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An Unusual Case of Symptomatic Pneumocephalus Following Lumbar Epidural Steroid Injection Performed with Saline Loss-of-Resistance Technique

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Background: Lumbar epidural steroid injections are commonly employed for management of chronic low back pain and lumbar radiculopathy, and are rarely associated with complications such as pneumocephalus. Pneumocephalus classically occurs in the setting of unintentional dural puncture during epidural steroid injection performed with the air loss-of-resistance technique. Herein, we present an unusual case of symptomatic pneumocephalus following lumbar epidural steroid injection without clear dural puncture performed with the saline loss-of-resistance technique.

Case Report: The patient is a 77-year-old woman with a past medical history of anxiety and low back pain due to degenerative disc disease and foraminal stenosis who was referred to our pain medicine clinic and underwent 5 successful, uncomplicated lumbar epidural steroid injections over the course of 2 years. During her sixth lumbar epidural injection, access to the epidural space was attempted at the L5-S1 interspace but this was not possible due to extensive bone overgrowth and osteophytes. Epidural access was obtained at L3-L4 via the saline loss-of-resistance technique, with clear epidural contrast medium flow prior to the injection of medication. The patient sat up from the procedure table and developed a severe left-frontal headache that lasted for fewer than 30 seconds; the headache recurred 2 additional times in the hour immediately following this procedure and was not accompanied by any neurologic deficits. She was referred to the emergency department for further evaluation, where noncontrast computed tomography head demonstrated pneumocephalus. She was managed conservatively with as-needed pain medications, and her headaches resolved without further intervention within one week of the procedure.

Conclusion: Pneumocephalus following epidural steroid injection most commonly occurs as a result of unintentional dural puncture and is much more common when the air, rather than saline, loss-of-resistance technique is employed. This case demonstrates that it is possible for a patient to develop symptomatic pneumocephalus following epidural steroid injection with the saline loss-of-resistance technique. It is prudent to consider performing epidural steroid injections with the saline loss-of-resistance technique to decrease the risk of postprocedural headache.

Key words: Lumbar epidural steroid injection, pneumocephalus, postprocedural headache

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BACKGROUND

Lumbar epidural steroid injection (LESI) is a commonly employed nonsurgical interventional technique utilized to treat lumbar radiculitis, lumbar spinal stenosis, and chronic low back pain. Serious complications resulting from LESI, including epidural abscess, epidural hematoma, intravascular injections, nerve trauma, dural puncture, disc entry, air embolism, and pneumocephalus, are rare (1). Pneumocephalus is defined as the presence of air in the epidural, subdural, or subarachnoid space and within the ventricles and parenchyma of the brain. It most commonly forms traumatically, spontaneously, as a sequela of infection or postprocedurally, and can be a complication of dural puncture from LESI (6-7). The incidence of pneumocephalus as a complication of LESI appears to be much higher after the use of air loss-of-resistance (LOR) than with saline LOR (5). Dural puncture incidence during LESI occurs in fewer than 1.3% of cases, and the incidence of postdural puncture headache (PDPH) when dural puncture does occur ranges from 16% to 86% (2-3). PDPH is more common in cases of dural puncture with pneumocephalus than in cases of dural puncture without pneumocephalus (4).

Pneumocephalus may be accompanied by neurologic signs, including cranial nerve deficits, focal weakness, nausea/vomiting, altered mental status, and coma in cases of tension pneumocephalus (8). While plain skull films can be used to diagnosed pneumocephalus, small quantities of air may be missed. Magnetic resonance imaging (MRI) of the brain has intermediate sensitivity and specificity for the diagnosis of pneumocephalus. Computed tomography (CT) of the head remains the gold standard for diagnosis and may detect as little as 0.55 mL of intracranial air (6). Treatment of simple (nontension) pneumocephalus includes bedrest, with the head of the bed elevated no more than 30 degrees, administration of analgesics, and administration of oxygen to quicken the resorption of intracranial nitrogen. We present a unique case of a patient found to have pneumocephalus immediately following a seemingly uncomplicated lumbar ESI performed with saline LOR.

