CLINICAL VIGNETTE

Unexplained Transient Coma during Emergent Caesarean Section on Patient with Preeclampsia

Olivia Kaminsky, MD and Peter Huynh, MD

Harbor-UCLA Department of Anesthesiology

Clinical Case

A 26-year-old female, Gravida-3, Para-1 presented for labor at 37 weeks and 6 days. She had a history of gestational thrombocytopenia (platelets 108) and preeclampsia without severe features. The patient requested an epidural to minimize pain associated with contractions during labor which was successfully placed on the second attempt.

When placing an epidural, the clinician first must identify an appropriate space in the lumbar spine to insert the epidural needle, known as a Tuohy needle. This space is typically around the third and fourth lumbar vertebrae to remain below the terminal end of the spinal cord and lower the risk of spinal cord injury if the needle were accidentally advanced beyond the epidural space into the intrathecal space. After anesthetizing the skin, the needle is advanced through five distinct tissue layers: skin, subcutaneous fat, supraspinous ligament, interspinous ligament, and ligamentum flavum. After this final layer, the Tuohy needle enters the epidural space, defined as a potential space between the ligamentum flavum and the dura mater. Anesthesiologists utilize the "loss of resistance" technique while opening this potential space to accommodate the epidural catheter. This technique refers to the fact that the interspinous ligament and ligamentum flavum are very dense tissue, and as the epidural needle tip advances just past the ligamentum flavum, there is an abrupt decrease in resistance, which alerts the anesthesiologist that the needle has entered the epidural space.[1] Once the epidural space is accessed, the epidural catheter is threaded through the Tuohy needle, the needle is removed, and the catheter is secured.

The first attempt at epidural placement for this patient was grossly unremarkable with loss of resistance, however there was difficulty threading the epidural catheter into the epidural space.

By standard of care, a test dose was then administered to further confirm proper placement of the catheter. First, the anesthesiologist aspirates from the epidural catheter to confirm that there is no return of blood or cerebral spinal fluid to ensure the catheter is not in an intravascular or intrathecal space. Next, the standard test dose of 3 ml of 1.5% lidocaine with epinephrine 1:200,000 is administered through the catheter while closely monitoring for hemodynamic changes or any symptoms that may suggest inaccurate positioning of the catheter. A negative test dose suggests that it should be safe to use the epidural catheter. Further testing is performed by applying an ice pack over the patient's skin. A reduced temperature sensation to the ice over the abdominal area compared to other areas such as the face and shoulders implies a properly working epidural. Once the test dose for this patient was confirmed negative, a standard infusion of 0.0625% bupivacaine with 2 mcg/cc fentanyl was started. The infusion ran for approximately twenty-five minutes, at which time an emergent caesarean section was started for intermittent fetal decelerations which did not respond to typical interventions by the obstetrics team, and ultimately progressed into brief loss of fetal cardiac activity.

Upon arrival of the anesthesia team, the patient had already been positioned on the operating room table with standard monitors in place. After confirming negative aspiration of the epidural catheter once more, the patient was given a total of 20 mL (600mg) of chloroprocaine, a typical first-line local anesthetic administered in emergent c-sections as it has the fastest onset time. Two minutes later, the baby was delivered with Apgar scores of 4, 9, and 9 at 1, 5, and 10 minutes. The patient had been verbalizing spontaneously and communicating appropriately with the anesthesia team throughout the entire delivery. Fifteen minutes later, she started requiring respiratory assistance by mask ventilation, and within the next thirty minutes, slowly became more somnolent, unresponsive, and finally apneic. She was subsequently intubated for airway protection using only low dose propofol for induction. No cough or gag reflex was appreciated during intubation.

Throughout this progression, the patient's systolic blood pressure decreased from a baseline in the 140s to 90s mmHg, stabilizing around 90mmHg with minimal intermittent phenylephrine support. Her heart rate reamined at 80-90 bpm. An arterial line was placed for close hemodynamic monitoring. Preliminary labs were drawn, including point-of-care testing of hemoglobin and hematocrit, and stat blood pH, base excess, oxygenation, electrolytes, and glucose. Results were all grossly within normal limits. Approximately thirty minutes after intubation, the patient began initiating breaths, appropriately responding to questions, and demonstrating return of motor

function. The decision was made to extubate, and she was successfully extubated to room air without any issues.

After extubation, additional laboratory and imaging studies were obtained. Given that the patient was on a magnesium drip during labor, and supratherapeutic magnesium levels are associated with somnolence, confusion, and lethargy, a magnesium level was checked, resulting within therapeutic range at 3.6 mEq/L. Brain imaging, however, revealed "mild diffuse pneumocephalus" with "foci of air in the left quadrigeminal plate cistern and per-pontine cistern, the suprasellar cistern, and the frontal horns of lateral ventricles".

The patient was evaluated by both the anesthesia and obstetrics teams after returning from the CT scanner. She was alert and oriented to name, place, time, and situation, with intact motor and sensory exams throughout all four extremities. She denied any other symptoms and reported her last memory to precede the operating room. In the postoperative period, the patient remained stable and safe for discharge shortly after delivery. She was fully functioning at her baseline mental and physical status and maintained no recollection of her immediate perioperative course.

