Case Report

A Case of Venous Air Embolism During Paediatric Craniotomy in Sitting Position

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Abstract

Careful positioning under anaesthesia is an important aspect of intraoperative management to minimize morbidity and mortality, particularly in patients undergoing neurosurgical procedures. Amongst the different positions used in neurosurgery, sitting position is rarely used now a days in the view of high risk of venous air embolism and hemodynamic instability. This article presents a case report of venous air embolism in a pediatric patient who underwent posterior fossa craniotomy in sitting position.

Key Words: Sitting Position, Venous Air Embolism, Posterior Fossa Craniotomy.

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Case Report

A six year old boy presented with intermittent headache of one year duration, developmental delay and diminished performance in school with recent episodes of giddiness and fall. The MRI brain revealed a quadrigeminal plate tumor causing compression of the aqueduct of sylvius with obstructive hydrocephalus. He was managed with a ventriculo-peritoneal shunt and was planned for a definitive surgical procedure for excising the tumor 3 months later. The suboccipital craniotomy and near total excision of the tumor was done in the sitting position through an infratentorial supracerebellar approach.

The patient weighed 17 Kgs and was 120cms tall. He suffered from no comorbidities and there were no signs of congenital anomalies or difficult airway. General and systemic examination revealed no abnormalities. The patient was kept fasting for an appropriate duration and was given IV atropine 0.3mg and induced with IV midazolam 0.5mg, IV fentanyl 40mcg and IV propofol 40mg. IV vecuronium 2mg was given and oro-tracheal intubation was carried out with 5.0mm sized cuffed, flexometallic ETT. In addition to two wide bore intravenous catheters, a 5Fr triple lumen central venous catheter was inserted through the right subclavian vein and an arterial line was placed in right radial artery for continuous arterial blood pressure monitoring. Anaesthesia was maintained with an intravenous infusion of propofol and inhaled isoflurane in air and oxygen. The patient was ventilated with pressure controlled IPPV and a PEEP of 6 cmH_2O.

The patient was adequately preloaded with intravenous fluids and the sitting position was gradually achieved over 10minutes to avoid hemodynamic collapse while positioning from supine to sitting. A horse shoe support was placed to support the face.

Patient in sitting position with horse shoe face support

During surgery the torcula (confluence of major cerebral venous sinuses) was accidentally opened and resulted in air entrainment. There was a sudden drop in oxygen saturation (SpO_2) to 60%, End tidal CO_2 to 16mmHg and invasive BP to 60/40 mm Hg. The heart rate remained stable throughout. The surgical area was immediately flooded with saline and packed with wet gauze swabs, FiO_2 was increased to 100% and PEEP was increased to 10 cm H_2O. Minimal amount of air was aspirated from right atrial catheter when around 80ml of blood was withdrawn. Fluid loading was done and IV ephedrine was given to stabilize the hemodynamics. SpO_2, $EtCO_2$ and IBP gradually returned to normal in about 10 minutes. The rent in the torcula was clipped and the surgery was continued in the same position. At the end of the procedure the patient was reversed, extubated uneventfully and shifted to the neurosurgical ICU for monitoring.The histopathological examination of the tumour revealed it to be a grade II glioma and the patient was referred for radiotherapy.

Discussion

The use of the sitting position in neurosurgery has decreased compared to the past. The major concern is high rate of occurrence of venous air embolism in craniotomies done in sitting position. The reported incidence of VAE as detected by Doppler sonography in adults ranges from 7 to 50%[1-3]. Harrison and colleagues reported a significantly lower incidence of VAE in the paediatric age group of 9.3%[4], much lower compared to adult age groups. There are two prospective paediatric studies in the literature on the incidence of VAE in the sitting position. Meyer and colleagues reported a 26% incidence of VAE in the sitting position using capnography, in 30 children who were not treated with lower body positive pressure (MAST) or PEEP[5]. Fuchs and colleagues studied 24 children undergoing neurosurgery in the sitting position and reported a VAE incidence of 37% as detected by Doppler[6].

VAE in sitting position

A negative venous pressure relative to atmospheric pressure is necessary for venous air entrainment to occur. In the sitting position the dural venous pressure in the torcula will be negative compared to atmospheric pressure which allows air entrainment into the venous sinuses. Though most of the institutes abandoned the use of sitting position for neurosurgical procedures, Harrison and colleagues reported a lower incidence of VAE (9.3%) and VAE associated hypotension in only 2% out of 407 patients in their 16 years experience[4]. There was no significant perioperative morbidity or mortality and their results suggest that the sitting position can be used safely for neurosurgical procedures in children.

