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Cerebral venous air embolism after neurosurgical intervention: A case report and literature review

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Abstract

Background: Cerebral venous air embolism is a rare complication after a routine neurosurgical intervention. The pathophysiology is not clear despite the existence of several etiologies.

Case presentation: A young male patient who was operated for a traumatic epidural hematoma. Postoperatively, he presented with confusion and headache without focal neurologic deficits. A brain computed tomography showed multiple cerebral venous gas emboli in the superior sagittal sinus. He received supportive treatment with a good clinical course.

Discussion: The mechanism for entrance of air into the cerebral venous circulation is thought to be through retrograde rise. It can be a potentially lethal complication with mortality dependent on the volume of air within the vessels, rate of accumulation and the affected territory.

Conclusion: The aim of this case report is to highlight an exceptional and understudied complication of neurosurgical intervention.

Keywords: Air embolism, neurosurgery, venous sinus

Introduction

The introduction of air into cerebral venous circulation called cerebral venous air embolism (CVAE) is an extremely rare complication after a neurosurgical surgery. The pathophysiology and treatment remain unclear ^[1]. Symptoms are often not specific and the diagnosis can be difficult ^[2].

We describe a male patient who presented with confusion after a surgery for traumatic extradural hematoma. A brain computed tomography (CT) showed air in the superior sagittal sinus.

The aim of this report is to highlight a rare and atypical complication entity, and Literature Review.

Case report

A 17-year-old boy, presented to the emergency department with head trauma following road traffic accident. His past medical history was unremarkable. The physical examination found a patient confused, disoriented, with violent and persistant vomiting, without any focal neurological deficits, the respiratory and hemodynamic parameters were normal. A brain computed tomography showed a right frontal extra dural hematoma which was considered suitable for immediate surgical evacuation.

In the operating room; routine non-invasive monitoring was established, including noninvasive blood pressure (NBP), heart rate (HR), pulse oximetry (SpO2), and electrocardiography (ECG). After obtaining a venous access with a 18 gauge cannula, preoxygenation was made with 100% of oxygen until expiratory oxygen more than 90%.; rapid sequence induction was performed with fentanyl (200 μ g), propofol (150 mg) and rocuronium (90 mg), sellick maneuver was performed and a size 7,5 endotracheal tube was placed. Sevoflurane (MAC 1.5) in a mix of air and oxygen (FiO2 50%) allowed a maintenance of anesthesia. The surgery was completed uneventfully. Throughout the whole operation, blood pressure, ventilation and oxygenation were adequate. There was no excessive blood loss. At the end of surgery Sevoflurane was stopped and tracheal tube was taken out in a deep plane of anesthesia. The patient went after the surgery to intensive care unit for post-operative monitoring.

Post-operatively, patient presented with confusion and headache without focal neurologic deficits. Head computed tomography performed revealed air bubbles in the superior sagittal sinus (Figure 1,2).

The hyperbaric chamber was not available. The patient received cautious intravenous hydration and 100% oxygen therapy with improvement in neurological condition after a few hours.

Discussions

Cerebral venous air embolism is a rare phenomenon and an atipycal complication after head surgery. Although its true incidence is unknown ^[3]. It can be a potentially lethal complication. Mortality dependent on the volume of air within the vessels, rate of accumulation and the affected territory ^[1].

The mechanism for entrance of air into the cerebral venous circulation is thought to be through retrograde rise. First described in a letter to the editor of Lancet^[4]. Subsequently, a laboratory model has demonstrated how this could happen, depending on flow dynamics, buoyancy depending on bubble size, and position of the thorax relative to the bed^[5]. There are different etiologies for venous gas embolism: after

manipulation of a central or peripheral vessel, cardiac or neurosurgical interventions, endoscopic procedures and high pressure mechanical ventilation ^[6].

Signs and symptoms are nonspecific with acute onset of various degrees of altered mental status, headache, vertigodizziness, chest pain, motor or sensory deficits, seizures, speech impairment, akinetic mutism, , syncope and blurred vision ^[7]. The brain-CT scan is sufficient to makeeasily the diagnosis.

The aim of treatment is to prevent continued entry of air into the venous circulation, reduce volume of air bubbles and optimizing perfusion and oxygenation of affected territories. A volume resuscitation to increase venous pressure is ensured by intravenous fluids and adequate oxygenation with high concentration (100%) via mask ^[8]. Hyperbaric oxygen therapy may be considered for more severe cases, with arterial cerebral air embolism but it is not the first line of therapy for CVAE ^[5-9], it may be considered for selected patients ^[10].



Fig 1: Conventional brain-CT scan, A: coronal and B: sagittalcuts showing air in the superior sagittal sinus (arrows).

Conclusion

This case demonstrates the need for awareness among anesthesiologists and neurosurgeons regarding the possibility of this complication following routine neurosurgical intervention and the importance of radiologic imaging to differentiate the diagnosis to other postoperative complications which need a reintervention.

Author's Contribution

Not available

Conflict of Interest

Not available

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