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Case Report

A catastrophic case of venous air embolism in a patient undergoing posterior fossa surgery

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ABSTRACT

Posterior cranial fossa surgeries pose increased risk of venous air embolism (VAE) due the associated pressure gradient. Early detection of VAE is of paramount importance to avoid the disastrous consequences. We, hereby report a case of catastrophic VAE in a 47-year-old male undergoing surgery for glioblastoma.

Keywords: Air entrainment, ETCO2, Venous embolism

INTRODUCTION

Posterior cranial fossa surgery provides a significant challenge to the anaesthetist and surgeon, due to life threatening complications like Venous air embolism (VAE). The risk of VAE is quite high because the operative site is usually kept above the level of heart, which leads to easy air entrainment due to pressure gradient.[1] Also, the venous sinuses in posterior cranial fossa area are non-collapsible because of their dural attachments and sub-occipital plexus of veins are held open by fascial attachment to cervical muscles leading to high incidence of VAE (45-50%).[2] Patient position specially sitting position carries the highest risk of VAE (25-40%) but it may also occur in prone position, lateral decubitus and supine position.[3,4] We, hereby report a case of massive VAE in a patient undergoing posterior fossa surgery in prone position.

CASE REPORT

A 47-year-old male patient presented with history of blurring of vision for 6 months. MR imaging of the brain revealed a large tumor of the size of 7.2x5.7x7cm in right occipito-parietal region with features suggestive of glioblastoma. Patient was planned for surgical resection of the tumor via occipital craniotomy. Investigations like haemogram, coagulation profile, RFT, LFT, chest X-ray, ECG were all normal. In the operation theater, patient's vitals like heart rate, blood pressure, SpO2, respiratory rate, temperature were monitored after attaching to the monitor. Patient was intubated with flexo-metallic tube after inducing with I.V Inj. fentanyl 100 ug, midazolam 1.5 mg, propofol 120 mg and atracurium 30 mg. Subclavian vein cannulation was done for central venous pressure (CVP) monitoring and drug institution. Radial artery was cannulated after performing Allen's test for beat-to-beat monitoring of blood pressure and intermittent ABG analysis was done. Patient was then placed with head up in prone position. The surgery was proceeding uneventfully till partial resection of tumour, with continuous monitoring of pulse, SpO2, ETCO2, ECG- ST segment analysis, temperature, IBP, CVP and intermittent ABG analysis, which were all within normal limits.

The patient had a sudden episode of fall in ETCO2 from 34 to 13 mm hg, HR from 90 to 48, BP from 104/63 to 80/42 mm hg, SpO2 dropped from 100 to 72 %, with strain pattern on ECG. The peak airway pressure (Ppeak) raised from 12 to 24 cm of H2O and base line CVP of 8 mm Hg was raised up to 20 mm Hg. Immediate possibility of VAE was suspected. Surgeons were informed and immediate resuscitative measures were started viz. operative site flooded with saline, head down with left lateral tilt (Durrant's position), I.V. inj. atropine 0.6 mg, ventilation with 100% O2 with PEEP increased to 8 and aspiration of air (approx. 80ml) was done through the distal lumen of subclavian line. Haemodynamics were supported with intravenous fluids and inotropes (nor-adrenaline@ 0.1-1ug/kg/min). Patients vitals normalized after 10 minutes of resuscitation and nor-adrenaline infusion was continued to maintain mean arterial pressure (MAP) to at least 65 mm hg. ABG analysis revealed hypoxia and hypercapnia. Surgery was resumed with the aim to immediately close the operation site. But, patient again had similar episode of VAE after 10 minutes of resuming surgery which lead to cardiac arrest. Patient was immediately turned supine and CPR was started according to ACLS guidelines. Nearly 50 ml of air was aspirated from the central line simultaneously. Patient revived again and was put on adrenaline infusion. Surgeons closed the operation site and patient was shifted to ICU for further management. Unfortunately, patient again had cardiac arrest in ICU after 30 minutes. CPR was started again and continued for 20 minutes but patient could not be revived this time.

DISCUSSION

Air embolism occurs due to entrapment of air into the vasculature because of a pressure gradient and a persistent communication between the source of air and blood vessels. Posterior fossa surgeries carry a higher risk for VAE when compared to other surgeries, because the positioning of the patient required for surgery favours the likelihood of a pressure gradient.[1] In our case, patient was positioned prone with head kept above the level of heart which created the pressure gradient. Small amount of air in the circulation does not cause any clinical manifestations as it is broken and get absorbed while moderate amount of air causes pulmonary vascular injury which may lead to pulmonary hypertension and pulmonary oedema. The entry of large bolus of air in venous system can cause an air lock in right side of the heart may cause right ventricular flow obstruction, and further may lead to systemic hypotension, myocardial ischemia, arrhythmia, hypoxemia from ventilation-perfusion mismatch, shunt and ultimately death. The fatal amount of air in human is reported to be either 200- and 300-ml bolus or 3-5 ml/kg.[5,6]

In high-risk cases, high index of suspicion along with appropriate monitoring devices may help in early detection of VAE. Trans-oesophageal echocardiography (TEE), precordial doppler ultrasound, end tidal nitrogen (ETN) and end-tidal carbon dioxide (ETCO2) can be useful in early detection of air in the vascular system.[7] TEE is the most sensitive tool in detecting very small volume of 0.02 ml/kg followed by precordial doppler and ETN. ETCO2 is the most common and easily available monitoring parameter, which will show immediate fall in its level in a case of VAE.[7,8]

The goal of treatment in VAE is to prevent further air entry by covering the surgical field with saline soaked dressing, tilting the table (Durant's position) to relieve air lock in the right side of heart, reduction in entrained air volume by air aspiration from central line and haemodynamic support by maintaining optimal fluid status and inotropic support.[7] Putting the patient on 100% oxygen support can maximize the patient's oxygenation and reduce the volume of embolus by eliminating nitrogen.[9] Rapid cardiopulmonary resuscitation with chest compression has been found to be effective in massive VAE. Mortality of VAE ranges from 48 to 80% and has a direct relation with the amount of air entrained, rate of accumulation of air, and position of the patient at the time of VAE.[10]

In our case, as we did not have sensitive tools like TEE or precordial Doppler. Continuous monitoring of ETCO2 along with high index of clinical suspicion (sudden fall in levels of ETCO2 with deranged haemodynamics) led to the diagnosis of VAE, which was confirmed by aspiration of high volume of air (approximately 80 ml). Patient was treated along the lines of VAE and was normalized initially. But the continuous exposure to air from surgical site led to disastrous consequences in the form of cardiovascular collapse and ultimately death of the patient.

CONCLUSION

More sensitive monitoring tools and usage of devices like TEE might have led to early detection of VAE and hence early treatment. The continuous exposure to air should be avoided by immediately closing the surgical site to prevent further and more severe episodes of VAE.

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