Diagnosis of VAE

The true incidence of VAE depends on the sensitivity of detection methods used during the procedure. The clinical presentation and various detection methods are detailed nicely in a review article[7]. In addition, many cases of VAE are subclinical and hence go unreported. The two main factors determining the morbidity and mortality of VAE are the volume of air entrained and the rate of accumulation. These variables are mainly affected by the position of the patient and height of the vein with respect to the right atrium. The lethal volume of air to cause VAE has been described as between 200 and 300 ml, or 3–5 ml/kg.

Clinically VAE manifests with tachyarrythmias, right heart strain pattern as well as ST and T wave changes in ECG. Myocardial ischemia may be observed with fall in blood pressure as the cardiac output decreases. Pulmonary artery pressure and central venous pressure increases as a consequence of increased filling pressure and right heart failure. There will be a significant fall in $EtCO_2$ with fall in arterial oxygen saturation(SaO_2). Cerebral hypoperfusion may occur due to reduced cardiac output which may manifest in the postoperative period as altered sensorium. Paradoxical air embolism may occur via a patent foramen ovale, a residual defect that is present in approximately 20% of adult population.

Specific monitoring modalities in the descending order of sensitivity and specificity of VAE detection are Transesophageal Echocardiography(TEE), precordial doppler, PA catheter, $EtCO_2$, ECG and esophageal stethescope. TEE is currently the most sensitive monitoring device for VAE, detecting as little as 0.02 ml/kg of air administered by bolus injection. But it is invasive, expensive and requires expertise. The precordial Doppler is the most sensitive of the noninvasive monitors, capable of detecting as little as 0.05 ml/kg of air. This is mostly used in obese patients and its position is confirmed by a bubble test. There will be a change in the character and intensity of the emitted sound. With greater air entrainment, a more ominous "mill wheel" murmur develops, indicating cardiovascular decompensation. A pulmonary artery catheter is a relatively insensitive monitor of air entrainment (0.25 ml/kg) and too invasive and hence is restricted to those patients with significant comorbidities. The fall in $EtCO_2$ occurs with air entrainment of 0.5ml/kg. The sensitivity of the esophageal stethoscope has been shown to be very low in detecting a mill wheel murmur. Timely anticipation of VAE during critical portions of the procedure is more vital than any detection device.

Prevention of VAE

Positioning: Head-up position places the patient at risk for VAE. In such situations, the propensity of incurring a negative pressure gradient between the open venous sinuses and the right atrium can be decreased by increasing right atrial pressure via leg elevation and flexion at the knees.

During insertion of central venous catheter: Insertion or removal of central venous catheter needs a few measures to prevent VAE like occlusion of needle hub or catheter during insertion or removal, insertion during exhalation phase of breathing as deep inspiration may increase the magnitude of negative pressure within the thorax. Trendlenberg position may be used during insertion of catheter as it may keep the CVP on the higher side.

Hydration: A well-hydrated patient reduces VAE risk. The right atrial pressure is to be maintained between 10 and 15 cm H_2O.

Use of PEEP: Though controversial, PEEP is being used in many institutes as a measure to minimize the risk of VAE. In addition PEEP of above 5 cm H_2O also helps to increase oxygen saturation.

Anti shock trousers: The military antishock trousers (MAST) during surgery has been shown to increase right atrial pressure in the sitting position. The right atrial pressure can be elevated and sustained above atmospheric pressure by maintaining military antishock trouser pressure greater than 50 cm H_2O[8]. There are risks of decreasing vital capacity, hypoperfusion of intraabdominal organs, and potential compartment syndromes with the use of this equipment.

Management of VAE

High vigilance and suspicion by the anaesthesia and surgical teams along with newer monitoring devices enable early diagnosis and prompt management of VAE. Stepwise management of VAE includes prevention of further air entrainment, reduction of volume of air already entrained, improved oxygenation and prompt hemodynamic support when needed.

Prevention of further air entrainment

When VAE is suspected the surgeon needs to be warned and surgical site should be flooded immediately with saline. Air entry can be further prevented by eliminating the possible negative air pressure gradient by changing the position of the patient and adjusting the tilt of the table. Also transient jugular venous compression increases venous pressure there by preventing further air entrainment into the open dural venous sinuses. Nitrous oxide has to be discontinued as it may expand the volume of air embolus. In addition to prevention of hypoxia, institution of high flow oxygen minimizes the size of air embolus.

Durant maneuver

Embolic obstruction in the right side of the heart by air embolus can be relieved either by placing the patient in a partial left lateral decubitus position (Durant maneuver)or by simply placing the patient in the Trendelenburg position.

Aspiration of air from right atrium

Central venous catheter is advisable to be placed in all such cases where there is higher chance of VAE instead of emergent catheter insertion for air aspiration during an acute setting of VAE-induced hemodynamic compromise. Central venous catheter is positioned with the tip 2 cm distal to the superior vena caval–right atrial junction[9]. Though success rate is very less and variable this procedure is life saving in cases of massive air embolism.

