CASE REPORTS


Subdural Intracranial Air: An Unusual Cause of Headache after Epidural Steroid Injection

JEFFREY A. KATZ, M.D.,* ROBERT LUKIN, M.D.,† PHILIP O. BRIDENBAUGH, M.D.,‡ LESLIE GUNZENHAUSER, M.D.§

Headache after epidural injection of local anesthetic is most often the result of an unintentional dural puncture. The characteristics of this form of headache are typically the same as those of a headache after spinal anesthesia. Another proposed cause of postepidural or postspinal headache is the introduction of air into the subarachnoid space. This form of headache differs from post-dural puncture headache (PDPH) in that it lacks a postural component.¹

The reported case is a radiographically proven post-epidural headache resulting from injection of air into the subdural space.

CASE REPORT

A 77-yr-old woman with a prior medical history significant for hypertension, Paget’s disease, peptic ulcer disease, and lumbar spinal stenosis presented to the Pain Control Center at the University of Cincinnati Medical Center for epidural steroid injections. She had received two lumbar epidural steroid injections 2 yr prior and had experienced prolonged relief of her low back and bilateral leg pain.

It was decided that the patient should receive a series of up to four lumbar epidural steroid injections. At biweekly intervals, the patient received three epidural injections of 0.125% bupivacaine 8 ml combined first with 12 mg, then 6 mg, and then 3 mg of a betamethasone suspension. Each injection was performed at the L4–L5 interspace by a midline approach with the loss-of-resistance technique using air. Each time, the patient had complete relief of pain; the relief lasted several days, and when the pain returned, it was of a lesser degree than before the block.

At the time of the fourth and final injection, the patient was noting only mild-to-moderate back pain. Epidural needle placement was performed as in the previous three blocks, with a 17-G Tuohy needle and loss of resistance to air. However, on this occasion when loss of resistance was encountered and 3 ml of air was injected rapidly through the Tuohy needle, there was immediate onset of a bifrontal and bitemporal headache. No such complaints had been noted during previous epidurals. After a negative aspiration, 6 ml of air was again injected in 3-ml increments in an effort to confirm needle placement. Five minutes after the final air injection, the headache still was present but had lessened, so 3 ml 2% lidocaine without epinephrine (60 mg) was injected as a test dose.

Approximately 8 min after the test dose, no evidence of neural blockade could be demonstrated, and it was believed that the headache was likely the result of transmitted pressure to the intracranial space by the rapid injection of air into the epidural space.² Eight milliliters of 0.125% bupivacaine with 3 mg betamethasone was injected over 60 s, and the needle was removed and the patient positioned supine with the head elevated 10°. Five min after the steroid injection, the patient noted nausea and dyspnea. Over the next 5 min, respirations became shallow and the dyspnea became so severe that assisted ventilation with O₂ was performed. Hypotension was treated with two doses of 10 mg ephedrine and 1 l crystalloid. Bilateral upper extremity weakness was noted as well as bilateral lower extremity paralysis. A sensory level of at least C6 was noted. The patient remained conscious and coherent throughout the episode. It was believed at the time that either a subdural or subarachnoid injection had been made.

Approximately 37 min after the epidural steroid injection, the patient was comfortable, was breathing spontaneously, and had regained full motor function of the arms but not of the legs. A T2 sensory level was present. Two hours after the steroid injection the patient was discharged with motor and sensory function intact. She still complained of a bi-

* Assistant Professor of Anesthesiology.
† Professor of Radiology.
‡ Professor and Chairman, Department of Anesthesia.
§ Resident in Anesthesiology.

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Address reprint requests to Dr. Katz: Department of Anesthesia, University of Cincinnati Medical Center, 231 Bethesda Avenue, Cincinnati, Ohio 45267-0531.

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temporal headache that was relieved with sitting and exacerbated with lying supine. She was given oral analgesics and was discharged.

Twelve hours after discharge from the Pain Control Center, the patient presented to the emergency room with persistent headache. Headache was still characterized as bifrontal and bitemporal, relieved with sitting and exacerbated with lying down. There were no associated neurologic findings. Plain skull radiographs were obtained on admission to the hospital and initially read as negative by a radiology resident, but a later reading by an experienced neuroradiologist (RL) confirmed air in the intracranial space at the apex of the tentorium. Radiographs showing lateral and Towne's views were obtained 12 h after admission, 24 h after the attempted epidural steroid injection (figs. 1 and 2). These confirmed an inverted V-shaped collection of air at the apex of the tentorium. The well-defined appearance and sharp margins of this collection indicated a subdural location. The radiographic diagnosis was consistent with anatomic observations that demonstrated that the subdural space communicates between the spinal canal and the cranium, unlike the epidural space, which ends at the foramen magnum. The patient reported at this time that the headache was worsened with any movement and was slightly lessened in the sitting position. The patient was reassured and discharged home on oral analgesics. The headache subsequently resolved over the next 5 days.

