



CASE REPORTS

Hyperbaric oxygen therapy in the treatment of pneumocephalus associated with epidural block: case report

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Abstract

Introduction: Pneumocephalus is a rare neuraxial blockade complication, which can be associated with severe neurologic changes.

Clinical case: A 51-year-old patient was submitted to left total knee arthroplasty. Postoperatively, a pneumocephalus associated with decreased consciousness was diagnosed as a complication of the epidural analgesia. The treatment used was Hyperbaric Oxygen Therapy (HBOT) due to the severity of symptoms. Just after one session of HBOT, there was nearly full pneumocephalus resorption and significant clinical recovery.

Conclusion: This case report enables anesthesiologists to recognize HBOT as a therapeutic option to be considered when treating severe cases of pneumocephalus.

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Introduction

Pneumocephalus is defined as the presence of air in the intracranial space, which can occur in any of the central nervous system compartments.¹ It is a rare complication of

neuraxial blockades that happens when air migrates from the site of the spinal puncture to the intracranial space.^{2–4}

The usual treatment for this condition is O₂ inhalation through a high-output mask, speeding up the reabsorption of nitrogen into the bloodstream and decreasing the volume of the intracranial gas bubble.¹ Hyperbaric Oxygen Therapy (HBOT) is an alternative option to conservative or surgical treatment due to its fast onset effect of reducing air volume and accelerating gas reabsorption.¹

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According to our literature search, there is only one published case in which HBOT was applied to a pneumocephalus related to neuraxial anesthesia technique.⁴ Here we report a patient that presented neurologic changes hours after a combined spinal epidural anesthesia secondary to pneumocephalus, successfully managed with HBOT.

Clinical case

A 51-year-old woman with past medical history of hypertension, obesity, peripheral venous insufficiency and gonarthrosis was submitted to total arthroplasty of the left knee. The procedure was performed under combined spinal epidural anesthesia. After intravenous administration of 100 µg fentanyl as premedication, at 4:30 PM spinal anesthesia was performed using 9 mg of hyperbaric bupivacaine associated with 5 µg sufentanil and, subsequently, an epidural catheter was inserted. The regional anesthesia technique details are unknown, particularly if the identification of the epidural space was performed with saline solution or air. During surgery, midazolam 3 mg and lidocaine 100 mg were administered intravenously at the end of surgery. The intraoperative period was uneventful. In the PostAnesthetic Care Unit (PACU), 4 mg morphine was administered via epidural catheter, the first injection being at 7 PM. Upon returning to the ward, drowsiness and slightly slurred speech were observed, assessed as being compatible with the immediate postoperative period.

At approximately 6:30 AM the medical emergency team was called due to altered consciousness (torpor state). At the initial assessment, the patient presented airway obstruction reversed by basic maneuvers, bradypnea requiring supplemental O₂ (SpO₂ 92% at 31% FiO₂), no signs of respiratory distress, and was hemodynamically stable. The initial neurologic examination was 4 on the Glasgow Coma Scale (E2V1M1), associated with poorly reactive miotic pupils, outward deviation of left eye, and right eye centered on the midline. After obtaining the result for blood glucose analysis of 182 mg.dL⁻¹, 0.4 mg naloxone was administered with partial improvement of consciousness to 12 points on the Glasgow Coma Scale (E4V4M4).

Neurology evaluation was requested, which reported persistent left central facial paresis, with no other neurologic deficits. Due to the persistent neurologic findings, after the administration of naloxone, a brain Computerized Tomography (CT) scan was requested, which revealed pneumocephalus at the suprasellar cistern, prepontine cistern and right Sylvian fissure (Fig. 1).

In view of these findings, we proposed the neuraxial injection of opioid and air as the combined etiology explaining the depressed level of consciousness and the focal neurologic deficit (facial paresis) that resulted from the anesthesia/analgesia management.

In view of the clinical severity, urgent treatment with hyperbaric oxygen therapy was proposed. Given the required management of the depressed level of consciousness, hypoventilation and the need for naloxone infusion, patient sedation and tracheal intubation were performed prior to transferring to the Hyperbaric Medicine Unit (UMH). The epidural catheter was removed before the patient entered the hyperbaric chamber.

