Case Report

Retrograde Cerebral Venous Gas Embolism: Could it be Possible?

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Abstract

Air embolism is well-known but uncommon and cerebral venous gas embolism (CVGE) is even more so because it goes unnoticed in an anaesthetized patient, especially where slow entrainment of small amounts of air takes place over a period of time. A young American Society of Anesthesiologists (ASA) status I patient underwent a lumbar spine surgery and presented in the post anaesthesia care unit (PACU) with seizures. Common causes of generalised tonic clonic seizures were ruled out and cerebral hypoperfusion as a result of retrograde CVGE was the retrospective diagnosis of exclusion. The patient developed retrograde CVGE in the absence of any intracardiac septal defect or patent foramen ovale. The prone positioning along with the anatomy of the valveless vertebral plexus of veins plays an important role in the development of retrograde CVGE. A high index of suspicion and awareness is warranted from anaesthesiologists so as not to delay diagnosis and treatment of this rare entity.

Key words: Cerebral venous gas embolism, hyperbaric oxygen therapy, postoperative seizures, retrograde

Introduction

The key to a non-epileptic patient having seizures in the immediate postoperative period is prompt symptomatic management followed by ruling out the possible causes.

Air embolism, though a well-known adverse event of medical therapy, is uncommon. Cerebral venous gas embolism (CVGE) is even more so. It usually arises in a patient who has survived a systemic air or gas embolism event, with or without a need for cardiopulmonary resuscitation (CPR). However, it may be more subtle or even unnoticed in an anaesthetized patient or in situations where slow entrainment of small amounts of air takes place over a period of time.[1] Reviewing the literature has not only raised a significant patient safety issue owing to undiagnosed cases but also an interesting new understanding of the mechanism in some cases. We present a case report of a young American Society of Anesthesiologists (ASA) I patient who underwent a lumbar spine surgery in the prone position and presented in the recovery room postoperatively with seizures. Common physiologic, pharmacologic, and pathologic causes of generalised tonic clonic seizures were ruled out. Retrograde CVGE was the diagnosis of exclusion.

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Case Report

A 34-year-old male, weighing 65 kg, ASA grade I patient scheduled for minimally invasive transforaminal lumbar interbody fusion L5-S1 in the prone position had an insignificant preoperative anaesthetic clinical evaluation; laboratory investigations were normal. General anaesthesia was administered utilizing standard ASA monitoring. The intraoperative period was uneventful and lasted 3 h.

An episode of generalised tonic clonic seizure (GTCS) occurred in the recovery room. He was very restless postictal. Blood pressure post GTCS was 140/90 mmHg, heart rate was 100/min, and electrocardiograph showed sinus rhythm. Blood glucose, arterial blood gas analysis, serum electrolytes, and core body temperature were all within normal range. He was intubated and shifted to the intensive care unit (ICU) on ventilator.

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Levetiracetam 1 g loading dose followed by 500 mg 12 h was given intravenously. Computed tomography (CT) of the brain done after an hour was normal. Patient was started on sedoanalgesia with intravenous midazolam (0.25 mcg/kg/min) and fentanyl (1mcg/kg/h).

Another episode of GTCS occurred the next day, and intravenous midazolam infusion 1 mcg/kg/min was started. Electroencephalogram (EEG) showed bilateral epileptiform waves. Magnetic resonance imaging (MRI) [Figure 1] of the brain showed abnormal areas of restricted diffusion with corresponding hypointensity on apparent diffusion coefficient (ADC) maps, noted in the bilateral frontoparietal and temporal cortical regions suggestive of fresh infarcts, were visualised on fluid-attenuated inversion recovery (FLAIR) images. MR angiography of the head and neck vessels was unremarkable. Two-dimensional echocardiography showed no evidence of intracardiac clot or any septal defect. Patient had multiple episodes of GTCS for 2 days. He was haemodynamically stable throughout his ICU stay. He was managed in the ICU with antibiotics, anti-platelets, anti-epileptics, and wound care and physiotherapy. He was weaned off from the ventilator gradually along with stepping down of the midazolam infusion, was extubated on the 5th postoperative day, remained seizure-free, and started obeying commands. He was discharged on the 10th postoperative day with no neurological sequelae.

Discussion

Post-operative seizures can be due to hypoxia, hypercarbia, hypoglycaemia, hypotension, electrolyte abnormalities, such as hyponatremia and hypocalcemia, pyrexia, acidosis, drug-related such as local anaesthetics, inhalational agents such as sevoflurane and opioids. Rarely tumour, an aneurysm, underlying seizure disorder, psychogenic seizures or pseudo seizures, cerebral venous air embolism, and global or focal cerebral ischaemia can be detected.

