Air embolism during lumbar surgery in the prone position

Lionel Bapteste[1], Zeinab Kamar[1], Anthony Mazaud[2], Baptiste Balança[1]

[1] *Anesthésie Réanimation Hospices Civils de Lyon, France*
[2] *Centre de médecine hyperbare Hospices Civils de Lyon, France*

Corresponding author: *Dr Lionel Bapteste, Anesthésie Réanimation Hospices Civils de Lyon, France*
lionel.bapteste@chu-lyon.fr

Key words
Anaesthesia; Case reports; Cerebral arterial gas embolism (CAGE); Hyperbaric oxygen treatment; Neurosurgery; Surgery

Abstract

(Bapteste L, Kamar Z, Mazaud A, Balança B. Air embolism during lumbar surgery in the prone position. Diving and Hyperbaric Medicine. 2021 September 30;51(3):303–305. doi: 10.28920/dhm51.3.303-305. PMID: 34547783.)
Only a few clinical cases of cerebral arterial gas embolism during spinal surgery are published. It seems important not to overlook this diagnosis in order to initiate rapid appropriate treatment. This was a suspected case of paradoxical gas embolism revealed postoperatively by neurological deficits and whose recovery was noted during hyperbaric oxygen treatment. Unfortunately, no complementary examination showed gas embolism and only the context, the clinical picture and the case evolution evoke this diagnosis. The diagnostic difficulty in the immediate postoperative period is highlighted.

Introduction

During lumbar surgery in the prone position, air embolism (AE) can occur and is likely underdiagnosed.[1] There are only a few reports of such a complication.[2] Interestingly, reported cases are typically fatal but AE can also occur with various clinical presentations. Therefore, this clinical scenario is important, as is early diagnosis of AE in this unusual context in order to be able to initiate appropriate treatment as early as possible. Herein is reported a probable case of a cerebral arterial gas embolism (CAGE) during lumbar spine surgery. To our knowledge, this is the only case of CAGE revealed by neurological deficits after lumbar spine surgery with a rapid and complete recovery after hyperbaric oxygen treatment.

Case report

The patient consented to publication of his case details.

A 62-year-old ,76 kg male patient with history of hypertension and type 2 diabetes mellitus treated with oral medications, presented for elective L4-5 laminectomy surgery. He had previously been operated on without any complication. Preoperatively, the patient was alert and did not have any neurological deficit.

General anaesthesia was conducted with total intravenous anaesthesia with a targeted controlled infusion of propofol and sufentanil. Boluses of ketamine and lidocaine were administered at the beginning of the procedure, and rocuronium was given prior to orotracheal intubation. The patient was then placed in a knee-chest prone position. Standard monitoring (electrocardiogram, non-invasive arterial blood pressure, pulse oximetry, end-tidal CO_2 [E_TCO_2]) was used during anaesthesia. The surgery lasted 115 minutes and the blood loss was estimated to be 100 mL. The arterial blood pressure was maintained using continuous infusion of norepinephrine. No adverse change in the electrocardiogram, hypotension, or fall in E_TCO_2 was observed during the surgery. Only one transient mild arterial oxygen desaturation (from 99% to 96%) was recorded. The patient received tramadol, nefopam, and paracetamol for post-operative analgesia. At the end of the procedure, the train-of-four indicated the presence of four twitches with T4/T1 of 99%. A mild desaturation (SpO_2 = 94% with FiO_2 = 70%) that lasted a few minutes was managed by a recruitment manoeuvre. The patient was subsequently extubated (76 min after the end of the surgery).

On emergence, he was extremely agitated; his restlessness was attributed to the post-operative back pain which was treated with a 1mg intravenous bolus of morphine. Soon after the injection the patient became stuporous. The examination revealed an anisocoria, right hemiplegia, and bradypnoea with a stable heart rate, blood pressure, and temperature. Shortly after, re-examination revealed that anisocoria and hemiplegia had resolved. However, due to progressive respiratory distress not responsive to naloxone and a decreasing level of consciousness, the patient was re-intubated (1 hour after the prior extubation). He was extubated again 103 min later, but was still agitated, aphasic, with mild tetraparesis.