The UMH has a trained team and equipment to deal with level III medical care (critical patient) in a category 1 multi-room chamber that is continuously monitored by a staff of the attending team (doctor or nurse).

The patient was maintained on invasive mechanical ventilation under sedation and analgesia with propofol, fentanyl, and muscle relaxation with rocuronium, continuously monitored with ECG, invasive blood pressure, urine output, and respiratory gases analysis. The protocol consisted of a hyperbaric oxygen therapy session according to the US Navy Treatment Table 6, maximum pressure of 2.8 ATA (absolute atmospheres) and total run time of 4 hours and 45 minutes. The therapy session was uneventful.

Then, the patient was sent back to the institution of origin, admitted to the Intensive Care Unit (ICU), where immediate tracheal extubation was performed. The reassessment brain CT scan showed signs of nearly complete pneumocephalus resorption, with only a small air-bubble present in the right paramedian side of the Sella turcica. Upon neurologic examination, the patient maintained a minor left nasolabial groove erasure, without any other neurologic deficit. Pain control was achieved with multimodal systemic analgesia combined with tramadol 100 mg every 8 hours.

The patient was kept in the ICU for two days showing good clinical progress, except for a respiratory infection treated with antibiotic therapy, not requiring O₂ supplement. The patient remained in the orthopedic ward for 7 days until being discharged home.

Discussion

Epidural anesthesia/analgesia is a technique widely performed in anesthetic practice, especially for labor analgesia. However, intracranial hypotension, toxicity by local anesthetics, total spinal anesthesia, unintentional vascular and dural puncture, low back pain, subdural blockade, and iatrogenic pneumocephalus are possible complications.²

The intracranial space behaves as a closed system due to its rigid structure comprising brain tissue, cerebrospinal fluid, and vascular system. Any disruption to this pressurized closed system that creates an opening point, has the potential to promote the development of pneumocephalus due to the entry of air.²

During epidural anesthesia, a path is created that can facilitate the air penetrating into any cranial spaces or into the cortex tissue itself. This air penetration is classified as intra-axial (cortex, ventricles, vasculature) and extra-axial (epidural, subdural, subarachnoid).²

Pneumocephalus is believed to be caused by two mechanisms: the ball valve and the inverted bottle effect.² According to the ball valve mechanism, air penetrates due to increased external pressure because of a dura defect that overcomes intracranial pressure. Once inside the skull, the air has no exit due to obstruction by the cranial structures. The inverted bottle mechanism states that when there is a negative intracranial pressure due to a cranial dura-mater defect or cerebrospinal fluid leak, there are air entries by aspiration to balance the pressure differential.²

The clinical presentation of pneumocephalus is variable and depends both on intracranial distribution and volume

