**Case Report**

**Venous Air Embolism During Neurosurgery in Sitting Position**

Brihaspati KC1, Surendra Bhusal1, Kaushal Tamang2, Diwan Shrestha3

1Department of Anesthesiology, National Academy of Medical Sciences, Kathmandu, Nepal

2Department of Anesthesiology, Sukraraj Tropical & Infectious Disease Hospital, Kathmandu,Nepal

3Department of Surgery, Bhaktapur Cancer Hospital, Bhaktapur, Nepal

Correspondence: Brihaspati K C, Nepal, National Academy of Medical Sciences, Mahaboudha, Kathmandu,

Email address: [kcbireshh@gmail.com](mailto:kcbireshh@gmail.com)

**Summary**

Venous air embolism (VAE) is a potentially fatal condition that occurs when air entersinto the vasculature and can be lethal. VAEs can occur in any neurosurgical procedure. Seated neurosurgical procedures carries higher risk for development of VAE. We report a 17 years/Male with diagnosis of Pineal region tumor with no known co-morbidities, who underwent Infra-tentorial Supra-cerebellar Occipital Craniotomy and excision of tumor in sitting position under General anesthesia. During the procedure after opening of dura and exploration of the mass the patient developed hemodynamic instability with low EtCO2, bradycardia and hypotension. With suspicion of venous air embolism blood aspirated through the central venous catheter which revealed bubbles of air. Bradycardia and hypotension responded to Atropine. Surgeons were informed immediately and surgical field was irrigated with normal saline and covered with wet gauzes, position of the patient was changed to supine.

**Keywords:** venous air embolism, case report, neurosurgery, neuro-anesthesia

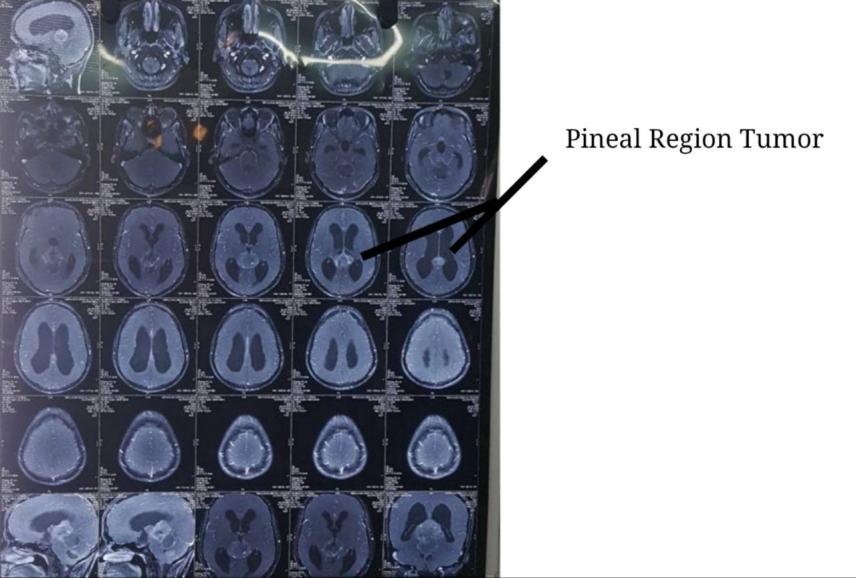
**Introduction**

Venous Air Embolism (VAE) is defined as introduction of air into the systemic venous system. VAE can occur in any procedure that involves the vasculature to be freely open with the environment with a pressure gradient[[1]](#endnote-2). It has been reported since 19th century[[2]](#endnote-3). VAE can occur during neurosurgical procedures, laparoscopic surgeries, central venous catheterization, blunt chest trauma, high-pressure mechanical ventilation, thoracocentesis, hemodialysis, and several other invasive vascular procedures. The incidence of VAE ranges between 16% and 86%[[3]](#endnote-4),[[4]](#endnote-5). However many cases of VAE are subclinical with no adverse outcome and thus go unreported. Cases of VAE without any clinical sequelae have also been reported[[5]](#endnote-6).