Discussion

The use of neuraxial anesthesia in obstetrics is very popular in the United States. Approximately seventy percent of pregnant women receive epidurals or other spinal anesthesia, an increase of about ten percent in the last ten years.[2] Despite the high prevalence of neuraxial anesthesia administered to patients, serious neurological complications from epidural anesthesia still occur, but are fortunately very rare. A systemic review found an incidence of transient neurologic injury in 0.02% of women, and permanent neurologic injury in only ~1 in 4,000,000 women.[3]

The specific etiology of this patient's apnea and transient loss of consciousness remain inconclusive. Considerations include a high spinal secondary to accidental dural puncture during epidural placement of the epidural, a subdural injection, and a case-reported phenomenon of "sensory deafferentation dependent sedation".

A high spinal is a rare, life-threatening complication which may occur if an epidural catheter is accidentally placed, or spontaneously migrates into, the intrathecal space. Epidural dosing of medications is typically much higher than spinal dosing. Thus, if the patient receives an epidurally dosed medication into the intrathecal space, the patient is immediately exposed to supratherapeutic doses. The medication may also spread further cephalad given the larger volume used in epidural injections, leading to anesthetization of critical areas of the spinal cord including those that control the cardiopulmonary systems. This may result in hypotension, bradycardia, respiratory depression, and loss of consciousness.[3] It is crucial to recognize a high spinal early and treat appropriately with hemodynamic and airway support. If, during epidural placement, the Touhey needle accidentally created a small perforation in the dura, the epidural infusion medication—and later—chloroprocaine may have slowly leaked into the intrathecal space. With time, enough medication may have accumulated in the intrathecal space to resemble a high spinal. It is reassuring that the epidural test dose was negative, which should provide confirmation that the epidural catheter was in the appropriate space. Furthermore, the patient remained on an epidural infusion for approximately twenty-five minutes without any signs indicating a high spinal and only became unresponsive approximately forty-five minutes after chloroprocaine was administered. This, along with gross hemodynamic stability throughout the case, would represent an unusual presentation of a high spinal.

Another consideration for this clinical change is a subdural injection, which remains a less well-recognized complication of neuraxial anesthesia. A subdural injection typically presents as a strong sensory block, with sparing of sympathetic and motor functions due to sparing of ventral nerve roots. The block often has a slow onset (15 to 20 minutes) with incidences of hypotension and lasts up to two hours followed by a full recovery. It can also present with progressive respiratory demise rather than a sudden apnea.[4] This patient demonstrated both a motor and sensory block, rather than an isolated sensory block, and did not show significant hypotension. However, the subdural injection remains an etiologic consideration because several unusual presentations of subdural blockade have been described in the literature, including differences in block onset time as well as differences in motor and sensory blockade levels.[5]

A phenomenon called "sensory deafferentation dependent sedation" has also described in several case reports, citing an "impaired alertness to be due to the loss of spinal cord sensory input to the brainstem".[6] These case reports include symptoms similar to those encountered by our patient, including complete loss of consciousness, stable blood pressure, complete recovery within 45 to120 minutes, and no sequelae. Such, it is reasonable to consider that a similar course of events occurred in our patient. Lastly, the CT findings of mild pneumocephalus may also point to suggest a brain etiology contributing to the symptoms observed.

This case highlights the importance of remaining vigilant at all points of a regional anesthetic administration. The anesthesiologist must constantly remain on high alert during any emergent or non-emergent delivery requiring anesthesia for any changes in patient's status throughout the entirety of the procedure, including throughout the entire perioperative window.

REFERENCES

1. **Dhansura, Tasneem, et al.** "Identification of the epidural space-loss of resistance to saline: An inexpensive modification." *Indian journal of anaesthesia* 59.10 (2015): 677.
2. **Butwick AJ, Wong CA, Guo N**. Maternal Body Mass Index and Use of Labor Neuraxial Analgesia: A

Population-based Retrospective Cohort Study. *Anesthesiology*. 2018 Sep;129(3):448-458. doi: 10.1097/ALN.0000000000002322. PMID: 29939847; PMCID: PMC6118130.

3. **Eason, Martin, and Martin E. Olsen**. "High spinal in an obstetric patient: A simulated emergency." *Simulation in Healthcare* 4.3 (2009): 179-183.
4. **Ruppen W, Derry S, McQuay H, Moore RA**. Incidence of epidural hematoma, infection, and neurologic injury in obstetric patients with epidural analgesia/anesthesia. *Anesthesiology*. 2006 Aug;105(2):394-9. doi: 10.1097/00000542-200608000-00023. PMID: 16871074.
5. **Agarwal D, Mohta M, Tyagi A, Sethi AK**. Subdural block and the anaesthetist. *Anaesth Intensive Care*. 2010 Jan;38(1):20-6. doi: 10.1177/0310057X1003800105. PMID: 20191772.
6. **Kim YD, Lee JH, Cheong YK**. Pneumocephalus in a patient with no cerebrospinal fluid leakage after lumbar epidural block - a case report -. *Korean J Pain*. 2012 Oct;25(4):262-6. doi: 10.3344/kjp.2012.25.4.262. Epub 2012 Oct 4. PMID: 23091688; PMCID: PMC3468804.