

Venous air embolism during ventriculoatrial shunt placement!

Sir,

Ventriculoatrial (VA) shunt channel cerebrospinal fluid (CSF) from the ventricle of brain into the right atrium of the heart. It is indicated in conditions where repetitive shunt revisions may be required due to ventriculoperitoneal (VP) shunt obstruction, infection or migration.^[1] Shunt occlusion, bacteraemia, cardiac tamponade, cardiac rupture, thromboembolism and intracranial haemorrhage are commonly encountered complications with VA shunts.^[2] However, venous air embolism (VAE) is a rare complication of VA shunt procedure. We report a case of intra-operative VAE and its successful management in a 5-year-old child who underwent a VA shunt surgery for hydrocephalus.

A 5-year-old, 11 kg, American Society of Anesthesiologists class I male child was planned for VA shunt placement under general anaesthesia. He was an operated case of occipital encephalocele with obstructive hydrocephalus and had CSF ascites following VP shunt placement. The patient had a stable intra-operative course until the surgeons dissected the subclavian vein. While inserting the shunt catheter into the vein, there was a sudden drop in end-tidal carbon oxide (EtCO₂) from 35 to 22 mmHg along with transient hypotension (detected by invasive arterial blood pressure). Careful inspection revealed an open shunt insertion site, as the gauge piece covering the site was displaced.

Immediately, patient's lungs were ventilated with 100% oxygen assuming VAE to be the cause of the event. The surgery was briefly interrupted, and the operative site was covered with saline-soaked gauze pieces. Hypotension was managed with intravenous (IV) fluid and mephentermine (1.5 mg) IV. The EtCO₂ returned to normal within next 5 min. Rest of the surgery was uneventful, and after tracheal extubation, the patient was shifted to Neurosurgical Intensive Care Unit.

VAE is the entrainment of air into the venous vasculature from the operative field or other communication with the environment; producing systemic effects.^[3] Position of the patient and height of the vein with respect to the right side of the heart determine the rate and volume of air entrainment which ultimately determine morbidity

and mortality after VAE.^[3] In our case, there was a pressure gradient between the shunt insertion site and right side of heart due to the elevated position of right shoulder leading to entrainment of air through exposed subclavian vein. In VA shunt placement, risk of VAE is maximum when central vein is opened for tube insertion, and children appear to at higher risk of VAE as compared to adults.^[4] However, EtCO₂ decrement is not specific to VAE, and acute hypotension or pulmonary embolism can also lead to a decrease in EtCO₂.^[5,6] There was no predisposing factor for pulmonary embolism, and there was no preceding hypotension before fall in EtCO₂ in our patient. Hence, we considered VAE to be the aetiology of the event and managed it.

VAE can occur during VA shunt procedure, especially during the inserting of the shunt into central vein. Meticulous surgical technique and high level of vigilance during insertion of VA shunt are warranted to avoid VAE. In the event of sudden and sustained fall in ETCO₂ in VA shunt surgery, VAE should be the first diagnosis and should be managed accordingly.

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Conflicts of interest

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