

CASE REPORT

Case Report: Pneumocephalus after labor epidural anesthesia [version 1; referees: 2 approved, 1 approved with reservations]

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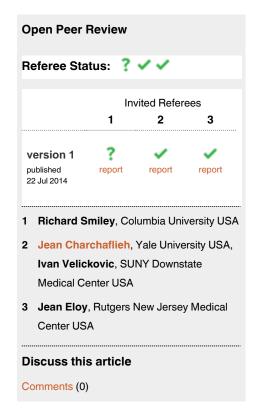
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Abstract

Lumbar epidural anesthesia is commonly used for labor analgesia. The 'loss-of-resistance' to air technique (LORA) is generally employed for recognition of the epidural space. One of the rare complications of this technique is pneumocephalus (PC). Here we describe the case of a parturient who developed a frontal headache when locating the epidural space using LORA. On the second day after epidural injection, the patient exhibited occipital headaches with gradual worsening. Computed tomography scans of the brain indicated PC. Following symptomatic treatment, our patient was discharged on the 13th day. We concluded that the amount of air used to identify the epidural space in LORA should be minimized, LORA should not be used after dural puncture and the use of saline avoids PC complications.



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Introduction

Lumbar epidural anesthesia is a commonly used technique for analgesia during labor. The complications often associated to this technique include unilateral analgesia, extended epidural blockade, unplanned puncture of the dura or of a blood vessel, post-puncture dural headache (PDPH), subdural blockade, placement of the catheter out of the epidural space and neurological complications¹. The 'loss-of-resistance' to air technique (LORA) is commonly employed for recognition of the epidural space. Nevertheless, one of its rare complications is pneumocephalus (PC)².

Cases of PC following neuroaxial anesthesia have been described either after an epidural using the LORA technique or after spinal anesthesia^{3,4}. The development of PC after spinal anesthesia is exceptionally rare^{5,6}. PC symptoms are difficult to distinguish from other complications of the epidural technique such as PDPH or neurotoxicity. Diagnosis relies on clinical impression and brain tomography (CT-scan)². Our goal is to report the case of a patient who presented PC following labor epidural anesthesia.

We attempted to approach the patient and the patient's family to obtain informed consent for publication of this report, However we decided to abandon telephone contact after numerous unsuccessful attempts. The Institutional Review Board at Complexo Hospitalario Universitario A Coruna determined that approval of the case report was not required.

Clinical case

A 34 year old healthy Caucasian parturient ASA II, G1PO was admitted at 38 weeks of gestation. Her clinical history included no allergies to medications, no relevant family history, mild bicuspid aortic valve stenosis, cervical aortic arch and mild postductal coarctation of the aorta. She was not taking any medications. She presented occasional asymptomatic palpitations at rest (few seconds long). Her cardiopathy was compensated and well controlled during pregnancy.

At 4-5 cm of cervical dilatation, at the request of analgesia, a lumbar epidural was proposed for management of labor pain. The patient was monitored with ECG, SpO2, non-invasive blood pressure (NIBP) every 5 minutes and a peripheral vessel catheter was in situ. Puncture was performed at the L4-L5 interspace with a 18 G Tuohy epidural needle (Perifix® 401 Braun Germany) (80 mm/31/4" long) via the median approach with the patient in the sitting position. After locating the epidural space using the LORA technique (approximately 3 ml of air), the patient developed a sudden intense frontal headache. No neurological, haemodynamic changes or breathing symptoms were detected. The needle was withdrawn without cerebrospinal fluid (CSF) flashback. The cephalalgia improved gradually ceasing several minutes later. An epidural catheter was introduced afterwards (Perifix® 401 Braun Germany, close-end, three lateral holes) in the L3-L4 interspace with the patient in the sitting position, after locating the epidural space using loss of resistance to saline (LORS). We confirmed its appropriate location by checking that CSF had not been aspirated prior to local anesthetic injection. The test dose of 3 ml of bupivacaine 0.25% and epinephrine resulted negative. An initial bolus of 5 ml of bupivacaine 0.25% and 50 mcg of fentanyl was administered and a continuous infusion of ropivacaine 0.18% in addition to 1 mcg of fentanyl per ml was

programmed at 7 ml/hour. This process was incident-free, being the epidural analgesia correct and labor with normal evolution.

