Neuroanesthesia and Intensive Care

Successful management of venous air embolism with inotropic support

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Purpose: Since venous air embolism may occur during many different types of surgery, management of this clinical emergency can be required in patients who do not have a previously established central venous access for aspiration of air. Recent reviews suggest that management of right heart syndromes in patients with embolism is critical in improving outcome.

Clinical Features: Abrupt decreases in oxygen saturation (from 98% to 40%) and end-tidal carbon dioxide tension (from 24 to 6 mm Hg), compatible with venous air embolism were observed in a 73-yr-old woman during craniotomy for meningioma in the supine position. Since no access for aspiration of air was readily available, therapy was directed at inotropic support of the right heart using a bolus of ephedrine. Cardiorespiratory variables rapidly returned to normal, and the patient recovered from anesthesia and surgery without sequelae.

Conclusions: Venous air embolism places an acute load on the right ventricle and may provoke right heart failure, even in the absence of total cardiovascular collapse. Treatment that supports right heart function may allow sufficient time for redistribution of embolized air and produce a good outcome when access for central aspiration of air is not available.

Objectif : Comme l'embolie gazeuse veineuse peut survenir pendant de nombreuses interventions chirurgicales de type différent, le traitement de cette urgence clinique peut être nécessaire chez des patients qui n'ont pas d'accès veineux central préétabli pour l'aspiration de l'air. Des révisions récentes suggèrent que le traitement des syndromes du cœur droit soit crucial pour l'évolution favorable des patients qui présentent une embolie.

Éléments cliniques : La baisse brutale de la saturation en oxygène (de 98 % à 40 %) et de la pression de fin d'expiration du gaz carbonique (de 24 à 6 mmHg), compatibles avec une embolie gazeuse veineuse, ont été observés chez une femme de 73 ans pendant une craniotomie pour l'ablation d'un méningiome, en décubitus dorsal. Aucun accès pour l'aspiration de l'air n'étant rapidement disponible, la thérapie a consisté en une assistance inotrope du cœur droit en utilisant un bolus d'éphédrine. Les variables cardiorespiratoires sont revenues rapidement à la normale et la patiente a connu une récupération anesthésique et chirurgicale sans séquelles.

Conclusion : L'embolie gazeuse veineuse provoque une brusque surcharge du ventricule droit et peut entraîner une défaillance du cœur droit, même en l'absence de collapsus cardiovasculaire. Le traitement qui consiste en un soutien de la fonction du cœur droit peut laisser suffisamment de temps pour la redistribution de l'air embolisé et donner un bon résultat lorsque l'accès veineux central pour l'aspiration de l'air n'est pas disponible.

CATASTROPHIC venous air embolism (VAE) requires rapid detection and treatment to prevent cardiovascular collapse. The application of end-tidal carbon dioxide monitoring as a standard of care for patients receiving general anesthesia provides a sensitive and specific method to detect the pulmonary consequences of VAE.¹ Emergency treatment focuses on supporting the circulation until the acute effects of the embolism abate and on removing the air, if possible, through an appropriately positioned multi-orifice central venous catheter.^{1,2} It is common practice to place central venous catheters in those patients undergoing procedures that are at high risk for air embolism such as neurosurgical procedures in the sitting position. However, the variety of procedures during which venous air embolism has been reported is extremely wide.² Thus, the anesthesiologist may be faced with management of venous air embolism in patients without pre-established central venous access. Recent reports suggest that immediate interventions directed at supporting the right heart might be beneficial for patients who experience acute embolism with air, thrombus, or amniotic fluid.²⁻⁴

We present a case of presumed venous air embolism in a patient undergoing craniotomy without previously established central venous access successfully treated with ephedrine.

