Intrauterine fetal bradycardia after accidental administration of the anesthetic agent in the subdural space during epidural labor analgesia
-A case report-

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Subdural injection of epidural anesthesia is rare and is usually undiagnosed during epidural anesthesia causing severely delayed maternal hypotension, hypoxia, and fetal distress. A 38-year-old primiparous woman was administered epidural labor analgesia at 40+6 weeks’ gestation, and developed progressive maternal respiratory depression, bradycardia, and hypotension after accidental subdural administration of the anesthetic agent. Furthermore, fetal distress occurred soon after administration. The patient was managed with oxygen, position changes, fluid resuscitation, and ephedrine. Intrauterine fetal resuscitation was successfully performed with atropine before cesarean section, and a healthy baby was delivered. Although subdural injection is uncommon, this case emphasizes the importance of anesthesiologists monitoring patients for a sufficient period after epidural labor analgesia, and being prepared to perform maternal or fetal resuscitation. (Korean J Anesthesiol 2013; 64: 529-532)

Key Words: Atropine, Epidural analgesia, Fetal bradycardia, Resuscitation, Subdural injection.
Case Report

A 38-year-old G1P0 primigravida was admitted to our hospital at 40th weeks gestation for a normal vaginal delivery. She had no history of medical illness or trauma. The initial fetal heart rate (HR) was 120–150 beats/min and showed good reactivity and variability. When the diameter of the cervical opening was approximately 4 cm, lumbar epidural analgesia was planned, and an intravenous (IV) infusion of lactated Ringer’s solution (1,000 ml) was administered. An 18-gauge Tuohy needle (Portex® Regional Anaesthesia Tray, Smiths Medical, UK) was blindly inserted into the lumbar epidural space at the L3-4 level in the left decubitus position. A Tuohy needle was advanced to 5 cm, at which point the epidural space was identified on the first attempt using the loss of resistance (LOR) technique. After ensuring that there was no cerebrospinal fluid (CSF) or blood leak, we slowly injected a test dose, i.e., 4 ml of 1% lidocaine with 15 μg of epinephrine. An epidural catheter (Portex® Regional Anaesthesia Tray, Smiths Medical, UK) was inserted without any resistance. The catheter tip was threaded 4 cm in the cephalad direction beyond the Tuohy needle, after catheter placement. We confirmed that there was no CSF and blood by aspirating the catheter. Then, we mixed 20 ml of 0.75% ropivacaine and 100 μg of fentanyl with normal saline (total volume, 100 ml), and subsequently injected 12 ml of the mixture. The rest of the mixture was injected into the epidural space with a patient controlled epidural analgesia (PCEA) pump (Pain Management Provider®, Abbott, USA) with a basal flow rate of 5 ml/h plus a 5 ml bolus with a 5 min lockout. At 5 minutes after the initial dose administration, her blood pressure (BP), HR, and respiratory rate (RR) were 120/70 mmHg, 72 beats/min, and 20 breaths/min, respectively. We subsequently assessed the extent of bilateral sensory blockade by observing the loss of cold sensation to alcohol sponges and the loss of sensation to pinpricks. At that time, the sensory level was located at the tenth thoracic dermatome, and the motor function was intact. At 15 minutes after the initial dose administration, the patient complained of a pulling sensation and tightness at the back of her neck. At 20 minutes after the initial dose administration, the patient showed drowsiness and a depressed mental status; therefore, we encouraged her to take deep breaths and stopped the PCEA pump. The total injected volume of