sitting position in neurosurgery are now rarely used. These positions are also prone to caused sciatic nerve injury, macroglossia, and tension pneumoencephalon, inter alia [8]. Neurosurgical operations performed in the lateral, supine, or prone positions have an incidence ranging from 15% to 25% [9]. The incidence of VAE in sitting position for cervical laminectomy is 10% and may be as high as 80% in seated posterior fosse surgery [10].

Venous air embolism in the prone position was first reported in 1969 during a craniotomy for a posterior fosse exploration in which the head was elevated 10 cm above the heart level and 10 cm H_2O of negative pressure was applied to the expiratory phase using a Bird ventilator [11]. In a case series of VAE in prone position reported by Albin et al, the critical factors related to the type of positioning used to secure the prone position are the use of the Hasting's (Canadian) frame and the four-poster frame which both decrease the intra-abdominal pressure and increase the inter-laminar space in the lumbar spine. In both positions, the abdomen hangs free and the lower extremities are dependent. Furthermore, the legs need to be wrapped to avoid pooling of blood. These case series indicate the need for serious consideration of complete monitoring for VAE whenever a patient is placed prone using any of the positioning techniques that allows for free movement of the abdomen [12].

Despite the highest incidence rates of VAE was occurred in neurosurgery with sitting position, VAE also can occur at the supine position as reported by a case report in 2017 [13]. It was reported that in neurosurgery in supine position may also had a big risk of sinus laceration. When the opened vein is failed to collapse, it may increase the risk of VAE. It only needs 5 cmH2O pressure differences to create suction effect in the open vein [8].

MANAGEMENT OF VAE

The primary goal in treatment of VAE is the prevention of further air entry and, if possible, a reduction in the volume of air entrained. The first thing to do is to inform the neurosurgeon to start the irrigation of the surgical field and provide coverage to any blood vessels that may be exposed. Then 100% oxygen should be used and always avoid using nitrous oxide or any air/oxygen mixtures. Repositioning of the patient is also important, if possible, the left lateral decubitus (Durant maneuver) is preferring therefore the air bubbles move toward the right atrium. If the patient has a

central venous catheter, any air lodged between the superior vena cava and the right atrium should be aspirated [14].

Transient bilateral jugular veins compression is done to reduce the inflow of air through the exposed venous sinuses. By decreasing the cerebral venous flow, the venous retrograde flow increases and the inflow of air is interrupted. However, this technique is controversial because it may increase the intracranial pressure, compress the carotid arteries and reduce brain perfusion [8].

A right atrial catheter (RAC) should be standard monitoring for cases with a high incidence of VAE such as seated craniotomy and possibly seated cervical laminectomy. The RAC is used for accurate measurement of RAP, central administration of vasoactive drugs, and to aspirate air from near the RA superior vena cava (WC) junction. Because the mechanism of cardiovascular collapse is right ventricular obstruction and impairment of forward flow, pharmacologic support should include isotropic drugs such as epinephrine to improve CO^2.

CONCLUSION

Venous air embolism (VAE) is a serious and dangerous neurosurgical complication with sitting position being the most common risk factor. Diagnostic tools that can be used to detect VAE are precordial Doppler, trans-esophageal echocardiography, CT-scan, expired nitrogen and pulmonary artery catheter (PAC). The primary goal in treatment of VAE is the prevention of further air entry and, if possible, a reduction in the volume of air entrained.

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