

## Persistent Cortical Blindness after a Thoracic Epidural Test Dose of Bupivacaine

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**T**HORACIC epidural anesthesia is considered as an essential component of the perioperative care for patients undergoing lung resection.<sup>1-3</sup> Although neurologic adverse events have been associated with this technique, permanent injury is rare.<sup>4</sup> These events primarily involve the peripheral nervous system, for example, nerve root injury.<sup>4</sup> We present a case of persistent cortical blindness after a test dose of bupivacaine was administered into an uneventfully placed thoracic epidural catheter.

### CASE REPORTS

A 53-yr-old woman, American Society of Anesthesiologists physical status II, height 172 cm, weight 76 kg, was scheduled for resection of a tumor in the lower lobe of the left lung. Based on previous investigations, this tumor was suspected to be a stage IIB large cell bronchial carcinoma. Her medical history included resection of a T<sub>1</sub>N<sub>0</sub> carcinoma of the left breast and resection of a melanoma of the left forearm. In addition to temazepam, the patient did not take any medication. There were no clinical signs of cerebral metastasis, while mediastinal metastasis was ruled out by computed and positron emission tomography. Blood pressure during anesthetic preassessment was 175/75 mmHg. On the day before surgery, the patient received a thoracic epidural catheter by an experienced cardiothoracic anesthesiologist.

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In compliance with recent recommendations,<sup>5</sup> the epidural catheter was inserted at the T2-3 intervertebral space. After inserting a large bore intravenous cannula and placing the patient in the sitting position, the epidural space was identified with an 18-gauge Perican Tuohy needle (Braun, Melsungen, Germany), using the midline approach and hanging drop technique. A Perifix epidural catheter was advanced 5 cm beyond the needle tip. After removal of the needle, the catheter was pulled back 2 cm because of paresthesia of the left arm and shoulder. There was no backflow of cerebrospinal fluid into the catheter either spontaneously or after aspiration with a syringe. No epidural medication was given at this time.

The next day, the patient was premedicated on the ward with 7.5 mg of midazolam orally. On arrival in the operating room, she was positioned on the operating table, and electrocardiogram, noninvasive blood pressure, and pulse oxymeter monitoring were attached. Also, a 20-gauge arterial cannula was placed in the left radial artery. The patient was fully awake and communication appeared normal. Three milliliter of bupivacaine (0.5%; DeltaSelect, Dreieich, Germany) was slowly injected by hand into the epidural catheter by the same anesthesiologist who had inserted the catheter. The solution had been drawn directly from the vial by the anesthesiologist just before injection, and no other drugs had been added. The vial had been stored at room temperature (approximately 70°F). Within 1 min after injection, the patient suddenly lost consciousness and could not be awoken. Respiration appeared adequate. Blood pressure increased with a peak systolic blood pressure of 210 mmHg. There was no bradycardia. Ten minutes after injection, the patient regained consciousness. She was clearly disoriented and complained that she saw "nothing." There were no signs of motor weakness. The surgery was cancelled, and the patient was transported to the intensive care unit for further observation.

In the intensive care unit, vital signs remained stable, and the patient was examined by a neurologist 40 min after the start of the event. The patient was awake but still could not see. Otherwise, cranial and peripheral neurologic function was normal, and there were no cardiac or carotid murmurs. Examination by an ophthalmologist revealed no intraocular or extraocular abnormalities that might explain the sudden visual loss. Because the patient reported to literally see "nothing," not even blackness, the patient was clinically diagnosed with cortical blindness. This was confirmed using magnetic resonance imaging (MRI) of the brain, showing increased signal intensity with signs of vasogenic cerebral edema in the occipital and thalamic areas, and caudal parts of the temporal areas on both sides, and in the left cerebellum (figs. 1A and B). This was suspect of ischemia and infarction in the distribution area of the posterior cerebral artery. However, magnetic resonance angiography showed no abnormalities in the vertebral and internal carotid arteries or in the circle of Willis and its branches (figs. 1C and D). There were no signs of increased intracranial pressure. An MRI and high resolution computed tomography (spatial resolution <0.35 mm<sup>3</sup>) of