On the second day postpartum, the patient presented occipital headaches which increased on standing, accompanied by tinnitus, nausea and vomiting, with gradual worsening. We found no alterations in the neurological examination. We decided conservative treatment with rest, NSAIDs, intravenous hyperhydration and 300 mg of oral caffeine twice daily, showing mild clinical improvement. However, on the 6th day after delivery, due to mild clinical worsening, and suspecting iatrogenic PC vs. PDPH, a brain CT-scan was performed. The CT scan revealed air in the temporal horns and right frontal horn of the lateral ventricle, showing a PC in the subarachnoid space and ventricles that was responsible for mild ventricular dilation (Figure 1 and Figure 2). A subdural collection that could

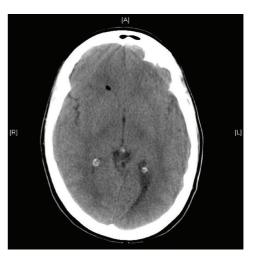


Figure 1. Axial CT scan shows the presence of a gas bubble in the ventricular system following dural puncture, in the right frontal cistern horn of the lateral ventricle.

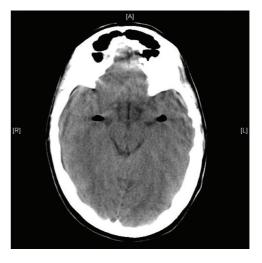


Figure 2. Axial CT scan shows the presence of a gas bubble in the ventricular system following dural puncture, in both temporal cistern horns.

correspond to an inflammatory reaction was also observed. At all times the neurological examination was normal. We consulted the Neurosurgery Service, which considered the PC not significant and recommended to continue with the conservative treatment and repeat the CT in 48–72 hours or if neurological changes occurred.

Despite treatment, at the 8th day after puncture, the patient's symptoms worsened at night with severe frontal headaches (Visual Analog Scale 6) and an increase in tinnitus. She was afebrile, the vital signs were unremarkable, and she had no focal neurological deficits. A control CT scan showed a mild regression of the PC in the ventricular system, but demonstrated persistence of the subdural collection. We decided to continue with the conservative treatment and performed control blood tests (routine hematological; liver and kidney function tests; serum electrolytes; coagulation profile).

On the 13th day after delivery, the follow-up was uneventful, the patient showed no abnormalities at the neurological examination and her vital signs were normal. The patient was finally discharged from the anesthesiology department being completely asymptomatic.

Discussion

PC is relatively common in neurosurgery⁷ and neuroradiology^{8,9}. It can be caused by trauma¹⁰ or infections^{11,12}. It may develop after lumbar puncture, epidural steroid injection, or Valsalva's maneuver^{13–15}. The development of PC after spinal or epidural anesthesia is extremely infrequent. The incidence of PC after epidural steroid injections or epidural anesthesia is unknown, and only few cases per year are described in the literature^{16,17}. Most cases of PC due to epidural techniques have been associated with LORA, as described in our case report.

PC, an unusual consequence of evident or unnoticed accidental dural puncture^{3,4,18}, develops from the injection of air into the subarachnoid or subdural space and cranial migration¹⁹. PC is not often followed by symptoms, but among those, headache is the most frequent^{20,21}. The appearance of other symptoms, such as signs of space-occupying lesions (focal neurologic deficits including cranial nerve palsies 19,20, or diverse motor signs) or augmented intracranial pressure and cardiovascular instability may develop depending on the spread and extent of intracranial air²². Headache is caused by the fast brain motion resulted from air injection and meningeal irritation²¹. Most cases consist of abrupt intense frontotemporal cephalea^{5,23,24}, as in our case study, having a premature beginning (same day) and commonly concluding within 5 days. It is exacerbated by motion and may not be alleviated by lying down². Roderick et al. outlined that 2 ml of air injected into the subarachnoid space was sufficient to provoke a symptomatic PC⁵.

In case of PDPH, due to CSF outflow, the pain is exacerbated by sitting or standing and is alleviated by lying down, having a characteristic occipital, frontal and post-orbital situation. It happens more often 24 to 48 hours after dural puncture²⁵ and is longer lasting than in PC². Although there may be subtle clinical differences with PC, their symptoms usually are interchangeable so the differential diagnosis must be done through CT.

A number of techniques to find the epidural space have been defined²⁶. LORA and LORS are the most common methods used².

Potential inconveniences of using saline comprise the difficulty to ascertain a meningeal puncture²⁷. On the other hand, if air is forced quickly by digital pressure, false positives may result, or gas embolism, subcutaneous emphysema²⁸ or multiradicular syndromes²⁹.

Accidental dural puncture is not always evident, as also shown in our case. Okell and Springge³⁰ described a 0.6% incidence of dural punctures in epidural anesthesia. These punctures were acknowledged by the loss of CSF, by aspiration through the catheter, by hypotension after injection of a test dose, and retrospectively. Hardy³¹ reported that an epidural catheter cannot easily be passed through the dura, but the arachnoid can be penetrated smoothly. The author deduced that when a catheter goes into the subarachnoid space it is due to its initial subdural placement and movement to the arachnoid, as the first hole in the dura yields migration of the catheter². Air introduced into the subdural space is more painful than in the subarachnoid space and reaches rapidly the head, because of its low pressure and diminished capacitance^{32,33}.

