Neuroanesthesia and Intensive Care

Venous air embolism during awake craniotomy in a supine patient

[Aéroembolie veineuse pendant la craniotomie chez un patient en décubitus dorsal]

Mrinalini Balki MBBS MD,* Pirjo H. Manninen MD FRCPC,* Glenn P. McGuire MD,*
Hossam El-Beheiry MBBCH PHD FRCPC,* Mark Bernstein MD FRCSC†

Purpose: To report a non-fatal case of intraoperative venous air embolism (VAE) during an awake craniotomy. VAE presented with unusual clinical features.

Clinical features: VAE during an awake craniotomy has not been reported frequently. The patient we describe presented with persistent coughing followed by tachypnea, hypoxia and reduction in end-tidal CO_2 during dural opening while undergoing an awake craniotomy in the supine position. Cardiovascular variables were stable during the episode except for transient hypertension. Having ruled out airway obstruction and low cardiac output, we concluded that air embolism was the cause. The patient responded immediately to the standard treatment of air embolism and recovered without any complication.

Conclusion: This case illustrates a VAE during an awake craniotomy and emphasizes the importance of early diagnosis in the management.

Objectif : *Présenter un cas non mortel d'aéroembolie veineuse peropératoire (AEV) survenue pendant une craniotomie vigile. L'AEV présentait des caractéristiques cliniques inhabituelles.*

Éléments cliniques : *L'AEV qui survient pendant la craniotomie vigile n'a pas été souvent rapportée. Le patient que nous décrivons a présenté une toux persistante suivie de tachypnée, d'hypoxie et d'une baisse du CO_2 télé-expiratoire pendant l'ouverture durale pour une craniotomie vigile en décubitus dorsal. Les variables hémodynamiques ont été stables pendant l'épisode, sauf pour l'hypertension transitoire. Nous avons d'abord écarté la possibilité d'une obstruction des voies aériennes et un faible débit cardiaque pour en arriver au diagnostic d'embolie gazeuse. Le patient a immédiatement réagi au traitement standard de l'embolie gazeuse et il s'est remis sans complication.*

Conclusion : *Ce cas illustre un AEV pendant une craniotomie vigile et souligne l'importance d'un diagnostic précoce pour le traitement.*

VENOUS air embolism (VAE) is a well-known potential complication in procedures where the operative field is above the level of the heart. The occurrence of a VAE has been reported in a variety of neurosurgical cases performed under general anesthesia.[1–3] However, its incidence in conscious patients during an awake craniotomy has not been widely discussed or reported.[4] An awake craniotomy is usually performed in order to allow for neurological assessment of the patient during the resection of a lesion close to areas of eloquent brain function.[5] During an awake craniotomy, the diagnosis of a VAE may be difficult because of the use of limited monitoring and the lack of a high index of suspicion.

Case report

A 61-yr-old male patient, weighing 78 kg was admitted for excision of a dominant hemisphere tumour. The patient presented with a one-month history of right leg weakness. His past medical history revealed diet controlled diabetes and hypertension treated with ramipril. Airway assessment and physical examination were normal. Laboratory findings were unremarkable.

Electrocardiography showed left axis deviation. Magnetic resonance imaging suggested a probable malignant brain tumour in the left premotor area.

From the Department of Anesthesia* and the Division of Neurosurgery,† The Toronto Western Hospital, University Health Network, University of Toronto, Toronto, Ontario, Canada.

Address correspondence to: Dr. Pirjo H. Manninen, Department of Anesthesia, The Toronto Western Hospital, 399 Bathurst Street, Toronto, Ontario M5T 2S8, Canada. Phone: 416-603-5118; Fax: 416-603-6494; E-mail: Pirjo.Manninen@uhn.on.ca

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An awake craniotomy was planned in order to perform cortical mapping before resection of the tumour. Routine monitors included use of the electrocardiogram (ECG), pulse oximeter, non-invasive blood pressure and end-tidal carbon dioxide ($EtCO_2$) sampling via a nasal cannula. Oxygen (O_2) administration was by nasal prongs at the rate of 3 $L \cdot min^{-1}$. Initial vital signs included: blood pressure 140/90 mmHg, heart rate 65 $beats \cdot min^{-1}$ and oxygen saturation (SpO_2) 99%. The patient was placed supine on the operating room table in a comfortable position with 20° head-up tilt. Intravenous sedation was started with midazolam 2 mg, fentanyl 50 μg and an infusion of propofol. As a frameless navigation system was used, a head frame was applied after infiltration of the scalp with 0.25% bupivacaine containing 1:200,000 epinephrine. The conscious sedation technique was then continued with an infusion of propofol (75–150 $\mu g \cdot kg^{-1} \cdot min^{-1}$) and additional fentanyl increments of 25 μg (total 150 μg) when required during the procedure.