CASE

The patient is a 77-year-old woman with a past medical history of hypertension, anxiety, irritable bowel syndrome, and low back pain due to multilevel degenerative disc disease, spondylolisthesis, and foraminal stenosis who was referred to our pain medicine clinic in December 2017 by neurosurgery for consideration of LESI. The patient had developed pain 20 years prior to presentation to our clinic; her pain had worsened after a remote motor vehicle accident. She had undergone remote LESIs at an outside pain clinic with benefit for 2 to 3 months, axial low back pain subsequently returned then worsened in the months prior to presentation to our clinic and did not improve sufficiently with physical therapy or over-the-counter pain medications. The patient preferred intermittent injection therapy to chronic oral medications if sufficient pain relief could be achieved with injection therapy. She had required no prior spinal surgeries. MRI lumbar spine without contrast medium (completed in 2016, largely unchanged in 2018) demonstrated grade 2 spondylolisthesis of L5 on S1 with bilateral pars defects and advanced bilateral foraminal stenosis at that level, multilevel broad-based disc bulging with advanced disc space narrowing from L2-L3 through L5-S1, and mild central canal stenosis at L4-L5 (per radiology read). The patient had no history of migraine or tension-type headaches, no history of diabetes, and did not take any blood thinners. She underwent successful LESI 4 times at L5-S1 and one time at L4-L5 through our clinic without complication between January 2018 and July 2019. She had good pain relief and functional improvement following injections that lasted 2.5 to 4-plus months.

The patient presented for repeat LESI in October 2019 in the setting of recurrence of her predominantly axial low back pain. There were no changes to her medical history or clinical presentation at her office visit in October 2019 for repeat LESI. For the LESI, the skin was prepped, draped, and anesthetized. A 20-guage Tuohy needle was advanced towards the L5-S1 interspace utilizing the LOR technique with preservative-free normal saline with verification of depth utilizing the contralateral oblique view. Placement of the needle in the L5-S1 interspace was not possible due to extensive bone overgrowth and osteophytes. The needle was withdrawn. The skin overlying the L3-L4 interspace was anesthetized and the Tuohy needle was advanced towards the L3-L4 interspace, again utilizing the LOR technique with preservative-free normal saline, and again with verification of depth utilizing the contralateral oblique view. After LOR was obtained, contrast medium was injected and demonstrated good epidural flow. A total of 80 mg of methylprednisolone plus 3 mL of preservative-free normal saline was injected and the needle was withdrawn. The patient tolerated the procedure well while lying supine. At no point was

cerebrospinal fluid (CSF) aspiration or leak observed during the procedure.

The patient developed a moderate-to-severe left frontal headache after sitting up from the exam table; this lasted for a few seconds and then resolved. She was able to walk to the recovery area and experienced a second identical headache, which again lasted a few seconds. Her vital signs were stable, with her blood pressure less than 180/90 mm Hg on multiple checks despite forgetting to take her prescribed lisinopril the morning of the procedure. No focal neurologic signs were present, cranial nerves were within normal limits, and strength in her bilateral upper and lower extremities was 5 out of 5 on exam by the chronic pain fellow. The patient denied nausea, disorientation, visual disturbance, slurred speech, weakness, fevers/chills, and other symptoms associated with the headache aside from feeling anxious. Her headache completely resolved within seconds as she lay on the exam table in a dark, quiet exam room. She was monitored in the clinic for 30 minutes after the procedure without recurrence of headache and was discharged with no headache. Her headache returned in the lobby as she was paying her parking ticket and again lasted less than 30 seconds. She returned to the clinic and was instructed to go to the emergency department (ED).

