

CASE REPORT

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Temporal insular glioma—rare case report for a venous air embolism

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Abstract

Background: Venous air embolism (VAE) is a well-known entity in the field of neuroanesthesia, with established surgeries and positions where its occurrence is high. The insular cortex is a deep area within the lateral sulcus, and surgeries in this area are not reported to develop VAE.

Case presentation: A young male being operated on the insular cortex developed VAE, had cardiac arrest, was revived, and was extubated with no residual deficit due to the untoward event.

Conclusions: An anesthesiologist should always be prepared for this complication due to its rapid presentation and fatal consequences.

Keywords: Venous air embolism, Temporal insular glioma, Phenytoin

Background

Venous air embolism (VAE) has been described as “the most uncontrollable cause of sudden death.” VAE is a well-known entity and has always kept us anesthesiologists on the edge due to its rapid presentation and fatal consequences.

VAE is the entrainment of air (or exogenously delivered gas) from open operative field or communication with the environment into the venous or arterial vasculature, producing systemic effects. The venous air embolism is air entry in systemic venous circulation reaching the right ventricle while arterial air embolism occurs due to the entry of air into the arterial circulation and is potentially life-threatening as it can lead to a circulatory deficiency in the body organ with poor collateral circulation (Shaikh and Ummunisa 2009).

Certain positions and surgeries are notorious for the occurrence of VAE like sitting craniotomies, infratentorial surgeries, posterior cervical spine surgeries, and craniostomy repairs. However, it may also occur in patients in lateral, supine, or prone (Vinay et al. 2013).

VAE is fairly rare in other surgeries but still reported in cases of cesarean sections and even urological surgeries. We as anesthesiologists are independently responsible for the development of VAE during procedures like insertion and removal of central venous catheter and epidural catheter insertion using loss of resistance to air technique (Mirski et al. 2007).

The insular cortex is a deep area within the lateral sulcus; shrouded by frontal, frontoparietal, and temporal opercula; and is surrounded by several white matter tracts and critically important vascular structures (Uddin et al. 2017). This is the first reported case of VAE in the location of the insular region.

Case presentation

A written informed consent was taken from the patient for publication of this case report. We report a case of a 23-year-old ASA physical status I male, with a history of intermittent headache. On imaging, computerized tomography showed a lesion measuring 6 mm × 5 mm × 7 mm in the left insular cortex. A surgical excision of the tumor was planned. Preoperative evaluation and laboratory investigations were all within normal limits. An informed high-risk consent was taken, and the patient was admitted inside the operation theater with a GCS of

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E4V5M6. Baseline vital parameters were pulse rate (HR) 78 beats per minute, non-invasive blood pressure (BP) 130/89 mmHg, and pulse oximetry (SpO_2) reading 100%. Intravenous access was obtained, and standard induction of anesthesia was done with fentanyl citrate (2 mcg/kg), propofol (1.5 mg/kg), and rocuronium bromide (1 mg/kg). A cuffed endotracheal tube size 8 mm internal diameter was placed under direct laryngoscopy, bilateral air entry checked, and fixed at a 20-cm mark.

Under all aseptic precautions, the left radial artery for invasive blood pressure and arterial blood gas monitoring was cannulated. The right subclavian vein was also cannulated and fixed at 13 cm for central venous pressure monitoring and guided fluid therapy.

The surgery was proceeding uneventfully with a blood loss of around 400 ml. While the surgeon was still excising the tumor, the patient's invasive blood pressure dropped from 135/72 to 80/38 mmHg. While the arterial line was being flushed, it was noticed that the plethysmograph trace was not recorded, heart rate dropped to 52 bpm, and the end-tidal carbon dioxide ($etCO_2$) dropped from 32 to 16 mmHg. The ongoing phenytoin infusion was stopped. The surgeon was informed to stop the surgery and flush the field with saline-soaked gauzes, and FiO_2 was increased to 100%. An attempt to give pressure on the jugular vein made us realize that the carotid pulse was also absent. A Trendelenberg position was done, and simultaneously, cardiopulmonary resuscitation was started according to the ACLS guidelines. Inj. adrenaline 1 mg was given and flushed with 20 ml saline. An experienced anesthesiologist was delegated the responsibility to aspirate blood from the in situ central venous catheter. A left lateral tilt was given in accordance with Durant's position. Now, about 7–8 ml of air was aspirated, and soon, all the vital parameters returned to normal, HR 128 bpm, BP 140/98 mmHg, SpO_2 100%, and $etCO_2$ 30 mmHg, and a palpable carotid pulse was felt. CPR was stopped and vital parameters checked again. The chest was auscultated for normal breath and cardiac sounds. A sample for ABG was sent immediately which showed pH 7.19, pCO_2 50 mmHg, pO_2 137 mmHg, HCO_3^- 19 $mmolL^{-1}$, lactate 6.4 $mmolL^{-1}$, and Hb 13.4 gm%. Corrective measures were done, and the surgeon was asked to resume the surgery in the supine position.