An hour after the start of surgery, during dural opening, the patient suddenly started coughing continuously. He was conscious and able to respond appropriately to verbal commands. The infusion of propofol was turned off and no more sedation was given. No initial cause could be found for this unexpected coughing. There was a negative preoperative history of any respiratory problems including smoking. After about ten minutes the $EtCO_2$ dropped from 28 to 16 mmHg, the SpO_2 decreased to 90% and the respiratory rate increased to $32 \cdot min^{-1}$ (Figure). At this point the hemodynamic variables were stable with a blood pressure of 134/84 mmHg and a heart rate of 64 $beats \cdot min^{-1}$. On further examination of the patient, there was no evidence of upper airway obstruction or any sign of respiratory distress like cyanosis, stridor, intercostal/subcostal indrawing, agitation or change in the level of consciousness. Auscultation of the chest revealed normal heart and breath sounds. ECG showed normal sinus rhythm and ST segments. Over the next ten minutes, the $EtCO_2$ and respiratory rate were unchanged but there was a further reduction in SpO_2 to 75% and coughing continued. At this time, the blood pressure started to rise and reached 160/90 mmHg. Heart rate was stable between 60 to 70 $beats \cdot min^{-1}$. There was no evidence of the usual bleeding at the surgical site indicating that the venous pressure might be lower than the atmospheric pressure. A presumptive diagnosis of VAE was made and the surgical team informed. Immediately, the patient was placed in a Trendelenburg position and O_2 was provided with a facemask. Coughing and hyperventilation stopped at once. Venous oozing was seen from the bone margin. The surgical field was flooded with normal saline and a saline soaked gauze held over the craniotomy site. Bleeding from the bone margin was controlled with the application of cautery and bone wax. Blood pressure returned to 130/80 mmHg within five minutes. $EtCO_2$ returned back to normal gradually over the next 20 min but SpO_2 remained at 94%. After a few minutes of observation to ensure continued patient stability, the propofol infusion was restarted to maintain a light level of sedation and the surgical procedure was completed uneventfully. On discharge from the postanesthesia care unit, the patient required 50% O_2 with a facemask to maintain SpO_2 of 95% overnight. The patient was discharged from the hospital after a week without any neurological deficits or any other adverse event.

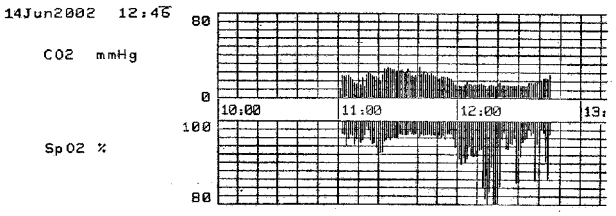

FIGURE Intraoperative tracing of the end-tidal carbon dioxide ($EtCO_2$) and oxygen saturation (SpO_2). At the time of the suspected venous air embolism there was a decrease in $EtCO_2$ from 28 to 16 mmHg.

Discussion

The incidence of VAE is known to be higher in neurosurgical procedures performed in the sitting position (10–80%).[1–3,6] In our hospital the prevalence of VAE in awake craniotomies was found to be 0.64% (3/470) during the last 11 years.[A] A search of the literature revealed only one case report on VAE in an awake craniotomy.[4] The risk of VAE is increased with the transmission of negative intrathoracic pressure to the central veins. Thus it is more likely to occur with spontaneous breathing, deep inspiration, dyspnea, hypovolemia and in the upright position.[1,6,7] The factors that determine the morbidity and mortality of any episode of VAE include the rate of air entrainment, the volume of air entrained and the position of the patient at the time VAE occurs.[2]

A Personal communication, Mark Bernstein, MD.