Case report

A 73-yr-old, 54 kg woman, ASA 2, received general anesthesia for craniotomy for removal of an incidental fronto-parietal meningioma. The patient presented with mild speech and memory impairment that was investigated without finding a specific cause. Magnetic resonance imaging studies revealed a parasagittal 3 x 2.5 cm convexity lesion consistent with a meningioma, in the left frontal lobe. The sagittal sinus was intact. The patient requested that the lesion be removed. Preoperatively, the patient was receiving treatment with L-thyroxine (0.05 mg daily) for hypothyroidism and with latanaprost (prostaglandin F_{2c} analogue) and levobunolol (non-cardioselective beta-adrenoreceptor blocking agent) eye drops for glaucoma.

Monitoring included ECG, pulse oximetry (sPO₂), end-tidal carbon dioxide partial pressure (P_{ET}CO₂), and non-invasive blood pressure (NIBP). A right radial arterial cannula was inserted for direct blood pressure measurement and arterial blood sampling. Anesthesia was induced with 4 µg·kg⁻¹ fentanyl and 5 mg·kg⁻¹ thiopental. Tracheal intubation was facilitated with 0.09 mg·kg⁻¹ rocuronium and 2 mg·kg⁻¹ succinylcholine. Cefazolin sodium, 2 g, and 10 mg dex-

TABLE Arterial blood gas tensions

	Time			
	08:55	10:17	10:25*	10:38
pH	7.46	7.46	7.39	7.38
PCO ₂ , mm Hg	33	32	38	40
PO ₂ , mm Hg	110	79	28	85
HCO ₃ meq·l ⁻¹	23	22	23	23
FiO ₂	0.3	0.3	1.0	1.0

*Sample taken before injection of ephedrine

amethazone were administered intravenously shortly after induction of anesthesia. Anesthesia was maintained with isoflurane and fentanyl. The lungs were mechanically ventilated using a tidal volume of 10 ml·kg⁻¹ at a frequency of 10 breath·min⁻¹. Fresh gas was oxygen/air (FiO₂ = 0.3), provided at a rate of 1 l·min⁻¹ to a semi-closed circle circuit with a carbon dioxide absorber in-line. The patient was positioned supine in the lawn-chair posture with a 15 head up tilt.

Anesthesia and surgery proceeded uneventfully until two hours into the procedure, when the anesthesiologist noted a transient decrease in arterial pressure, sPO₂ and P_{ET}CO₂ that could not be explained on the basis of anesthetic or surgical conditions (Figure at 09:55). At this time, the neurosurgeons were proceeding with microdissection of the lesion that was located on the surface of the brain, requiring little retraction. Control of bleeding points was meticulous, with minimal estimated blood loss for the previous 45 min. Search of the operative field by the surgical team did not reveal any sources for VAE. The episode resolved without any specific management and the surgical team continued with microdissection of the tumor. Twenty-two minutes later a progressive and profound depression of both sPO₂ and P_{ET}CO₂ occurred (Figure at 10:17-10:25). Blood pressure initially showed a very slight increase during this period and then began to decline. Heart rate at 10:17 was 55 beat·min, increasing to 65 beat·min at 10:25. The differential diagnosis at this time included pulmonary embolism with air or thrombus, cardiac arrhythmia, pneumothorax, displacement of the endotracheal tube with a failure of ventilation and a mechanical malfunction of the anesthesia machine or circle breathing system. Auscultation of the chest by one of the authors (MPP) revealed bilaterally symmetrical breath sounds and no heart sounds or murmurs. Normal sinus rhythm was maintained throughout the event and peak airway pressures remained unchanged. A physical check of the anesthesia machine, breathing tubes and the patient was performed to confirm that the lungs were being appropriately ventilated. A pre-

FIGURE The graphical trends recording from the patient's monitor showing (A) $P_{ET}CO_2$, (B) sPO_2 , and (C) systolic, mean and diastolic pressure during two episodes of presumed venous air embolism starting at 09:55 and 10:17. Ephedrine (25 mg) was rapidly injected intravenously at 10:26 (X). In panel A, the inspired CO_2 is 0 throughout and is shown by a line along the abscissa.