All kind of neurosurgery carries risk of developing VAE however neurosurgery performed in a sitting position carries the highest risk of VAE, with an incidence of about 45% of the cases[[6]](#endnote-7).Whereas, neurosurgical procedures in lateral, supine, or prone positions showed an incidence of 15% to 25%[[7]](#endnote-8). VAEs is also seen in pediatric neurosurgery with incidence of (0.42-9.8%)[[8]](#endnote-9),[[9]](#endnote-10). VAE can also occur during gynaecological-obstetric procedures (11-97%) [[10]](#endnote-11) and in 69% of laparoscopic cases[[11]](#endnote-12).VAE being potentially fatal, VAE associated mortality is unclear in neurosurgical cases. But there are some case reports of massive air embolism leading to fatal outcome[[12]](#endnote-13).

**Case Presentation**

A 17-year-old, 60 kg, 152.4 cm, male patient, with no past medical or surgical history, non-smoker was admitted for Occipital headache on and off for 6 months and blurring of vision for 2 months. He denied having any allergies. An MRI was done which showed a Large multilobulated solid cystic lesion measuring 4.6X4.3X7.5cm in the pineal region with with leptomeningeal metastasis in left anterior temporal convexity as shown in the Fig. 1. His pre-opeartive vitals were within normal limits. The preoperative investigations were normal so the patient was scheduled for Infra-tentorial Supra-cerebellar Occipital Craniotomy and excision of tumor in sitting position under General anesthesia. The patient was considered as an American Society of Anesthesiologists physical status two with a Mallampati class II.



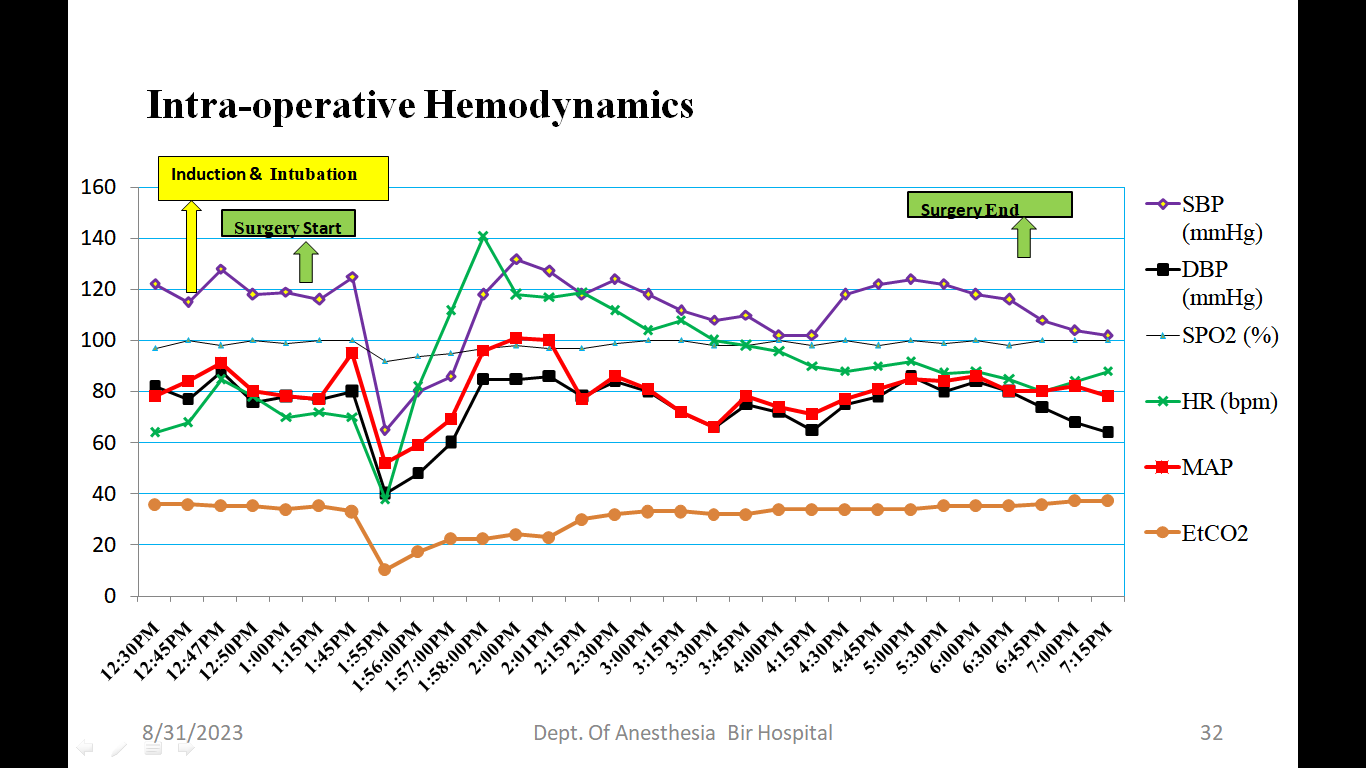
**Fig 1:** Plain CT head showing Pineal region Tumor