The concept of 'retrograde passage of air bubbles' is a new theory of an old phenomenon, first described in a letter to the editor published in The Lancet.[2] It occurs with (1) Direct injection of gas into the arterial circulation during angiography, pulmonary barotrauma enabling gas to enter the pulmonary veins, then via the left heart to the systemic arterial system, including the coronary and cerebral arteries; (2) paradoxically, from the venous system through an intracardiac right-to-left shunt, or where an intracardiac shunt is excluded, arteriovenous malformations in the lungs or overwhelming of the pulmonary capillary filter mechanism have been demonstrated to allow gas to enter the systemic arterial system; (3) the concept of CVGE should be recognized as a third mechanism of cerebral gas embolism.[3]

A unique finding has been the discovery of CVGE in certain cases after CPR, insertion of peripheral or central lines could be implicated, or pulmonary barotrauma with air entering the circulation may be the reason. However, those who have CVGE obviously were due to retrograde flow. This may occur

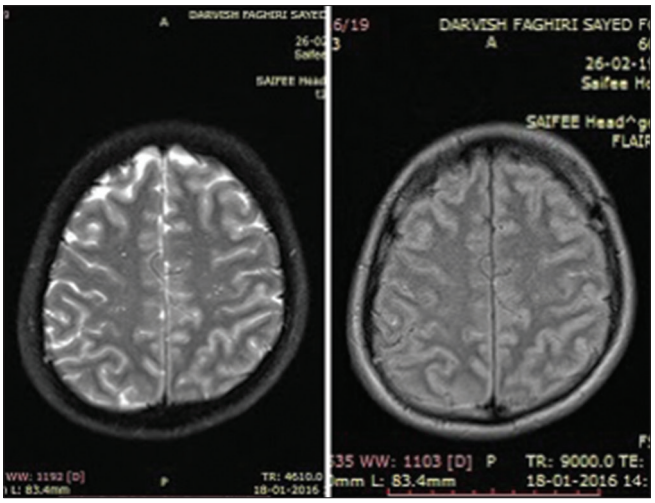

Figure 1: MRI study showing infarcts on FLAIR imaging and ADC maps

due to a low cardiac output state allowing retrograde flow of bubbles either in the supine CPR position or more likely from the thoracic compressions.[1]

If a bolus of air lodges into the pulmonary arteries and causes obstruction to the pulmonary outflow, a right-to-left shunt through a patent foramen ovale is likely to occur, with subsequent cerebral air embolism. In the absence of a patent foramen ovale, venous air must traverse the pulmonary vasculature to enter the cerebral circulation. The pulmonary arterioles and capillaries are generally considered an effective filter for thrombi, platelet aggregates and fat emboli, however, trapping of air may not be so effective. In a canine model, the lung is a very effective filter for air bubbles of greater than 22 μ in diameter when infused slowly.[4] However, a bolus injection of 30 ml of air into the central vein exceeded the filtering capacity of the lung and produced embolisation through the left heart and into the arterial circulation.

The vertebral venous plexus (VVP) is angiographically linked to the cranial venous system, as demonstrated by Batson in 1940. The VVP is considered to be a part of the cerebrospinal venous system (CSVS) which is regarded as a unique, large-capacitance, ebb and flow, valve-less plexiform venous network in which flow is bidirectional that plays an important role in intracranial pressure homeostasis with changes in posture and intrathoracic or intraabdominal pressure and in venous outflow from the brain, whereas in disease states, it provides a potential route for the spread of tumour, infection, or emboli.

Vertebral venous air embolism has been reported as a complication of colonoscopy,[5] following spinal surgery[6] and intraoperative use of irrigation with hydrogen peroxide of spinal surgical sites.[7] Complications of percutaenous vertebroplasty and kyphoplasty may have resulted from cement embolization including cerebral cement emboli and pulmonary cement emboli.[8] In addition, intracranial and pulmonary emboli reported during hip hemiarthroplasty may have occurred via the CSVS.[9]

The size of surgically derived emboli is unknown. We had excluded other causes of postoperative seizures as blood glucose, arterial blood gas analysis, core body temperature and serum electrolytes were normal. Further, there was no possibility of a drug-induced cause. We had maintained MAC value of sevoflurane at 1.85% and had not used any local anaesthetics. Intravenous fentanyl 100 mcg was used before intubation and no additional dose was required during surgery. CT brain and MRI ruled out other possible rare causes of CVGE such as tumour or aneurysm.

We assume that small continuous amount of air must have been entrained during the surgery and must have travelled in retrograde fashion via the valve-less vertebral venous plexus to the cerebral circulation as well as to the pulmonary circulation. However, pulmonary embolisation was not significant enough. The air entrainment into the venous system includes two risk factors, i.e., a direct communication between air and non-collapsible veins and a pressure gradient favouring the air entry. It has been explained how a significant gradient may exist with the patient in prone position with abdomen hanging free, and reducing caval pressures, hence increasing the chance of air entrainment into the venous circulation. This presumption was agreed upon by the neurologist as all other possible causes were excluded and patient recovered well with no functional neurological deficit.

Modern MRI techniques are highly sensitive and specific for the detection of acute ischaemic cerebral lesions. Diffusion-weighted MRI permits the detection of even very small, acute infarction at almost any anatomical location and serves as an essential surrogate endpoint for ischaemic stroke.

Retrograde CVGE can be treated with hyperbaric oxygen therapy[10] and other ancillary approaches such as antiplatelets to counteract platelet aggregation associated with air embolism, steroids to prevent cerebral oedema, haemodilution by dextran 40, control of seizures by anticonvulsants and measures to improve cerebral metabolism.

The most important consideration in the treatment of cerebral air embolism is awareness, anticipation and preparedness.

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Conflicts of interest

There are no conflicts of interest.

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