These findings explain the sequence in our case. We found no evidence of dural puncture with CSF flashback with the needle insertion at the L4-L5 interspace, but the dura had already been penetrated, allowing the passage of air likely into the subdural space and causing the abrupt headache. Despite our uncertainty regarding dural puncture, we correctly avoided LORA and used LORS for the following attempt to localize the epidural space.

In our case, PC was diagnosed from the CT scan. While subdural space is not straightly connected to the subarachnoid space³⁴, it is annexed to the floor of the third ventricle in the cranial cavity from the lower border of the second sacral vertebra^{35,36}.

The treatment of PC consists on administration of 40–100% oxygen in the supine position³⁷. This is to favor the reabsorption of intracranial air by intensifying the diffusion concentration gradient for nitrogen between the air collection and the surrounding cerebral tissue³⁸. Nitrous oxide should be avoided to prevent the expansion of PC³⁹. In addition, we should administer aggressive hydration, caffeine, or analgesics^{40,41}. Epidural infusion or blood patch have no effect on PC^{3,19}.

There is usually reabsorption of the air within 3–5 days from the epidural injection and patients commonly improve without any neurologic abnormalities. Our patient was discharged after clinical-radiologic resolution on the 13th day. If tension PC occurs, a neurosurgical emergency treatment may be necessary^{21,23,42}.

In conclusion, the way to minimize the likelihood of PC when performing epidural block with the loss of resistance technique is to use saline instead of air^{43,44}. When air is used, we should minimize its extent. In addition, LORA should not be used after dural puncture.

After epidural block, and particularly when dural puncture is performed, the patient should be monitored carefully. We should identify that the headache from PC after epidural anesthesia occurs commonly immediately after puncture. Likewise, we should recognize that symptoms of PC are similar to those of PDPH and that a differential diagnosis is established by imaging techniques.

Lastly, we should be able to assess that PC may be spontaneously absorbed, managed with symptomatic treatment.

Consent

After numerous unsuccessful attempts to contact the patient and the patient's family to obtain informed written consent for publication of this report, the Institutional Review Board at our Institution determined that approval of the case report was not required.

Author contributions

Beatriz Nistal-Nuño: writing and elaboration of the entire manuscript (all sections of the manuscript-abstract, introduction, case

description, discussion, references, including its translation and editing), collection of data, and interpretation of the clinical case.

Manuel Gomez-Rios: collection of data.

Both authors revised the manuscript and agreed to the final content.

Competing interests

No competing interests were disclosed.

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Open Peer Review

Current Referee Status:





Version 1

Referee Report 14 August 2014

doi:10.5256/f1000research.5014.r5559



Jean Eloy

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The case report: "Pneumocephalus after epidural anesthesia" is a well written manuscript. The Hypothesis is sound and the originality of the manuscript evident. The report is also relevant to clinical practice, shows appropriate methodology and is properly referenced. However, in the introduction the author states that the development of PC after spinal anesthesia is exceptionally rare - which it is not. I would omit the word exceptionally from the manuscript, as PC is often confused with post dural puncture headache and is often underreported.

As the author mentioned in the conclusion section, using loss of resistance to saline instead of air is a way to minimize the incidence of PC. However the author does not expand on which technique is used the most by practitioners and taught in residency programs. Although this case report is hardly the definitive paper on Pneumocephalus after epidural analgesia, it certainly offers guidance and raises awareness on its incidence.

- 1. From the case report, the author report sudden onset of frontal headache that improved within several minutes. There was no mention in the case report or the discussion on when the epidural catheter was removed and the temporal relationship with the patient occipital headaches.
- 2. The signs and symptoms of PDPH and PC are the same. Are the symptoms reported by the author the results of PDPH or PC? This has not been made

I have read this submission. I believe that I have an appropriate level of expertise to confirm that it is of an acceptable scientific standard.

Competing Interests: No competing interests were disclosed.

Referee Report 04 August 2014

doi:10.5256/f1000research.5014.r5543



Jean Charchaflieh, Ivan Velickovic

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The authors provided an excellent description of a case of pneumocephalus (PC) after epidural catheterization, without evident dural puncture, with very informative discussion of the pathophysiology, symptomatology and management. The authors discussed briefly some of the advantages and disadvantages of using loss of resistance to air (LORA) versus loss of resistance to saline (LORS) in identifying the epidural space. The authors provided three recommendations for the prevention of PC after epidural catheterization, which are not necessarily supported by empirical evidence or consensus of practitioners. The three recommendations are:

1. Use LORS instead of LORA to minimize the likelihood of PC after epidural catheterization.

This recommendation is not supported by empiric evidence or consensus of practitioners. Indeed one of the cited references in support of this recommendation indicates that all three reported cases of PC occurred after LORS; and that the general practice in UK is about 50% LORS and 50% LORA.