A brain magnetic resonance imaging (MRI) with contrast injection (FLAIR, T2*, diffusion and perfusion weighted imaging, 3D, time of flight, and contrast-enhanced MR angiography sequences of the supra-aortic vessels) was performed urgently (5 h after the end of the surgery) and

revealed no sign of stroke. Upon re-examination, the patient had dysarthria, hyperreflexia and a severe tetraparesis; the NIH Stroke Scale scored was 21. The blood serum chemistry was normal. Faced with this picture of unexplained polymorphic neurological failure, a CAGE was suspected and the patient was placed in a hyperbaric chamber. He underwent a 180-min session of HELIOX B30 (compression to 405.2 kPa [four atmospheres absolute] breathing a 60% oxygen–40% helium mixture), 9 h after the end of surgery. Thirty minutes after the beginning of the session the patient displayed an improvement in phasic disorders and partial recovery of his tetraparesis. At the end of the session, the phasic disturbances had disappeared and a mild tetraparesis persisted. The symptoms were completely resolved a few hours later after the patient returned to the ward.

Discussion

Gas embolism refers to the entry of gas (often air) into the vasculature, which requires a pressure gradient favouring the passage of the gas in the blood vessel. In surgery this can occur particularly in sitting position but the risk of gras entrainment (particularly into the venous circulation) exists any time the operating site is above the level of the heart. During the prone position two factors can contribute to an AE: a gravitational gradient between the heart and the operative site, and a negative pressure within the epidural veins secondary to the decompression of the abdomen.[1] Air bubbles can then migrate into the systemic circulation via physiologic shunts or incomplete filtration by the pulmonary capillaries,[3] also called paradoxical air embolism. When bubbles reach the cerebral vasculature (i.e., CAGE), they may cause variable neurological symptoms from a sudden change in sensorium to disorientation or coma, mimicking an ischaemic stroke.[4] Although the neurological symptoms most commonly result from cerebral focal ischaemia and oedema from air emboli lodged in small cerebral arteries, these bubbles also induce an inflammatory process damaging the endothelial cells of the vessel wall. As with sitting surgery, surgery in the prone position is likely to have a higher than reported incidence of micro paradoxical embolism. However, the clinical significance of micro-CAGE is not clear.[5]

The outcome of CAGE depends on the diagnosis and treatment delays, which should be as short as possible. During the post-operative period, the early recognition of symptoms is difficult due to the confounding effects of anaesthesia. The motor deficit can be due to a residual muscle relaxation or a metabolic disturbance; the restlessness to pain or a bladder retention or drug side effects; the decrease in alertness to residual drug effects; and the pupillary anomaly to some drug side effects or an ischaemic optic neuropathy. In any case, a surgical complication must always be considered. However, certain intraoperative observations, such as unexplained sudden hemodynamic or respiratory failure or a fall of E_TCO_2, should raise a flag whenever the procedure is being performed in a risky position. CAGE can be responsible for various neurological disorders such as headache, alterations in consciousness, seizures, focal or multi-focal motor deficit, pyramidal syndrome, cranial nerve deficit, visual disorders, sensory disorders, and phasic disorders. Its onset is abrupt but its clinical expression may vary over time. It may also be associated with respiratory and/or haemodynamic signs such as pulmonary oedema, acute respiratory distress syndrome, tachycardia, ST segment changes, right heart failure, or cardiac arrest.[6] A high suspicion of AE is thus sufficient to justify treatment. Sometimes diagnosis of CAGE (by directly visualising vascular gas) can be made using cerebral imaging (computed tomography or MRI). However, air bubbles can be partially resorbed within the first hours, and the presence of air can no longer be demonstrated using brain imaging. Therefore, normal imaging should not exclude a diagnosis of CAGE.[7]