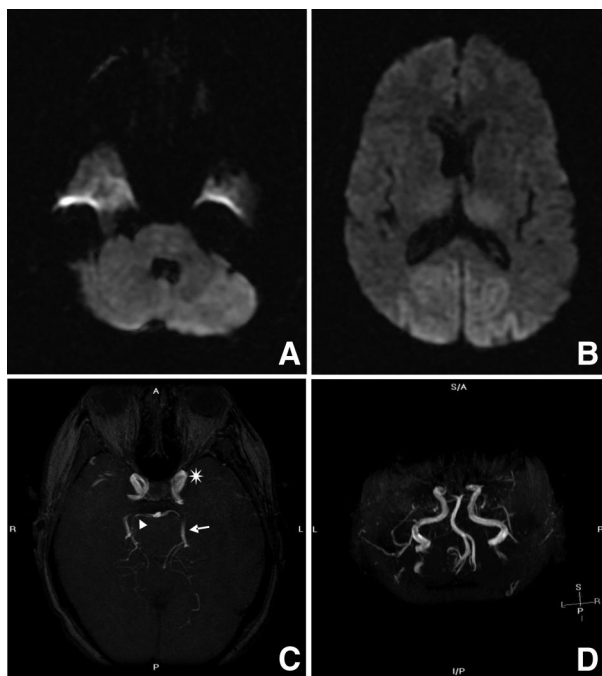


Fig. 1. Axial diffusion-weighted magnetic resonance imaging performed within 2 h of onset of symptoms demonstrates high signal intensity in (A) the left cerebellum and (B) both occipital lobes and posterior part of the thalami, indicating reduced diffusion in these areas. This finding is consistent with early ischemic changes in the vascular territory of the posterior circulation. 3D time-of-flight magnetic resonance angiography demonstrates normal flow in the posterior circulation: (C) axial slab maximum intensity projection showing patent posterior cerebral arteries (arrow), superior cerebellar arteries (arrow head), and normal internal carotid arteries (asterisk); (D) posterior maximum intensity projection view showing patent vertebrobasilar system.

the thoracic spine revealed that the epidural catheter was inserted at the T2–3 intervertebral space, while the catheter tip was positioned in the dorsal epidural space at the level of the T1 vertebral body (fig. 2). The tip was not located in a blood vessel, and the catheter followed a straight path from insertion site to final tip position (fig. 2). No abnormalities were identified in the spinal cord. To enhance the visualization of the catheter on T1-weighted MRI, 3 ml of gadolinium MRI contrast medium was injected through the epidural catheter. Five min after this injection, the patient again lost consciousness, with hypertensive peaks up to 240 mmHg systolic and generalized convulsions with extensor posturing of both upper and lower extremities. She was intubated and mechanically ventilated. Continuous infusions of nitroglycerin and nicardipine were started to keep systolic blood pressure around 150 mmHg. The patient was extubated 2 days later, after which she was fully conscious. Intermittent neurologic evaluations did not reveal motor or reflex abnormalities. Analysis of the vial showed a bupivacaine solution of 5 mg/ml, pH 6.0, and 300 mOsmol/kg, consistent with the information supplied by the manufacturer. Allergy to bupivacaine was ruled out later by skin testing.

The patient was discharged from the intensive care unit after 3 days. The cortical blindness remained unchanged, and she suffered from cognitive disorders such as anterograde amnesia and disorientation in time and place. Five



Fig. 2. Multislice spiral computed tomography (64 × 0.75 mm slices) with sagittal curved multiplanar reconstruction imaging demonstrates proper midline placement of the epidural catheter tip in the posterior epidural space. The entrance site of the epidural catheter is at the T2–3 intervertebral space. The catheter tip (asterisk) is identified at the level of first thoracic vertebral body.

weeks after the event, she underwent a lobectomy of the left lower lobe, which contained a large metastasis of a melanoma. No tumor cells were found in the peribronchial and mediastinal lymph nodes. The patient was discharged home 10 days after surgery and has been enrolled in a rehabilitation program. One year after the incident, there has been no significant improvement in her vision and cognitive disorders.