In the recovery room intravenous access was secured with 18 G cannula on non-dominant forearm. Normal Saline at 5ml/kg/hr was started to keep the line patent. In the Operative room he was pre-oxygenated with tight fitted mask with 100% oxygen at 6L/min. Simultaneously, he was induced with propofol 2mg/kg and fentanyl 2 mcg/kg. Once the possibility of ventilation was confirmed, muscle relaxant vecuronium 6 mg was given, then ventilated with oxygen and 1.5% isoflurane for three min. Then he was intubated with endotracheal tube of an internal diameter of 7.5 mm and fixed once placement was confirmed with bilateral equal air entry. Anesthesia was maintained with oxygen 2-3l/min, isoflurane 1-1.5% and vecuronium. A central catheter was placed into right internal jugular vein and an arterial line was placed into left radial artery, before incision the patient received 2 liters of normal saline. The position of patient was changed to sitting position as shown in Fig 2. Patient head secured in a three pin head holder after infiltration of the scalp and periosteum at pin site. About 1 inch space between the chin and chest confirmed to prevent cervical cord stretching and obstruction of venous drainage. Routine monitoring included 5-lead ECG, invasive and non-invasive BP, SpO2, Temperature and end tidal carbon dioxide measurements.



**Fig 2:** Sitting position for posterior craniotomy

With all stable vitals surgery was commenced. After 55 minutes of surgery during opening of dura a marked decrease in the end tidal carbon dioxide (ETCO2) from 33 to 10 was noticed. Heart rate falls from 70bpm to 38bpm, with a decrease of the blood pressure from 125/80 to 65/40mm of Hg and saturation decreased upto 92% as shown in Fig 3. Inj Atropine 0.6mg was given, surgeon were informed promptly and surgical field was irrigated with normal saline and covered with wet gauzes. On suspicion of venous air embolism blood was aspirated from central venous catheter which revealed bubbles of air (approx 38ml of air). The diagnosis of venous air embolism was established. Position of the patient was changed from sitting to supine. Inhalational agent was turned off and FiO2 increased to 100% PEEP of 5cm of H2O was added. His EtCO2 began to increase. His saturation of O2 increased upto 98% and her heart rate and blood pressure returned to normal limits. The end tidal carbon dioxide concentration returned to 30 after 20 minutes.



**Fig. 3:** Intra-operative Vitals

Despite the VAE incident, the surgical procedure was allowed to continue after ensuring the condition of the patient was stable and the ETCO2 concentration had returned to normal. The remainder of the surgery was completed without further complications. Post-operatively, the patient was closely monitored in the intensive care unit for signs of neurologic deficits or cardiac compromise. No adverse neurologic or cardiac sequelae were noted during the postoperative period. The patient was shifted to ward on 3rd postoperative day and discharged after 9 days and followed up on an outpatient basis without any evidence of VAE related complications.

**Discussion**

Sitting position for neurosurgery is commonly used for better approach to posterior and midline lesions which provides optimum access, lowers intracranial pressure, improved cerebral venous drainage, gravitational drainage of CSF and blood. Whereas performing surgeries in sitting position and avoiding physiological challenges has always been challenging job for an anesthesiologist. VAE, Hemodynamic instability and Nerve compression are some complication related to surgery in sitting position.

VAE may occur in any procedure where the operative site is above the level of the heart and non-collapsible veins exposed to atmospheric pressure or when pressurized air is introduced into the body cavity. There is high chance of air inflow into the venous system when there is pressure difference of 5 cm of H2O between central venous system and environmental pressure[[13]](#endnote-14),[[14]](#endnote-15). The clinical manifestation of VAE depend upon the the amount and rate of entry of air into the vein. It may vary from asymptomatic to chest pain, hypotension, bradycardia, arrhythmia and cardiac arrest[[15]](#endnote-16). The lethal dose of air embolism for human is 3-4ml/kg[[16]](#endnote-17). For an adult VAE with around 100ml volume of air is required to have clinical manifestation[[17]](#endnote-18).