Thereafter, the surgery went uneventful with a total blood loss of 650 ml and urine output 1500 ml (pre-arrest 1000 ml and post-arrest 500 ml). The patient was shifted intubated to the neurosurgical ICU for further monitoring, assessment, and post-cardiac arrest care. ECG, chest X-ray, and ABG reports were within normal limits. He regained consciousness and was extubated after 5 h of surgery. Post-extubation GCS was 15/15, and ABG was within normal limits. He was kept in the

ICU for observation for another 24 h and then shifted to the ward for another 3 days and discharged thereafter.

Discussion

VAE is a well-read and reported complication. Various invasive and non-invasive techniques have been used for its detection. Transoesophageal echocardiography is the most sensitive (0.02 ml/kg of air) method used for the detection of VAE, but its high sensitivity has made it highly non-specific for emboli that may cause cardiovascular compromise (Mirski et al. 2007; Uddin et al. 2017; Jaffe et al. 1995).

Other techniques are transcranial Doppler, precordial Doppler, end-tidal nitrogen and carbon dioxide levels, ECG, and oxygen saturation. Out of these, $etCO_2$ is the most practical and sensitive modality used. A change of 5 mmHg in a time span of 5 min is diagnostic of VAE (Mohd Nazaruddin et al. 2013).

The most important factor for the sequel of VAE after its occurrence is the rate and volume at which the air enters the vasculature. More than 5 ml/kg immediately causes an air-lock scenario and hence right heart failure followed by cardiac arrest. Any value less than this causes a spectrum of signs and symptoms ranging from dyspnoea, wheezing, coughing, and chest pain in awake patients to rapid fall in $etCO_2$, SpO_2 , BP, HR, ST segment and T wave changes, myocardial infarction, and arrest in anesthetized patients. Principles of management of VAE are preventing any further air entrainment, decreasing the size of the already entered air emboli and hemodynamic support (Mirski et al. 2007). Therefore, the surgeon should immediately flood the field with saline and seal any open sinuses, diploic veins in the bones of the cranium in order to prevent any further air entrainment. Attempts to increase the central venous pressures by pushing intravenous fluids and giving Durant's position (head low and right up) will help to remove the air emboli from the right ventricle outflow tract, hence maintain circulation (Durant et al. 1947). It is advised to place the tip of the central venous catheter approximately 1–3 cm above the sino-atrial node in order for it to be able to aspirate the air emboli (Schlichter and Smith 2016). Intermittent jugular venous compression is also advised to prevent further air entry (Lossaso et al. 1992).

We report this case as our patient was being operated on the insula, which is located in the supratentorial region and is away from the major sinuses. This cortex also called the "island of Reil" is deeply located within the lateral sulcus (Uddin et al. 2017). Blood supply is from the M2 segment of the middle cerebral artery and drains into the deep middle cerebral vein (Mavridis 2014). Up to 25% of low-grade and 10% high-grade gliomas are found in this region. Resection is technically challenging because of the deep-seated lesion. Reports of VAE from this area could not be found.

Though the aspiration of air from the central venous catheter was in averment with the diagnosis of VAE, another etiology of the intraoperative events could be the ongoing phenytoin drip; 1000 mg of phenytoin sodium was loaded in a 250-ml of normal saline and started at a slow rate of about 10 ml/min (40 mg/min). Phenytoin is a known antiepileptic drug and a class IB antiarrhythmic drug. It affects the atrio-ventricular nodal conduction by either enhancing or delaying it, thus causing tachyarrhythmias or bradyarrhythmias (Wheless 1998; Su et al. 2009). It is advised that the patients receiving intravenous phenytoin be closely observed, and the drug is not administered at a rate of more than 1–2 mg/kg/min (Parsai et al. 2016).

Conclusions

Though VAE is associated with certain surgeries and positions, still, an anesthesiologist should always keep it in mind in other cases too in order to act rapidly and prevent any end-organ damage. Other differential diagnoses should also be considered before concluding it as a VAE.

Abbreviations

VAE: Venous air embolism; ASA: American Society of Anesthesiologists; GCS: Glasgow Coma Scale; Bpm: Beats per minute; ACLS: Advanced cardiac life support; CPR: Cardiopulmonary resuscitation; ICU: Intensive care unit; ABG: Arterial blood gasses

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Authors' contributions

SJ conducted the case and wrote the manuscript. DK, NC, and MK assisted in the case and did the research work. All the authors have read and approved the final manuscript.

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Ethics approval and consent to participate

Ethics approval is not required, and consent was taken.

Consent for publication

Written informed consent was taken from the patient for the publication of this case.

Competing interests

The authors declare that they have no competing interests.

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