43 in main article

As the authors noted, one of the potential disadvantages of LORS is clouding the certainty of the occurrence of dural puncture.^{27 in main article}

Another potential disadvantage of LORS is creating a pocket of fluid in front of ligamentum flavum that allows placing the epidural catheter in front of ligamentum flavum instead of the epidural space.

The argument that LORS minimizes the likelihood of PC after epidural catherization is not clear cut and not the overriding consideration in choosing LORA vs. LORS.

2. Minimize the amount of air when using LORA.

When using LORA, most practitioners would argue in favor of limiting the amount of injected air to 1-2 ml, and against the use of multiple confirmatory injections of air into the epidural space. However as the presented case demonstrate, PC can occur even when performing LORA with approximately 3 ml of air and without evident dural puncture.

3. LORA should not be used after dural puncture.

This recommendation is not supported by empiric evidence and not universally endorsed by practitioners. As noted above, LORA can be more advantageous than LORS at detecting dural puncture. As for the immediate management of dural puncture, interventions can vary from inserting a catheter into the intrathecal space to reattempting the epidural catheterization at a different lumbar level. No specific technique has proven an advantage or disadvantage regarding the risk of PC. Indeed, as the presented case demonstrates, PC can occur even when LORS is used after initial LORA without excessive amount of air and without evident dural puncture.

It is worth noting that post-dural puncture headache (PDPH) can co-exist and outlast headache due to PC. One of the referees (IV) reported a case of PC for 3 days, followed by resolution of PC and subsequent occurrence of typical PDPH, which resolved with treatment with epidural blood patch (Velickovic & Pavlik, 2007).

Overall, the authors presented a well-balanced case report and discussion, which can serve as a handy review for diagnosis and management of PC after epidural catheterization.



We have read this submission. We believe that we have an appropriate level of expertise to confirm that it is of an acceptable scientific standard.

Competing Interests: No competing interests were disclosed.

Referee Report 29 July 2014

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Richard Smiley

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The authors have reported an interesting case of headache after a lumbar epidural procedure. There is value in reminding anesthesiologists of the potential for pneumocephalus (PC) when air is used for the loss-of-resistance procedure. Their description makes it clear that the patient developed an almost immediate headache, which resolved rather quickly. This was followed by a more chronic, at least partially postural headache starting day 2 after the procedure. While the initial headache was almost certainly due to air injection into the subdural or subarachnoid space and a resulting PC, the subsequent headache may well have been a more classic post-dural puncture headache (PDPH), perhaps with some residual from the PC, given its time course (starting day 2, continuing for the better part of two weeks, which is later and longer than most PC headaches, but exactly what one might expect from a PDPH).

The authors mention that PC headaches typically last less than 5 days (the few we have seen have tended to last hours not days). A bit more discussion of this possibility/probability could be in the manuscript. It is possible, especially when one considers that the neurosurgeons considered the PC ""not significant," that the patient could have been treated with a epidural blood patch to relieve her headache and shorten her hospital stay. In general, PC headaches are not as obviously postural as PDPH, so while certainly symptoms overlap, I am not sure it is fair for the authors to state that the symptoms are "interchangeable."

The authors suggest that because no CSF was seen with the initial placement the Tuohy needle may have been in the subdural space, and give an anatomic explanation of how air gets form the subdural to the intracerebral subarachnoid space; this is a possible explanation, but it is also possible that the air was injected subarachnoid but the needle withdrawn before CSF was appreciated. It is interesting that Roderick, in 1985 (ref 5 in this case report) reported a PC after injection of ~ 2ml air during attempted spinal anesthesia; in that case too there were problems obtaining CSF; perhaps they were subdural.

There are some unimportant issues with English usage that do not detract from understanding the main points of the case report.

I am not entirely sure I understand the mechanism of headache from PC as described in the case report. The authors cite "fast brain motion" and "meningeal irritation." Any brain movement must be short-lived, explaining only the immediate, initial headache, and I am not sure how air is much of an "irritant." Unless the air is of a very significant volume, it seems likely that it has quite small effects after a few hours or a day or two; hence my suspicion this case is more a PDPH than PC headache.



I have read this submission. I believe that I have an appropriate level of expertise to confirm that it is of an acceptable scientific standard, however I have significant reservations, as outlined above.

Competing Interests: No competing interests were disclosed.