Hemodynamic support

Hemodynamic instability is an indication of massive air embolism which needs prompt action. The increase in right ventricular afterload because of VAE leads to acute right heart failure followed by decrease in left ventricular outflow. Inotropic support will correct hypotension and better optimize the myocardial perfusion.

CPR

Chest compressions are proved to mechanically force the air out of the pulmonary outflow tract in to the smaller pulmonary vessels, thus improving forward blood flow. Cardio pulmonary resuscitation with defibrillator may be needed in massive VAE.

HBOT

Hyperbaric oxygen therapy (HBOT) is believed to be beneficial due to a reduction in the size of the air bubbles secondary to accelerated nitrogen resorption. Another advantage of HBO therapy is the increased oxygen content of the blood. The HBO therapy is appropriate for paradoxical cerebral air embolism when it can be done within 6 hours from its occurance[10].

Summary

VAE is a silent but dangerous and life threatening condition which needs prompt attention. Planning the appropriate level of monitoring in procedures with high risk of VAE and prompt stepwise management are key to patient safety. Appropriate level of PEEP, intravenous fluid loading, immediate recognition of rent in the dural sinus by the surgeon and the evidence of VAE in the monitor, flooding of the surgical site with saline and application of saline soaked gauzes, prompt aspiration from right atrial catheter and suturing of the rent in the dural venous sinus are the steps done in this patient which probably has reduced the severity of VAE and prevented major hemodynamic compromise.

References

1) Papadopoulos G, Kuhly P, Brock M, et al. Venous and paradoxical air embolism in sitting position. A prospective study. ActaNeurochir 1994;126:140-3

2) Black S, Ockert DB, Oliver WC, Jr, Cucchiara RK. Outcome following posterior fossa craniectomy in patients in the sitting or horizontal positions. Anaesthesiology 1988;69:49-56

3) Young ML, Smith DS, Murtagh F, Vasquez A, Leritt J. Comparison of surgical and anaesthetic complications in neurosurgical patients experiencing venous air embolism in the sitting position. Neurosurgery 1986;18:157-61

4) Harrison EA, Mackersie A, McEwan A, Facer E. The sitting position for neurosurgery in children: a review of 16 years experience. British Journal of Anaesthesia 2002; 88(1): 12-17

5) Meyer PG, Cuttaree H, Charron B, Jarreau, Perie AC. Prevention of venous air embolism in paediatric neurosurgical procedures performed in sitting position by combined use of MAST suit and PEEP. British Journal of Anaesthesia 1994; 73 :795-800

6) Fuchs G, Schwarz G, Stein J, Kaltenbock F, Baumgartner A, Oberbauer RW. Doppler color-flow imaging: screening of patent foramen ovale in children scheduled for neurosurgery in sitting position, J NeurosurgAnaesthesiol 1998; 10:5-9

7) Mirski MA, Lele AV, Fitzsimmons L , Toung TJK. Diagnosis and treatment of Vascular Air Embolism. Anesthesiology 2007; 106:164–77

8) Hanna PG, Gravenstein N, Pashayan AG. In vitro comparison of central venous catheters for aspiration of venous air embolism: Effect of catheter type, catheter tip position, and cardiac inclination. J ClinAnesth 1991; 3:90–4

9) Meyer PG, Cuttaree H, Charron B, Jarreau MM, Peri AC, Sainte-Rose C. Prevention of venous air embolism in paediatric neurosurgical procedures performed in the sitting position by combined use of MAST suit and PEEP. Br J Anaesth 1994; 73:795–800

10) Blanc P, Boussuges A, Henriette K, Sainty JM, Deleflie M: Iatrogenic cerebral air embolism: Importance of an early hyperbaric oxygenation. Intensive Care Med 2002; 28: 559–63

Answer to : **Diagnose the condition**

ECG shows narrow QRS complex with a ventricular rate of 45/min without a preceding P wave.But at the end of QRs complex there is a sharp deflection suggestive of retrograde P wave from AV node.

Possibilities are Sinoatrial nodal arrest or sinus exit block. TYPES OF SA NODAL DYSFUNCTION — What we call the sinoatrial (SA) node actually represents the integrated activity of pacemaker cells, sometimes called P cells in a compact region at the junction of the high right atrium and the superior vena cava. Perinodal cells, sometimes called transitional or (T) cells, transmit the electrical impulse from the SA node to the right atrium. Thus, SA nodal dysfunction can result from abnormalities in impulse generation by the P cells or in conduction across the T cells.

SA nodal pauses and arrest may result from abnormalities in impulse generation by the P cells. SA nodal exit block may be due to abnormalities in conduction across the T cells.

The mass of the SA node is too small to create an electrical signal that can be routinely recorded on the surface ECG. As a result, we generally infer SA nodal activity from the ECG appearance of the response to that activity: atrial activation as manifested by the P wave. Patient needs pacemaker (DDDR).

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