

LOSS OF CONSCIOUSNESS IN A PARTURIENT FOLLOWING THE ADMINISTRATION OF A TEST DOSE FOR EPIDURAL ANESTHESIA

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Introduction

Neuraxial anesthesia is the technique of choice in pregnant patients, unless otherwise indicated. Epidural anesthesia is widely used in the settings of normal vaginal delivery. Inadvertent dural puncture is not an uncommon complication of epidural catheter insertion with an incidence of 0.04-6%¹. Other complications include intrathecal (0.53%)² or subdural (0.8%)³ epidural catheter misplacement leading to total spinal blockade and subdural blockade, respectively. We report a case in which a parturient became unconscious 20 minutes following the administration of an epidural test dose, composed of 45 mg Lidocaine and 15 mcg Adrenaline.

Case Report

A 30-year-old, 80 kg primigravid, and full term parturient with a smooth course of pregnancy, presented for scheduled induction of labor. Her past medical history was unremarkable except for asthma controlled with bronchodilators as needed. Epidural anesthesia to control labor pain was requested.

The patient was placed in a seated position. Using the midline approach, an 18-gauge Tuohy needle was placed in the interspace at L3-L4 level. The ligamentum flavum was felt at 4.5 cm from the skin. The needle was further introduced carefully and the epidural space was identified with the loss of resistance to air technique. However, just after reaching the epidural space, the patient moved her back unexpectedly. Aspiration from the Tuohy needle using a 3 cc syringe was negative, and 2 ml of 2% Lidocaine were injected via the Tuohy needle to numb the epidural space. A catheter was inserted and easily threaded through the Tuohy needle, which was then withdrawn over the catheter. Despite the negative aspiration from the Tuohy needle, aspiration from the catheter was positive suggesting its possible intrathecal placement.

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A 3 ml test dose, composed of 45 mg of isobaric Lidocaine and 15 mcg of Adrenaline was administered through the catheter. Shortly afterwards, the patient lost sensory and motor powers in both her lower extremities, which confirmed the intrathecal placement of the catheter.

We elected to remove the catheter and resite it to a different interspace after the patient recovered from the effect of the test dose. However, as we were waiting she started to complain of difficulty in breathing. The patient had transient episodes of apnea and gradually felt drowsy but was responsive to verbal stimulation. Her vital signs were within the normal ranges, except for a mild desaturation to around 94% on room air that responded immediately to oxygen administration via face mask. Twenty minutes later, the patient developed complete loss of consciousness and stopped responding to both verbal and deep pain stimuli. Her vitals remained stable, with a blood pressure of 120/70 and a heart rate of 90 bpm. On physical exam, her pupils were about 4 mm in width and reactive to light.

Decision was to proceed with an emergency cesarean section under general anesthesia. In the operating room, the patient was breathing spontaneously with an evident regular end tidal CO₂ waveform. She was preoxygenated and given 320 mg of Thiopental and 100 mg of Succinylcholine prior to intubation. The course of the surgery was uneventful.

At the end of the surgery, all anesthetics were discontinued and 10 minutes later the patient started to gradually regain her consciousness. After meeting the criteria, the trachea was extubated. CT scan of the head without contrast was done. Results showed pneumocephaly in the cavernous sinuses, suprasellar cistern and the CSF space of the upper cervical spine, most likely sequelae of epidural intervention. The CT scan was otherwise unremarkable.

The patient was taken to the recovery room for post-operative monitoring. She was unable to recall the events that happened after the epidural test dose, except for the difficulty in breathing. After 3 hours of monitoring, the patient was transferred to the regular floor. She complained of incisional pain, which was controlled by Acetaminophen and NSAIDs. The patient and the baby were stable throughout the course of their stay, and 4 days later they were both discharged. Of

note, the patient never complained of post epidural puncture headache during her stay at the hospital nor after discharge.

Discussion

The use of epidural test dose has been advocated in routine practice to check for a proper placement of epidural catheters⁴. Even in the presence of negative aspiration, neither the intravascular⁵ nor the intrathecal placement can be excluded⁶. There has been a lot of controversy regarding the composition of the test dose⁷ since complications such as high⁸, prolonged⁹ and even total spinal anesthesia^{8,9,10} have been reported. In our clinical practice, we shifted towards using 45 mg of Lidocaine instead of 60 mg since it is in our belief that such a dose is safer and as sensitive in detecting possible intrathecal catheter placement. A recent study by Pratt et al¹¹ suggested an even further decrease of Lidocaine in the composition of a test dose. Results of the study showed that 30 mg of Lidocaine was just as effective as the 45 mg of Lidocaine, with a very high negative predictive value for both dosages. Our patient developed loss of consciousness after administration of 45 mg Lidocaine in the subarachnoid space. The injection of local anesthetic, to numb the epidural space in order to avoid pain from catheter insertion, confounded the result of the fluid aspirated by the catheter. Therefore, the administration of the test dose, which was believed to be safe was necessary to detect the actual site of our catheter.