**DISCUSSION**

One of the most common complications after lumbar epidural needle placement is unintentional dural puncture. The incidence of headache after dural puncture with a 16–18-G epidural needle has been reported to be as high as 70–80%. Features typical of PDPH include its postural nature (aggravated by sitting or standing, relieved by lying down) and its occipital, frontal, and postorbital location. Typically, PDPH has an onset 24–48 h post-puncture (although headache within 1 h has been
reported\(^6\), but all of these reports are based on punctures done with smaller spinal needles rather than with epidural needles.\(^6\) The presentation of headache in the current case was not, however, typical for PDPH and probably was the result of another mechanism.

Another proposed cause of PDPH is the unintentional injection of air into the subarachnoid space.\(^7\) The high frequency and severity of headaches after injection of air into the subarachnoid space has been well documented in studies of pneumoencephalography.\(^1\)\(^,\)\(^6\)\(^-\)\(^10\) This form of headache is characterized by early onset (same day) and recovery over the next 5 days; aggravation with any motion; and lack of relief with lying down.\(^1\)\(^1\)\(^1\) In one case, an elderly patient presented with headache and obtundation 48 h after an epidural anesthetic for a bilateral aortofemoral bypass grafting.\(^7\) Subarachnoid air was found on computed tomography (CT) scan and was confirmed with skull radiographs. However, no description of the anesthetic technique was provided, although the authors claim that there was no method other than the epidural by which air could have entered the skull.\(^7\) Another reported case demonstrated air in the intracranial subarachnoid space by CT scan after an attempted epidural injection.\(^12\) In that case, the local anesthetic injected produced a total spinal anesthetic. The major symptom after recovery from the anesthetic was slowness in return of mental status; there was no mention of headache. The patient’s symptoms resolved the day after the injection, and a repeat CT scan demonstrated no residual air.\(^12\) We were unable to locate any other radiologically confirmed reports demonstrating headache after air injection during epidural needle placement.

With the exception of the two reported cases mentioned above, previous reports of unintentional subarachnoid injections of local anesthetics during attempted epidural anesthesia are primarily limited to injections made through an epidural catheter.\(^13\) The small diameter of these catheters and their tendency to kink would prevent one from being able to aspirate cerebrospinal fluid (CSF) even if one were in the subarachnoid space. This then might result in a spinal anesthetic when an epidural was intended. However, in the current case, all injections were performed through a 17-G Tuohy needle. While it may be possible that a dural “flap” allows injection through the needle but not aspiration of CSF, it would not explain why air would preferentially enter the CSF on injection while the injected lidocaine test dose would flow outside the subarachnoid space. Therefore, it is unlikely that the tip of the Tuohy needle was located intrathecally.

Postpneumoencephalography headache could be the result of either CSF leakage through the dural puncture site or intrathecal air or both. One article discriminated between headache caused by dural puncture and headache caused by intrathecal air by determining the presence of postural dependence.\(^8\) If the headache was relieved entirely by lying supine, it was identified as purely PDPH, but if it was unchanged by position, it was identified as purely the result of intrathecal air. If the headache was partly but not entirely postural in nature (i.e., if headache was reduced but not entirely relieved by lying down), it was then identified as due to both PDPH and intrathecal air.\(^9\) No further testing (such as pain relief with epidural blood patch) was performed to confirm the proposed etiologies of the headaches.\(^9\)

Radiographically confirmed subdural injections of local anesthetics have been described frequently in the literature.\(^14\)\(^-\)\(^17\) It has been characterized previously by a delayed (20–30-min) onset of extensive (as high as C5) sensory blockade after the injection of relatively small volumes of local anesthetic (3.5–10 ml). In the current case, the lack of CSF return through the Tuohy needle, the delayed onset of the sensory and motor blockade by 2% lidocaine, and the extensive spread of anesthesia produced by a limited volume of local anesthetic are consistent with previous reports of subdural anesthesia.

In summary, we propose that subdural air is a possible cause of headache after epidural needle placement. Characteristics of this situation are the lack of CSF on needle placement, an unusually large spread of anesthesia after anesthetic injection, and the lack of postural influence on headache severity.

