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Atypical Rapid Onset of Lumbar Epidural Anesthesia after Confirming Negative Results of a Test Dose

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Introduction

Test dose injection in epidural anesthesia is typically performed to rule out misplacement of the epidural catheter or needle.

Here, we report a case of rapid onset of lumbar epidural anesthesia immediately after injecting a main dose, while confirming no aspiration of cerebrospinal fluid or blood, and no motor or sensory blockade following injection of a test dose.

Case report

The patient was a 75-year-old woman (height, 145 cm; weight, 48 kg) undergoing surgical repair of a left inguinal hernia with lumbar epidural anesthesia. She did not complain of any neurological symptoms affecting the lower limbs and had no prior history of lumbar surgery, external injuries, or neuraxial blockade. Findings of a preoperative physical examination were normal.

Lumbar puncture was performed at the L1–L2 interspace by using a 1-cm-lateral paramedian approach with an 18-gauge Tuohy needle in the right lateral decubitus position. Loss of resistance to saline injection was used to identify the epidural space, which was at a depth of 5.5 cm. A single-orifice, open-ended, epidural catheter was inserted 3 cm beyond the needle tip. The above procedure was successfully performed once without any complication or complaint of severe pain or numbness. After confirmation of no aspiration of blood or cerebrospinal fluid, 3 ml of 2% lidocaine was injected as a test dose. After 3 min, no evidence of sensory or motor blockade of the lower limbs was observed. Consequently, 7 ml of 2% lidocaine was injected as a main dose through the epidural catheter over 30 s. Immediately after the injection, Bromage scale 1 motor blockade and L1 sensory blockade were observed. Twenty seconds later, motor blockade progressed to Bromage scale 3 and sensory blockade also rose to the level of Th 2. The patient complained of dysarthria, dyspnea, and numbness of both upper limbs. Subsequently, her oxygen saturation (SpO_2) level decreased from 98% to 88%. Inhalation of 100% oxygen at 10 l/min was administered and SpO_2 levels recovered to 98%. However, the patient became unable to vocalize and subsequently lost consciousness. As the patient's respiratory condition remained stable, her ability to breathe naturally was observed. Further attempts were made to aspirate the epidural catheter, but it was not able to draw fluid or blood. Subsequently, her blood pressure decreased from 125/77 to 68/35 mmHg and her heart rate decreased from 72 to 45 beats/min. Consequently, ephedrine (8 mg) and atropine (0.5 mg) were administered, which resulted in recovery of blood pressure and heart rate to 101/56 mmHg and 65 beats/min, respectively. Following this, her hemodynamics remained stable and the surgery was performed as scheduled.

The surgical procedure was uneventful with a duration of 70 min. We frequently tried to aspirate the catheter intraoperatively but were unable to draw fluid.

Postoperatively, the patient awoke naturally and her vital signs remained stable. Since she was able to breathe without complaining of dyspnea, she was transferred to the surgical ward. At that time, the epidural catheter was removed and no leakage of fluid from the puncture site was observed.

At 160 min after the epidural injection, the patient completely recovered from sensory and motor blockade. Magnetic resonance imaging of the spine showed only slight spinal stenosis at the L3–L4 level. The postoperative course was uncomplicated and the patient was discharged on foot on postoperative day 4.

Discussion

It has been customary in our institution to inject 3 ml of 2% lidocaine (60 mg of lidocaine) as a test dose after insertion of a catheter into the epidural space to rule out misplacement. Then, 3 min afterward, the patient's condition is evaluated according to Bromage scale as a diagnostic test, because the onset of neural blockade with 3 ml of 2% lidocaine via intrathecal injection is within approximately 3 min.[1] Thus far, we have never encountered a case of rapid onset of epidural anesthesia, as with this case.

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Colonna-Romano et al.[2] reported the diagnostic accuracy of an intrathecal test dose of 45 mg of lidocaine with 0.015 mg of epinephrine in epidural anesthesia as follows: sensitivity and specificity of 100% [95% confidence interval (CI) = 78%–100%] and 93% (95% CI = 66%–100%), respectively, and negative and positive predictive values of 100% and 25% (assuming a 2% prevalence of an intentional spinal), respectively, by the ability to raise the legs as an indicator. In general, a diagnostic test with a sensitivity of more than 80% is considered effective[3] and indicates that the validity of a test dose in epidural anesthesia has been sufficiently confirmed.

Several studies[4,5] have reported that the interval from injection of a test dose to evaluate the signs of neural blockade is approximately within 3 min, as with the present case. However, Colonna-Romano et al.[2] concluded that 4 min is needed to recognize the signs of misplacement of an epidural catheter. Although this patient was of small stature (height, 145 cm), there was a possibility that neural blockade was evaluated too quickly. Furthermore, the test dose injected into the epidural space may also have been much more than needed. For these reasons, the rapid onset of neural blockade may have been an effect of the test dose or synergism with main dose. If the effect of the test dose had been evaluated 4–5 min after injection, it may have been possible to anticipate the possibility of rapid neural blockade onset. Hence, we have revised the interval time from injection of a test dose to evaluate the signs of neural blockade from 3 min to 5 min, and amended the test dose of lidocaine from 60 mg to 45 mg. Furthermore, it is also important to administer an anesthetic in multiple smaller doses over a longer period of time and frequently confirm the levels of blockade in epidural anesthesia.

In the present case, the epidural catheter was removed after the operation. And, we could not confirm its misplacement on a radiographic image. Therefore, based on the clinical findings, subarachnoid injection or subdural injection was considered to be the cause of the atypical rapid onset of neural blockade. It was not possible to aspirate cerebrospinal fluid despite having multiple attempts in this case. However, even though cerebrospinal fluid could not be aspirated, there is no guarantee that no local anesthetic was delivered into the subarachnoid space.[6] For a subdural block, the symptoms will vary according to the catheter tip location.[3] Therefore, the use of a conventional test dose may fail to reveal subdural catheter misplacement.

In this case, we used a single-orifice, open-ended, epidural catheter. A multi-orifice, close-ended, catheter, which is another epidural catheter type, is easier and less painful to place, and makes sensory blockade to be more exact. However, it has the risk of hazardous dual compartmental misplacement.[7] Occasionally, a distal opening of catheter may lie within the subarachnoid or subdural space, while a proximal orifice simultaneously retains normal access to the epidural space. Using such improperly placed catheter can cause not only atypical blockade but also secondary dural injury. Thinking of the malpositioning of epidural catheter, it may be better to use a single-orifice type catheter.

The incidences of subarachnoid and subdural injection are reportedly 0.5%–2.0%[2,8] and 0.8%–1.0%,[9] respectively. However, even when epidural blockade is performed by trained anesthesiologists, the needle can partly pierce the dura mater at an incidence of up to 7%.[3] These complications may occur more frequently than generally considered.

In conclusion, atypical rapid onset of epidural anesthesia may occur, even after confirming negative results of the test dose and aspiration test. The findings presented are from a single case report, and thus, further studies are required to verify these findings.

Consent

Written informed consent was obtained from the patient for publication of this case report.

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