In our case, the loss of consciousness the patient suffered from can be explained by different mechanisms. Palkar et al¹⁰ reported a 36 year old parturient who had a total spinal anesthesia following administration of an epidural test dose, similar in composition to what we administered. However, they reported that the patient developed hypotension and required the use of Ephedrine. Some researchers believe that the hypoperfusion to the brain and the brain stem caused by severe drop in blood pressure is a potential mechanism for total spinal anesthesia. Although this seems to be a convincing theory, many cases of total spinal anesthesia were reported in the absence of hypotension. De Saram et al¹² reported 3 cases of total spinal anesthesia with loss of

consciousness and without cardiovascular instability. Similarly, Siddik-Sayyid et al¹³ reported a case of total spinal anesthesia despite a relatively stable blood pressure and heart rate after spinal anesthesia following ongoing epidural analgesia. Even though none of the above mentioned physicians described major change in vital signs, they all administered vasopressors preemptively as per standard technique in their clinical practice. De Saram gave all his patients 33 mg of intramuscular Ephedrine and Siddik-Sayyid started a Neosynepirne infusion just after the spinal anesthesia was administered. The fact that the above mentioned patients had stable blood pressures and heart rates suggests that the vital structures in their brains and brain stems were well perfused; despite that, they all developed total spinal anesthesia. In other words, hypoperfusion of the brain and the brainstem might not be the only mechanism behind total spinal anesthesia. In fact, it might be the direct effect of local anesthetics, which in case of progressive cephalad spread, can reach the brain and the brain stem and interfere directly with their neuronal function¹³.

Our patient had stable vital signs and did not require any vasopressor administration, yet she developed loss of consciousness. We believe this can be attributable to the direct effect of local anesthetic, which has spread in routes other than the subarachnoid space. The vascular route can easily be excluded since the patient did not have an increased heart rate or blood pressure or signs of local anesthesia toxicity. Moreover, the dose used was much less than the toxic dose of local anesthetic. The subdural route, on the other hand, is a potential mechanism that can explain the extensive spread of a relatively low amount of local anesthetic. Lubenow et al described 2 major and 3 minor criteria for the diagnosis of a subdural blockade. Major criteria included: 1) negative aspiration test and 2) extensive sensory block. Minor criteria included: 1) delayed onset by 10 minutes or more of a sensory or motor nerve block, 2) variable motor block and 3) sympatholysis out of proportion to the administered dose of local

anesthesia. A subdural injection is considered to have occurred if both of the major criteria and at least one of the minor criteria are present³. In our case, both major and minor criteria were met. Therefore, we believe the Tuohy needle did not actually puncture but dissected the dura matter, and the local anesthetic, injected to numb the epidural space, went subdurally. In addition, the dissection created a weakness point in the dura, which led to its puncture while threading the catheter and the subsequent placement of the test dose intrathecally.

As for the CT findings, our patient was found to have pneumocephaly in the cavernous sinuses, suprasellar cistern and the CSF space of the upper cervical spine. Pneumocephalus is a rare yet documented complication of epidural intervention. Even a small volume of air introduced intrathecally, can be enough to cause significant pneumocephalus¹⁴. Cases of severe acute headache due to iatrogenic pneumocephalus were reported following epidural anesthesia, especially when complicated by inadvertent dural puncture¹⁵, and even after spinal anesthesia¹⁴. Our patient did not complain of headache nor did she develop any other neurological symptom during her hospital stay and after discharge. Therefore, the pneumocephaly noted by the CT scan was mostly benign and did not contribute to the patient's loss of consciousness.

In conclusion, we report loss of consciousness in a parturient after receiving an epidural test dose for normal vaginal delivery. Whether our catheter was placed subdurally or intrathecally, we believe that the subdural distribution of the local anesthetic injected is responsible for the loss of consciousness. We recommend against using high concentration of local anesthetic to anesthetize the epidural space before threading the catheter since accidental subdural or intrathecal injection of such doses may lead to undesired consequences for both, the mother and the baby.

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