sumptive diagnosis of VAE was made and the surgical team informed. The patient was positioned level and the surgical field flooded with irrigation solution. Fresh gas flow was increased to 6 l·min⁻¹ oxygen and air was discontinued. At 10:26, with no signs of improvement in sPO_2 and $P_{ET}CO_2$, a 25 mg bolus of ephedrine was

rapidly injected intravenously. Immediately arterial blood pressure (Figure) and heart rate increased (from 65 to 80 beat·min⁻¹); recovery of sPO_2 and $P_{ET}CO_2$ followed (Figure). Although no definitive source for VAE was discovered, when the head was returned to the neutral position, a bleeding site in the dura under the bone

margin was identified and cauterized. After 20 min observation to ensure continued patient stability, the surgical procedure was completed uneventfully. The postoperative course was complicated only by mild transient dysphasia that the neurosurgeon thought was explainable on the basis of the location of the surgery and the lesion.

Discussion

With the advent of transesophageal echocardiography (TEE), reports concerning the early findings after acute venous embolism are starting to emerge, emphasizing the role of acute right heart failure in venous embolic syndromes.^{3,4} Venous gas embolism has recently been reviewed.² Acute pulmonary embolism causes an abrupt increase in pulmonary vascular resistance, leading to right ventricular overload with increased intraventricular pressures and ventricular distension. Left ventricular output may subsequently decrease, due to a combination of decreased pulmonary venous return and right-to-left shift of the intraventricular septum. If peripheral vasoconstriction is insufficient to compensate then systemic hypotension may ensue. Although there are differences in the pathophysiological consequences of acute pulmonary embolism, depending upon the nature of the embolic material, Schmidt and Wood⁴ have recently summarized the critical care management of right heart syndromes that occur as a result of a variety of precipitating events. They propose⁴ that treatment of right heart syndromes be directed at maintaining the mean arterial pressure to provide adequate perfusion to the strained right ventricle and inotropic support for the right ventricle during the acute post-embolic period. Dobutamine, which interacts with both α_1 - and β_1 -adrenoreceptors,⁵ has been recommended⁴ as the drug of first choice for all right heart syndromes, in an initial dose of $5 \mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$, increasing the dose by $5 \mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ every 10 min until a satisfactory response is achieved. In the event of a failure to respond to dobutamine, the addition or substitution of norepinephrine infused at a rate of 0.4 to $4 \mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ has been suggested.⁴ In the present case, a moderately large dose of ephedrine was given for immediate support while a dobutamine infusion was being prepared. Ephedrine was specifically selected over phenylephrine (an α_1 -selective adrenergic agonist)⁵ because of the inotropic properties of ephedrine, an α - and β -adrenergic agonist, which in addition enhances norepinephrine release from sympathetic nerve endings.⁵ The heart rate response to the ephedrine was probably blunted by the systemic effects of the levobunolol eye drops. Fortunately, the patient recovered after ephedrine administration and no dobutamine was required.

Current neuroanesthesia textbooks^{1,6} emphasize that the treatment of VAE is based upon early detection, rapid aspiration from a central venous catheter, and sealing off the source of air entry. The published literature concerning the human responses to inotropic therapy in acute embolic syndromes is very limited: our literature search failed to identify any relevant articles in the three years since the publication of Schmidt and Wood's textbook chapter devoted to right heart syndromes.⁴ The recommendations of Schmidt and Wood⁴ which prompted the authors to try inotropic therapy with ephedrine are based primarily on experiments in animal models of pulmonary embolism (exemplified by the work published by Prewitt's laboratory^{7,8}) and clinical studies in patients with pulmonary thromboembolism.⁹ As the availability of TEE in the operating room increases, we expect that more information concerning the human pathophysiological consequences of acute venous air embolism and its treatment will be reported.

In summary, we have presented the case of patient undergoing craniotomy who showed cardiorespiratory findings that were suggestive of acute venous air embolism. Inotropic treatment with ephedrine appeared to rapidly reverse the pulmonary circulatory abnormalities. Early consideration should be given to inotropic support of the right ventricle when VAE is suspected.

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