the drug mixture was 1.5 ml. At that time, her vital signs were as follows: BP, 90/60 mmHg; HR, 60 beats/min; and RR, 8 breaths/min. The sensory level was at the eighth thoracic dermatome; however, no change in motor function was observed. Because of the reduced RR and depressed mental status, the patient was placed in the Trendelenburg position and was administered 100% O2 via a facial mask at a flow rate of 5 L/min. At 3 min after position change, her vital signs were as follows: BP, 76/49 mmHg; HR, 55 beats/min; RR, 8 breaths/min; and SpO2 95%. The sensory level was the third thoracic dermatome, but her motor functions were not affected. After the patient was injected with 10 mg of ephedrine, an obstetrician examined the fetal status via ultrasonography. The fetus showed severe bradycardia, (HR, 70 beats/min); despite being in the normal range (HR, 100–150 beats/min) before this event. Therefore, we decided to perform an emergency cesarean section, which was planned to occur within 30 min, followed by prompt intrauterine fetal resuscitation. At that time, the patient’s vital signs were as follows: BP, 80/51 mmHg; HR, 52 beats/min; RR, 8 breaths/min; and SpO2 92%. We moved the patient to the left lateral recumbent position, injected 0.5 mg of atropine via the maternal IV line, started rapid fluid replacement, and continuously monitored the fetal HR. At 5 min after intrauterine fetal resuscitation, the fetal HR was 100 beats/min, but the patient still showed a depressed mental status and complained of dyspnea. Artificial respiration was performed immediately by using AMBU bag because the patient’s SpO2 decreased to 82%. Consequently, her SpO2 recovered to 95%, while the fetal HR increased to 120 beats/min and showed good variability. The fetal HR had decreased for approximately 10 minutes. Fifteen minutes after recovery of the SpO2 level, the patient was transferred to the operating room on artificial ventilation. The arterial blood gas analysis performed immediately before the operation revealed the following: pH, 7.086; PaCO2, 66.6 mmHg; PaO2, 218.6 mmHg; HCO3-, 19.6 mEq/L; base excess, −10.8; and SO2 99.0%. The patient’s preoperative vital signs were as follows: BP, 94/62 mmHg; HR, 90 beats/min; and RR, 8 breaths/min. Anesthesia was induced by the administration of 4 mg/kg of IV thiopental, while tracheal intubation was facilitated by the administration of 1 mg/kg of IV succinylcholine. After tracheal intubation, anesthesia was maintained with O2 air, and 1.5–2.0 vol% sevoflurane. For the muscular block, 4 mg of vecuronium was administered. A live infant, weighing 3.06 kg, was delivered. The incision to delivery time was 3 min. The Apgar score was 8/9. After 40 minutes, the patient was transferred to a recovery room after extubation, because she showed complete recovery during the emergence period. After that point, her vital signs were stable, tidal volume > 400 ml, RR was 12 breaths/min, and SpO2 was 99% in room air. A neurological examination performed in the recovery room, showed that the patient had an alert mental status, and that her orientation-related mental functions were normal. The sensory and motor functions of the patient recovered completely, but she complained of mild numbness in her thighs and calves. Arterial blood gas analysis performed in the recovery room revealed the following: pH, 7.350; PaCO2, 30.7 mmHg; PaO2, 156.3 mmHg; HCO3-, 16.6 mEq/L; base excess, −7.8; and O2 saturation, 98.9%. After 1 hour in the recovery room, the patient was transferred to the...
general ward. After 2 days, no post-dural puncture headache or other neurological complications were observed, therefore, she was discharged along with the infant, who did not show complications, or neurological abnormalities.