The patient presented to the ED and was without headache during her entire ED course. Physical exam and vital signs during her ED course were unremarkable. CT head was performed to rule out subarachnoid hemorrhage (Fig.1), and per radiology read demonstrated the following: "There is pneumocephalus primarily

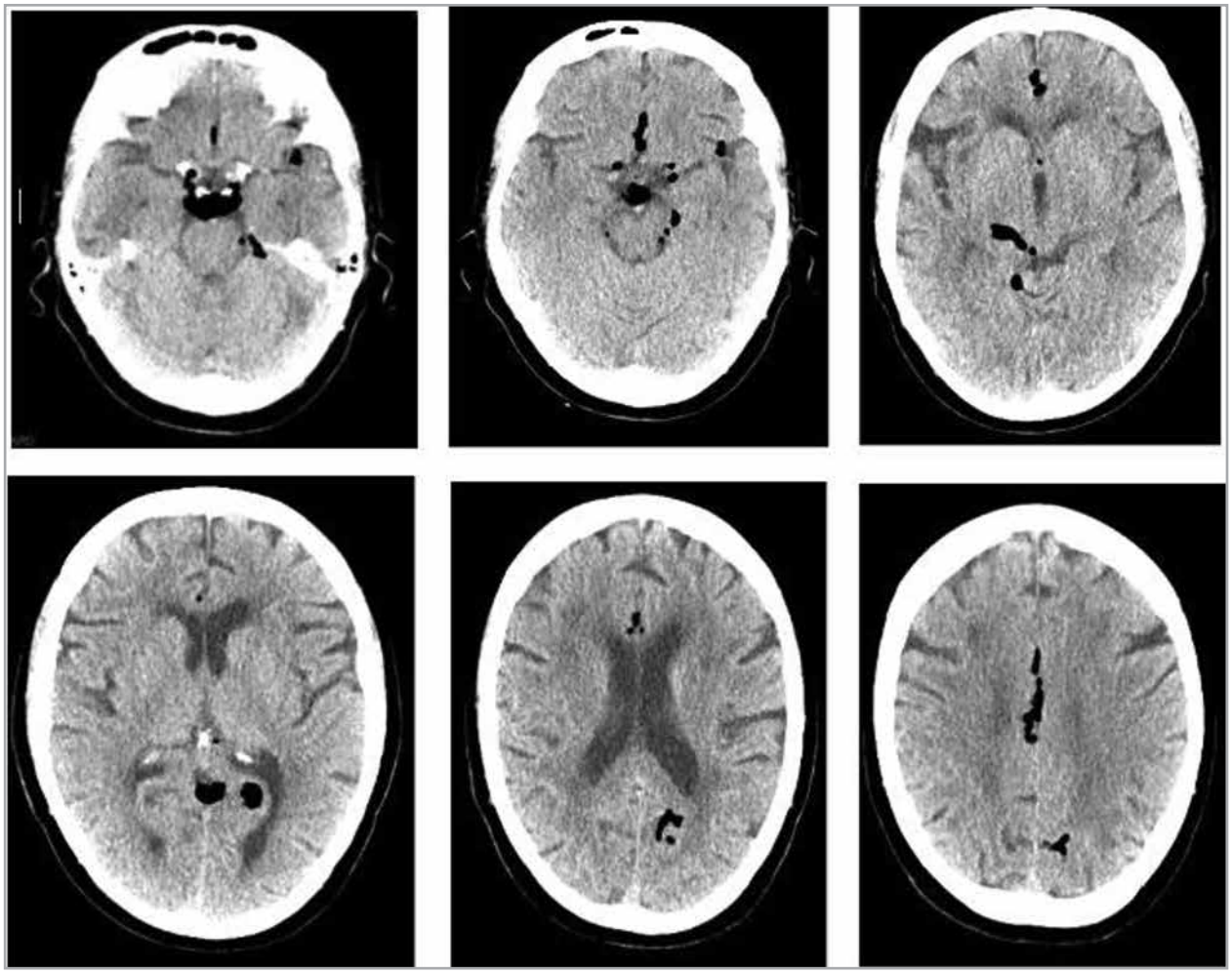

Fig. 1. Select axial slices from noncontrast CT head performed during ED course, which demonstrate the finding of pneumocephalus.