## Discussion

We present a case of sudden loss of consciousness after a thoracic epidural test dose of bupivacaine, accompanied by hypertension and visual loss. Although the first two symptoms resolved quickly, visual loss and cognitive disorders have unfortunately persisted.

Ischemic events in the brain and/or spinal cord after epidural injections have been reported before.<sup>6</sup> Similar to our case, ischemic brain injury has occurred in the absence of cerebrovascular abnormalities.<sup>7</sup> However, these reports exclusively concern transforaminal injections of particulate steroids, during which the epidural needle tip is close to the vertebral artery, and a particulate injectate is used. This may explain why several cases of cerebral embolic events have been reported with this particular technique. In our case, the rapid loss of consciousness and convulsions may suggest an intraarterial injection. However, intraarterial injections are not common in mid-thoracic epidural anesthesia, and they are generally not accompanied by hypertension and rapid return of consciousness. Furthermore, on computed tomography and MRI examinations, the epidural catheter tip was not located in a blood vessel. These examinations also ruled

out subdural or subarachnoid position of the catheter tip. Given the hypertension, subarachnoid injection or an allergic reaction seems unlikely. Also, allergy testing to bupivacaine was negative.

Epidural injection has been shown to increase intracranial pressure both in humans<sup>8</sup> and in a porcine model,<sup>9</sup> resulting in markedly reduced cerebral blood flow.<sup>9</sup> However, these effects are particularly pronounced when intracranial pressure is already increased before injection, whereas our patient did not show any signs of increased intracranial pressure preoperatively. Furthermore, visual loss has been reported after rapid epidural fluid infusions to induce epidural anesthesia or to facilitate epiduroscopy.<sup>10</sup> However, the volumes of epidurally injected solutions varied from 20 to 120 ml, compared with 3 ml in our report, and the common finding in these cases was retinal hemorrhage.

We are unaware of reports describing a similar sequence of events. While we can only speculate on an explanation, a possible mechanism is the following. In the thoracic and cervical epidural regions, the anterior and posterior epidural venous plexuses become more developed compared with the lumbar epidural region.<sup>11</sup> In the presence of local epidural fat pads consisting of fibrous strands, a small volume of injected fluid may have compressed venous and/or arterial structures supplying the spinal cord. A disturbance of the vascular supply to the cord may have caused autonomic dysreflexia, resulting in massive secretion of epinephrine and hypertensive crises, similar to the situation in patients with high spinal cord transections.<sup>12</sup> These hypertensive crises may have led to the development of hypertensive encephalopathy, which typically affects the posterior area of the brain.<sup>13,14</sup> The fact that a second episode of loss of consciousness and hypertension occurred after an epidural injection with MRI contrast medium supports the assumption that volume expansion of the epidural space is a more likely culprit than action by the local anesthetic. Alternatively, autonomic dysreflexia may coexist with catecholamine secreting tumors.<sup>15</sup> However, although bronchial carcinomas have been associated with neuroendocrine activity,<sup>16</sup> melanomas are not known to produce catecholamines.

Although the use of thoracic epidural anesthesia has increased tremendously over the last decade and a half, persistent neurologic injury related to its use remains rare. Regardless of the mechanism, by presenting this case report of cortical blindness after a thoracic epidural test dose, we hope to increase the awareness among anesthesiologists that serious adverse events may occur, even when the procedure has been carried out correctly. However, because the mechanism

remains unclear, it is difficult to provide recommendations to prevent this complication.

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