Recovery from CAGE can be spontaneous as reported in many cases but the use of hyperbaric oxygen is the treatment of choice,[8] as it decreases bubble size and increases oxygen solubility in plasma.[9] Access to a hyperbaric medicine centre can be the limiting factor for this treatment. Although hyperbaric oxygen treatment should be started as soon as possible there have been reports of significant clinical improvement even after considerable delays in treatment.[10] In the case presented here, once other differential diagnoses such as residual muscle relaxation, metabolic disturbances, ischaemic or haemorrhagic stroke were ruled out, the most plausible diagnosis was CAGE . Other differential diagnoses were mentioned and were not completely excluded (i.e., drug overdose, acute anticholinergic syndrome), although the clinical features were hardly compatible. Moreover, the rapid favourable evolution during hyperbaric oxygen treatment supported the CAGE hypothesis.

Conclusions

CAGE is a serious and often fatal event that can occur in every procedure at risk, i.e., when a pressure gradient favours the passage of the gas in a blood vessel. Due to the lack of symptom specificity, a high index of suspicion should be maintained to allow for prompt diagnosis and rapid treatment of the condition, and ultimately rapid recovery and reduced morbidity.

References

1 McDouall SF, Shlugman D. Fatal venous air embolism during lumbar surgery: The tip of an iceberg? Eur J Anaesthesiol. 2007;24:803–5. doi: 10.1017/s0265021506002201. PMID: 17924475.

2 Miyakoshi N, Hongo M, Kasukawa Y, Ishikawa Y, Kudo D, Shimada Y. Intraoperative visible air bubbling recorded as a sign of massive venous air embolism during prone position surgery for extensive ossification of spinal ligaments: a case report with a video clip. World Neurosurg. 2019;131:38–42. doi: 10.1016/j.wneu.2019.07.166. PMID: 31369880.

3 Berlot G, Rinaldi A, Moscheni M, Ferluga M, Rossini P. Uncommon occurrences of air embolism: Description of cases

and review of the literature. Case Rep Crit Care. 2018;2018:1–7. doi: 10.1155/2018/5808390. PMID: 30073096.

4 Suri V, Gupta R, Sharma G, Suri K. An unusual cause of ischemic stroke – Cerebral air embolism. Ann Indian Acad Neurol. 2014;17:89–91. doi: 10.4103/0972-2327.128562. PMID: 24753668.

5 Rodriguez RA, Sinclair B, Weatherdon D, Letts M. Patent foramen ovale and brain microembolization during scoliosis surgery in adolescents. Spine. 2001;26:1719–21. doi: 10.1097/00007632-200108010-00017. PMID: 11474360.

6 Coulter TD, Wiedemann HP. Gas embolism. N Engl J Med. 2000;342:2000–2. doi: 10.1056/nejm200006293422617. PMID: 10877658.

7 Kaichi Y, Kakeda S, Korogi Y, Nezu T, Aoki S, Matsumoto M, et al. Changes over time in intracranial air in patients with cerebral air embolism: radiological study in two cases. Case Rep Neurol Med. 2015;2015:1–5. doi: 10.1155/2015/491017. PMID: 26640730.

8 Moon RE. Hyperbaric treatment of air or gas embolism: Current recommendations. Undersea Hyperb Med. 2019;46:673–83. PMID: 31683367.

9 Sviri S, Woods WPD, van Heerden PV. Air embolism – a case series and review. Crit Care Resusc. 2004;6:271–6. PMID: 16556106.

10 Covington D, Bielawski A, Sadler C, Latham E. A favorable outcome despite a 39-hour treatment delay for arterial gas embolism: case report. Undersea Hyperb Med. 2016;43:457–61. PMID: 28763175.

Conflicts of interest and funding: nil

Submitted: 25 March 2021
Accepted after revision: 05 June 2021