The predominant symptoms in our patient were coughing followed by tachypnea, hypoxemia and a decrease in $EtCO_2$. The patient was lightly sedated and responding to verbal commands without any alteration in his level of consciousness. There was no evidence of oropharyngeal secretions and no sign of upper airway obstruction. The patient denied history of any respiratory problems and his chest was clear to auscultation. Thus upper and lower airway obstruction, which could lead to coughing and hypoxemia, were ruled out. Other conditions reflecting changes in $EtCO_2$ and SpO_2 as a result of low cardiac output states due to pulmonary thromboembolism or fat embolism, myocardial ischemia, pulmonary edema and hemorrhage were unlikely in this situation. The patient had no complaints of chest pain, dyspnea, dysphoria or any evidence of hemodynamic instability with changes in ECG.

VAE in this patient was suspected by the development of a persistent decrease in $EtCO_2$, hypoxemia and respiratory irregularities. As is common in our institution the patient was positioned supine with elevation of the head thus increasing the gradient between the surgical site and the right atrium and encouraging air entry. There was no oozing or the usual bleeding from the operative site when the event happened. This suggests that air might be going in through venous conduits that do not collapse easily such as diploic veins in the bone margin due to a favourable hydrostatic gradient.

The pathophysiological consequences of air embolism are well known. Air emboli lodged in the pulmonary vessels cause sympathetic reflex vasoconstriction leading to ventilation-perfusion abnormalities with increase in the physiological dead space. This results in decreased $EtCO_2$, increased $PaCO_2$ and hypoxemia.[1–3,6] Microvascular bubbles also activate the release of endothelial mediators including excessive complement production, cytokine release and production of reactive O_2 molecules.[6,8] Whether these factors contributed to coughing in our patient is unclear. Late sequelae, when a large volume of air is entrained quickly, include persistent pulmonary perfusion deficits, frank pulmonary edema and adult respiratory distress syndrome.[6,8] There was no evidence of these late sequelae in our patient except for the decrease in SpO_2 postoperatively.

A small amount of air entrained may be of little consequence, however, if the air is entrained rapidly or its cumulative volume is large, changes in cardiovascular variables in the form of hypotension and tachycardia will be manifested. These hemodynamic effects can be explained on the basis of diminished cardiac output due to decreased pulmonary venous return occurring as a result of increased pulmonary vascular resistance and right ventricular overload caused by VAE.[1–3,6,9] The hemodynamic variables were stable in this case except for transient hypertension. The rise in blood pressure could be because the patient was awake and distressed due to cough and sedation had been stopped. In contrast, in the case report described by Scuplak *et al.*,[4] the patient presented with hypoxemia and chest pain with ST-T changes on ECG. Treatment was initiated on the presumptive diagnosis of myocardial ischemia. Later, when reduction in $EtCO_2$ was noticed on capnography, the diagnosis of air embolism was made. The patient subsequently developed complications in the form of pulmonary edema and pleural effusion.

VAE can produce a broad array of signs, hence monitoring and early diagnosis is essential to prevent the consequences. Monitoring in awake patients tends to be less comprehensive with more reliance on verbal communication than technology.[10] The precordial Doppler is a standard for VAE, however its use in awake craniotomy has not been described.[2,10]

Rapid treatment of a VAE is important to decrease morbidity and mortality. Immediate change to a Trendelenburg position will increase venous pressure at the operative site and reduce air entrainment, as was done in this patient. Emergency treatment may also focus on inotropic support of the right ventricle to allow sufficient time for redistribution of embolized air and on removing the air if possible through an appropriately positioned central venous catheter.[1–3,6,9] Ephedrine, dobutamine and norepinephrine have been recommended for right heart syndromes.[9] Since there were no significant hemodynamic effects in this patient, pharmacological treatment was not required.

In conclusion, VAE may occur in patients undergoing an awake craniotomy. Its presentation may take different forms such as coughing, dysrhythmias, hypotension or chest pain along with persistent hypoxemia and reduction in end-expiratory CO_2. The anesthesiologist must be vigilant for such unusual signs and symptoms. Perhaps consideration should be given to the use of a precordial Doppler in these patients. Early suspicion and recognition of VAE and institution of aggressive treatment will help to avoid fatal consequences in such cases.

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