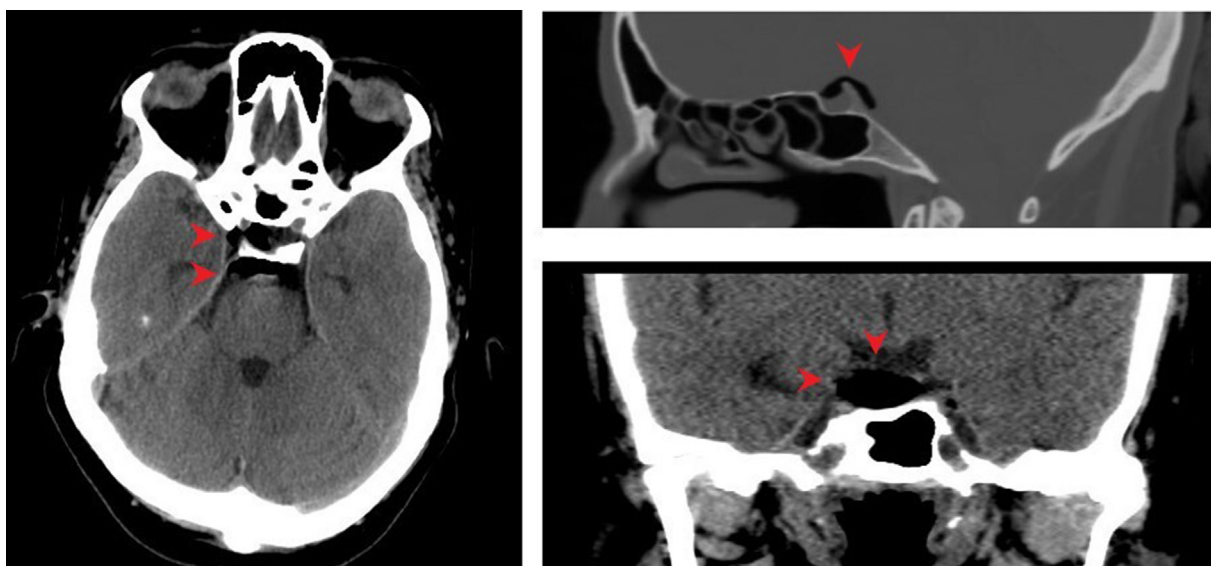


Figure 1 CT brain scan performed before HBOT treatment. Arrows – pneumocephalus at suprasellar space and prepontine cistern.

of air.² Most of the cases of patients with pneumocephalus are asymptomatic, however headache is the most common complaint. Other symptoms may include nausea, vomiting, seizures, syncope, agitation, delirium, changes in deep reflexes and in the level of consciousness.² This clinical spectrum overlaps the symptoms of a much more frequent complication of neuraxial blockades: Postdural Puncture Headache (PDPH).

This case describes a recognized, albeit infrequent, complication of neuraxial techniques that results from unintentional administration of drugs and air into the intrathecal space, with potentially severe and persistent neurologic damage, and which may have variable and late manifestations that delay its early diagnosis.

Of the cases described in the literature in which the epidural technique led to an unintentional dura mater puncture and consequent air injection, only one used the loss of resistance technique with saline solution.³ All other described cases, according to our literature search, resulted from the loss of resistance technique using air.^{2,4} In this situation, clinical symptoms are typically immediate and mediated by the fast migration of the air.²⁻⁴

However, this present case is distinguished by its late onset, which raises some questions related to what caused opioid and air entry into the intrathecal space, and which was not elucidated by the anesthesia record. Most probably, morphine administered at the PACU was injected into the intrathecal space and together with an air bubble (due to an error in the administration technique) promoted the displacement of a pre-existing air bubble from the spinal to the intracranial space, driven by the positive pressure of the injection and the possible slightly head up positioning of the patient on the PACU bed.

This case report highlights the relevance of stringent adherence to the techniques for handling epidural catheters, and administering drugs via this route, in addition to identifying the epidural space using saline solution, as preventive strategies for pneumocephalus of iatrogenic etiology.

Nitrogen makes up 79% of atmospheric air and is a poorly soluble gas. For this reason, when present in tissues, it persists for extended periods, favoring the compressive effect of the air bubble over the nervous system structures, with consequent injury.

Hyperbaric oxygen therapy consists of the administration of pure O₂ in a high-pressure environment. The therapeutic effect of HBOT derives from the significant increase in partial oxygen pressure in all body tissues, according to the physiological diffusion gradient, i.e., the oxygen cascade. In the case of pneumocephalus, hyperbaric oxygen therapy works by two mechanisms: high pressure immediately decreases the volume of air pocket, according to Boyle's Law (the volume of a gas is inversely proportional to the pressure)⁴; in addition, inhalation of 100% oxygen significantly accelerates nitrogen elimination, consequently, air pocket reabsorption.¹

HBOT is not an ordinary therapeutic option for treating pneumocephalus. However, the severity of the condition encouraged us to use this technique, due to its greater effectiveness in treating the cause, already extensively revealed in cases of gas embolism and decompression illness that are caused by deleterious effects of gas pockets over tissues.⁵

After just one session, HBOT enabled nearly the complete resorption of the intracranial gas pocket and the fast and clinically significant neurologic recovery. These results match those described by Shih et al.,⁴ revealing the effectiveness of this treatment in this rare anesthetic complication.

This case report allows raising awareness of anesthesiologists to consider HBOT as a therapeutic option for the treatment of a severe case of pneumocephalus after epidural anesthesia.

Conflicts of interest

The authors declare no conflicts of interest.

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