VAE leads to trapping of air bubbles in the pulmonary vessels leading to ventilation-perfusion mismatch[[18]](#endnote-19),[[19]](#endnote-20). This will manifest as decreased in PaO2 and Increased PaCO2. The Entrapment of air bubbles in the pulmonary microcirculation may lead to cellular injury and lung edema. This will cause release of vasoactive mediators[[20]](#endnote-21),[[21]](#endnote-22) and increased microvascular permeability leading to increased flow of protein-rich lymph[[22]](#endnote-23) This blocking effect of air bubbles and pulmonary artery vasoconstriction due to vasoactive mediators leads to acute cor pulmonale and right ventricular decompensation secondary to the acute rise in right ventricular afterload. Consequently, the preloading of left ventricle and cardiac output can be severely diminished, followed by cardiac arrest[[23]](#endnote-24)

In awake patient, VAE can manifest as shortness of breath, chest tightness, bradycardia, hypotension or decreased oxygen saturation. In unconscious patients, severe VAE may manifest as acute decrease in end-tidal carbon dioxide (ETCO2), increase in end-tidal nitrogen (ETN2), hypotension or hypoxia inexplicable by deep anesthesia or hypovolemia. In our patient the reduction in EtCO2 was remarkably short-lived following position change. There was no hypoxemia either during or after the VAE, indicating that oxygenation and O2 exchange remained adequate. MAP reductions occurred after reductions in EtCO2 in our patient. This is presumably on the basis of decreased systemic cardiac output due to decreased right ventricular cardiac output with pulmonary vascular obstruction and hypertension. Bradycardia was responsive to Atropine after which heart rate and blood pressure raised.

Capnography is readily available and economic diagnostic tool with moderate sensitivity and specificity for diagnosing VAE in patient under general anesthesia. A sudden drop in end tidal CO2 associated with hypotension like in the case we presented is highly suggestive of VAE[[24]](#endnote-25). Precordial Doppler is highly sensitive method to detect VAE as compared to capnography and other conventional monitoring methods. However being subjective, false negative findings may be seen. Transesophageal echocardiography (TEE) is the most sensitive invasive method to detect VAE[[25]](#endnote-26). It can detect small air volume upto 0.01-0.19ml/kg in the heart[[26]](#endnote-27). But it requires adequate training of the anesthesiologist. Other diagnostic methods used to diagnose VAE are measurement of expired nitrogen, transcranial droppler and CT scan[[27]](#endnote-28).

Prevention is the key for management of VAE. Proper hydration prior to the procedure is considered to reduce the occurrence of VAE. Even after significant VAE, the greatest risk to the patient is continued entry of air into the venous system. Immediate alert to the surgeon on suspicion of VAE to start irrigation of surgical field and coverage of the exposed blood vessels will reduce the magnitude of VAE. In our case surgeon were informed promptly and the exposed surgical was irrigated and covered which prevents further embolism. Preventive measures such as reducing the pressure gradient through repositioning remain important. The urgent removal of the intravascular air demonstrated by Magendie in 1821[[28]](#endnote-29), with a catheter to aid aspiration remains valid today. In the presented case immediate reposition and aspiration of air via central catheter leads to brief VAE only. Anticipating the high risk procedures for VAE and proper monitoring are highly important for patient safety. Nitrous oxide should be discontinued and oxygen increased to 100%. Immediate use of ionotropic support should be considered for catastrophic VAE with cardiovascular collapse and if necessary, cardiopulmonary resuscitation may be required.

Regarding the case presented above, we realized that preoperative hydration didn’t help much in preventing VAE. Further this case made us to realize that VAE can occurs in a perfectly healthy patient without the presence of a patent foramen oval. In our case after stabilization of the patient there was no need to call off the surgery. Early detection and supportive measures are sufficient to stabilize the patient in order to proceed with the surgery.

**Conclusion**

Venous air embolism is a potentially life-threatening neurosurgical complication. The semi-sitting or the sitting position is the most important risk factor. Vigilant supervision, use of advanced 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. The anesthesiologist must be aware that surgery in the head-up position places the patient at risk for VAE. Emphasis should be given to the prevention and instant recognition of the event and to the use all available tools (fluids, positive inotropic agents) in the management of cardiovascular catastrophes.

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**CONFLICT OF INTEREST**

The authors declare that they have no competing interests.

**CONSENT**

Written informed consent was obtained from the patient for publication of this case report and any accompanying image.

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