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Massive Pulmonary Embolism Following Tourniquet Deflation

BRIAN J. McGrath, M.D.,* JUDITH HSIA, M.D.,† BURTON EPSTEIN, M.D.‡

Intraoperative pulmonary embolism is an unusual complication of lower extremity surgery. We report two cases of cardiac arrest due to pulmonary embolism associated with pneumatic tourniquet deflation during knee surgery. The diagnosis in the second case was facilitated by use of transesophageal echocardiography (TEE).

CASE REPORTS

Case 1. A 55-yr-old man underwent an outpatient arthroscopy after injuring the right knee 16 days prior to surgery. Past medical history was significant for ethanol abuse, heavy smoking, and degenerative joint disease. He was taking no medications and denied any allergies. Physical examination was unremarkable. Preoperative laboratory evaluation included normal electrocardiogram, chest x-ray, serum electrolytes, and platelet count. The hematocrit was 55%.

Intraoperative monitoring consisted of continuous electrocardiography, noninvasive blood pressure cuff, esophageal stethoscope, and temperature probe. Anesthesia was induced with intravenous thiopental and fentanyl. Tracheal intubation was facilitated with succinylcholine. Anesthesia was maintained with N₂O in O₂ and isoflurane. Paralysis was maintained with atracurium. A pneumatic tourniquet was inflated around the right thigh, and surgery proceeded without incident. The tourniquet was deflated after 45 min of use. The anesthetic was discontinued, and muscle relaxant was reversed. The patient awakened, and his trachea was extubated.

Approximately 5 min after tourniquet deflation, the patient developed progressively cyanosis, tachycardia, and then bradycardia. His trachea was reintubated; his lungs were ventilated with 100% O₂; and chest compressions were begun. Intravenous epinephrine, atropine, calcium, lidocaine and direct current (DC) cardioversion were given for asystole and subsequent ventricular tachycardia. An episode of atrial fibrillation was treated with cardioversion and intravenous verapamil. A sinus rhythm with a systolic blood pressure of 90 mmHg was obtained with high-dose dopamine and phenylephrine infusions. The initial arterial blood gases during the arrest were: pH 7.21, arterial CO₂ tension (PaCO₂) 96 mmHg, and arterial O₂ tension (PaO₂) 76 mmHg (FiO₂ 1.0). The electrocardiogram showed a new incomplete right bundle branch block with significant ST segment depressions in the lateral leads. After transfer to the intensive care unit, pulmonary angiography revealed multiple large emboli in the right middle and lower lobe arteries. Anticoagulation with heparin was accompanied by steady progressive improvement over the next several days. The patient was discharged from the hospital 17 days later receiving coumadin therapy with no apparent sequelae.

Case 2. A 63-yr-old woman was admitted for elective right total knee replacement. Past medical history included long standing hypertension, one episode of congestive heart failure secondary to uncontrolled hypertension, hypothyroidism, and rheumatoid arthritis, which had severely limited her activity. Preoperative medications were enalapril, nifedipine, prednisone, and thyroid replacement. Past surgical history included an uncomplicated left total knee replacement under general anesthesia 3 months prior to this admission. Venography was performed after this operation as part of a research study and revealed two small clots in the superficial veins of the left calf, for which treatment was not believed to be indicated. The patient denied any allergies. Physical examination was unremarkable. Blood pressure was 150/80 mmHg. Preoperative laboratory evaluation included normal chest x-ray, electrocardiogram, serum electrolytes, prothrombin time, partial tissue thromboplastin time, and platelet count. An echocardiogram showed normal left ventricular function. The hematocrit was 31.5%. Preoperative noninvasive vascular studies were obtained as a result of the previous venogram findings and showed no evidence of deep venous thrombosis.

Intraoperative monitoring consisted of a radial arterial catheter, continuous electrocardiography, capnography, pulse oximetry, temperature probe, and esophageal stethoscope. Anesthesia was induced with intravenous etomidate and fentanyl followed by tracheal intubation facilitated with succinylcholine. Anesthesia was maintained with N₂O in O₂ and isoflurane. Paralysis was maintained with vecuronium. After inflation of a pneumatic tourniquet around the right thigh, surgery proceeded without incident.

Upon completion of the procedure, the tourniquet was deflated, after 99 min of use. Anesthesia was discontinued, and neuromuscular blockade was reversed. The patient regained consciousness with a blood pressure of 170/80 mmHg; pulse 92 beats per min, oxyhemoglobin saturation 100%, and end-tidal CO₂ tension (PETCO₂) 33 mmHg.