centered within the basal cisterns with air extending to involve the prepontine and quadrigeminal cistern on the left, the left sylvian fissure, along the falx and medial left occipital sulci. No acute intracranial hemorrhage, mass effect, midline shift, or extra-axial collection is identified. The ventricles are normal in size and configuration for the patient's age. The gray-white differentiation is preserved. The basal cisterns are patent. The paranasal sinuses and mastoid air cells are well aerated. The calvarium is intact." Neurosurgery was contacted via telephone and recommended against any further neurosurgical intervention or evaluation. Repeat CT head was not recommended. The patient was advised that the area of pneumocephalus would gradually reabsorb, but should she develop any severe worsening headache, numbness, tingling, weakness, or vomiting she should return to the ED. The patient was then discharged in stable condition.

The patient developed intermittent brief, nonpositional headaches over the subsequent week which gradually lessened in frequency and severity before resolving. At no point did she develop weakness, cranial nerve deficits, or other neurologic symptoms. She cancelled a flight that she had planned one week after injection due to the pneumocephalus. She did not have recurrence of headache after the one-week postprocedural time period. The patient underwent caudal ESI 4 months later without incident.

DISCUSSION

This is a unique case of headache following LESI likely secondary to small-volume pneumocephalus of unusual etiology. This patient's headache was most likely related to her pneumocephalus given the character of her headache (which occurred immediately following LESI), the short duration, the nonpositional quality, and spontaneous resolution without treatment. Headaches due to CSF leak typically begin more than one hour after a dural puncture occurs, are positional, and last longer than 30 seconds at a time (1,5). While pneumocephalus is commonly associated with headache, patients vary in terms of the volume of air needed to precipitate symptoms, with some patients experiencing headache with as little as 2 mL of air in the subarachnoid space and others asymptomatic with as much as 50 mL of air in the subarachnoid space (9). Given that CT head can detect as little as 0.55 mL of air in the subarachnoid space and the mechanism of air entry into the subarachnoid space, in this case, it is likely that our patient was very sensitive to small volumes of air (5-6).

There was no clear evidence of dural puncture during the case, with no CSF leak evident when epidural access was attempted at the L5-S1 interspace and very straightforward epidural access obtained at the L3-L4 interspace with clear epidural spread of contrast medium prior to steroid injection. Even in the absence of a clear CSF leak, the etiology of pneumocephalus is most likely secondary to unintentional dural puncture. A less-likely explanation is that low pressure within the patient's venous system and subarachnoid space during inspiration allowed air that had been introduced to the epidural space to be forced into the epidural venous plexus and translated to the subarachnoid space in the setting of high-pressure, preservative-free normal saline injection at the L5-S1 interspace (10).

CONCLUSION

If the etiology of our patient's headache was pneumocephalus secondary to dural puncture, this case is unusual, as a saline rather than air LOR technique was utilized to obtain epidural access. A review of the literature reveals that pneumocephalus following ESI is rare, with fewer than 40 cases published since 1919 (9). The vast majority of cases with documented pneumocephalus utilize an air LOR technique (9). One case of pneumocephalus after cervical ESI utilizing a hanging drop technique was reported; all other reported cases found via literature review occurred in the setting of air LOR technique (10). Use of an air-filled LOR syringe is associated with increased risk of both PDPH and pneumocephalus (5). In one case series of 3730 ESIs comparing air vs saline-filled LOR syringes, there was no difference in the incidence of dural puncture (48 vs 51) but a much higher incidence of PDPH with air-filled LOR (32 vs 5) (5). This supports the use of a saline LOR technique during ESI, which should prevent most cases of post-ESI pneumocephalus and decrease incidence of PDPH.

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