A Precipitous Cause of Iatrogenic Venous Air Embolism in Pediatric Posterior Fossa Tumor

Sir,

We report a 6-year-old male child diagnosed as a case of posterior fossa tumor posted for midline suboccipital craniotomy (MLSOC) and excision in prone position. He presented to the emergency department with complaints of severe headache and recurrent vomiting. Computerized tomography (CT) scan of the brain showed posterior fossa tumor and hydrocephalus suggestive of medulloblastoma. He had undergone emergency ventriculoperitoneal shunt surgery under general anesthesia 7 days back. Presently, he is posted for posterior fossa tumor resection under general anesthesia. He was advised to take a night dose of anticonvulsant drug. General anesthesia was induced with fentanyl (2 mcg/kg), propofol (2 mg/kg), and rocuronium (1 mg/kg). A flexometallic cuffed endotracheal tube of 5.0 mmID was used to secure the airway. Invasive lines, right subclavian 6 Fr catheter and left dorsalis arterial cannula, were secured under aseptic precautions. The central and peripheral lines were disconnected and blocked just before turning the patient to prone position. While turning the patient to prone position, pulse oximeter probe was kept attached to the finger for monitoring. A single dose of muscle relaxant was repeated before turning the patient. All monitors were reattached, pressure points were padded, and abdominal compression was checked and assured once the patient was turned to prone position. Immediately, after few minutes there was a sudden fall in end-tidal carbon dioxide ($EtCO_2$) from 25 mmHg to 6 mmHg, followed by a drop in oxygen saturation from 100% to 59%, hypotension (85/53 mmHg to 79/44 mmHg), and increase in airway pressure from 24 cmH_2O to 40 cmH_2O [Figure 1]. Nitrous oxide was stopped immediately and ventilated with 100% oxygen. The surgeon was informed and asked to instill normal saline at the surgical site, which was a small skin incision. While planning to give rapid intravenous (IV) fluid for resuscitation, a small volume of air was observed in the extension lines and was stopped immediately. All extension lines were flushed properly before giving the fluid. A single dose of vasopressor (mephentermine 3 mg) was required to maintain the blood pressure. On central line aspiration, 5–10 mL of air was aspirated. The patient regained 100% oxygen saturation, normotension, and normocarbia within 2–3 min of management. The surgery was allowed to continue. After 6 h of surgery, the patient's remaining intraoperative course was uneventful and his trachea was extubated after complete neuromuscular blockade reversal. He was shifted to the neurointensive care unit (NICU) for further management and later shifted to the ward after 2 days.

Figure 1: Red arrow showing the rise in peak airway pressure from 24 cmH_2O to 40 cmH_2O. White arrow showing sudden fall in $EtCO_2$ from 24 mmHg to 6 mmHg

Venous air embolism (VAE) is a potentially lethal complication during surgery either because venous pressures being subatmospheric or because air is forced under pressure into the body cavity.[1] Few case reports of accidental delivery of air have been mentioned, where the lethal volume has been described at 3–5 mL/kg.[2] The authors of these case reports suggest that the closer the vein of entrainment is to the right heart, the smaller the required lethal volume. Clinical features include a sudden fall in $EtCO_2$, saturation of oxygen, decrease in cardiac output due to right ventricular obstruction, and increased airway pressure. VAE increases microvascular permeability, causing injury to the pulmonary capillary network leading to pulmonary edema.[3] $EtCO_2$ is the most convenient monitoring that can be employed to detect VAE but the specificity is less.[4] A change in the saturation of oxygen is not an early sign and it requires severe physiological disturbance to manifest as fall in its values. The management of a possible VAE has to be a quick response. The surgeon should be informed to access and eliminate any possible entry site. To maximize the patient's oxygenation, 100% oxygen and discontinuation of nitrous oxide has to be done to reduce the size of the embolus. The use of nitrous oxide can dramatically increase the size of the entrapped air inside the body cavity.[5] In case of a severe hemodynamic disturbance, cardiopulmonary resuscitation with chest compressions have to be done. Closed-chest compression forces air out of the pulmonary outflow tract into the smaller pulmonary vessels, improving the forward blood flow. Aspiration of air from the central venous access and providing hemodynamic support with the use of vasopressors, form the rest of the management of VAE. It is

said that the optimal management of VAE is prevention. In our case, it was most likely due to the accidental air injection during the switchover of the IV extension tubing between the peripheral and central catheters. Our case report is a kind reminder for the need for vigilant monitoring and prevention of VAE as the best modality to prevent any catastrophes during surgery.

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Conflicts of interest

There are no conflicts of interest.

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