Discussion

Critical complications during the administration of the obstetric epidural anesthetic agents include intravascular injection (1 in every 5,000 epidural cases), intrathecal injection (1 in every 2,900 epidural cases), subdural injection (1 in every 4,200 epidurals cases), and considerable or total spinal block (1 in every 16,200 epidural cases) [1].

Our patient showed progressive changes in mental status, respiratory depression, bradycardia, and hypotension during lumbar epidural analgesia, however, her motor functions were relatively unaffected. We could not perform any additional radiological tests to determine the location of the epidural catheter tip since the epidural catheter was accidentally removed when the patient was transferred to the operating room. However, on the basis of the patient’s symptoms and signs, subdural injection was inferred as the cause of these complications. Many case reports have shown that subdural injection results in distinct effects in patients, such as moderate hypotension, slow symptoms onset, progressive respiratory depression, lack of coordination, and complete recovery after almost 2 hours [2].

In fact, the needle or catheter can be accurately located during epidural anesthesia by using many methods. LOR is one of the traditional techniques for locating the epidural space, while intravascular and subarachnoid injection can be avoided by the aspiration of CSF or blood via a needle or catheter. Administration of a test dose, i.e., a small amount of a local anesthetic and epinephrine mixture, aids in accurate location of the epidural space. If possible, the epidural anesthetic agent should be administered under guidance such as, that of C-arm imaging [3]. However, the results of many studies have indicated that subdural injection is a negative finding as in the current case [4]. Because C-arm guidance for anesthesia is contraindicated in pregnant women because of the required radiation exposure, the possibility of subdural injection during obstetric epidural anesthesia can not yet be completely negated. One study that used radiographs and contrast agents to monitor the needle showed partially inaccurate placement of the Tuohy needle in 17% of the cases and partial subdural placement in 7% of the cases despite the procedures being performed by experienced personnel using conventional methods to identify the dural space [5]. Several considerations should be made to minimize unrecognized subdural injections. First, anesthesiologists must be careful when handling Tuohy needles in the epidural space; second, difficult blocks and previous back surgery are predisposing factors of accidental subdural injection; third, a repetitive neuroaxial block on the same intervertebral space can cause accidental subdural injection; and fourth, continuous epidural catheter techniques should be conducted in a fractioned manner [6].

Temporary hypotension during neuraxial epidural anesthesia may not affect the mother and fetus, but it can be fatal to both if it persists. Several measures are considered for the treatment of hypotension: intravenous administration of fluids with 500 ml of colloid or 1,000 ml of crystalloid, avoidance of uterine aortocaval compression, vigilant monitoring of blood pressure, and IV vaspressors, such as 5–10 mg of ephedrine or 40–100 μg of phenylephrine are helpful [7]. Anesthesiologists generally use ephedrine in gradually increasing doses and continuously monitor blood pressure. According to some studies, phenylephrine is more effective and safer than ephedrine [8]. Atropine is known to rapidly cross the placenta and directly cause fetal tachycardia [9]. In instrumented studies on sheep, atropine was found to improve fetal oxygenation during fetal hypoxic bradycardia [10]. Unlike atropine, glycopyrrolate does not cross the placenta; therefore, it does not cause fetal tachycardia [11]. In healthy parturients, the plasma, placental artery, and vein concentrations of atropine were determined after IV or intramuscular administration of 0.01 mg/kg atropine, showing faster distribution and elimination of atropine and higher mean peak maternal plasma levels for IV administration than those observed for intramuscular administration [12]. Therefore, IV atropine is more effective in emergencies like fetal distress [13].

A few interventions must be considered during intruterine resuscitation in cases of fetal distress. The first is change in position; use of the left lateral recumbent position or, if required, the right lateral or knee-elbow position can help the fetus [13,14]. The second is rapid fluid replacement and supplementation with 100% O₂ [13,14]. The third is suppression of uterine contractions and an intra-amniotic infusion of warmed crystalloid solution [14]. The purpose of these interventions is to correct fetal hypoxia and acidosis [14]. If fetal distress dose not alleviate, emergency cesarean section can be considered [15]. In the case of our patient, although the fetal heart rate returned to the normal range after appropriate management, the mother’s symptoms were likely to aggravate the condition. Consequently, we decided to perform a cesarean section.

In conclusion, complications like injecting an anesthetic agent into the subdural space during epidural labor analgesia can be fatal for both the mother and the fetus. Therefore, anesthesiologists should have thorough knowledge about the complications involved in epidural anesthesia administration and their characteristic symptoms as well as be prepared for maternal and intruterine fetal resuscitation before proceeding with an epidural analgesia block at locations other than the
operating room. Furthermore, the patient and fetus should be closely monitored for a sufficient amount of time owing to the possibility of delayed onset of epidural anesthesia-related complications.

References

1. Jenkins JG. Some immediate serious complications of obstetric epidural analgesia and anaesthesia: a prospective study of 145,550 epidurals. Int J Obstet Anesth 2005; 14: 37-42.
2. Collier C. Total spinal or massive subdural block? Anaesth Intensive Care 1982; 10: 92-3.
3. Yun JS, Kang SY, Cho JS, Choi JB, Lee YW. Accidental intradural injection during attempted epidural block -A case report. Korean J Anesthesiol 2011; 60: 205-8.
4. Reynolds F, Speedy HM. The subdural space: the third place to go astray. Anaesthesia 1990; 45: 120-3.
5. Mehta M, Salmon N. Extraludal block. Confirmation of the injection site by X-ray monitoring. Anaesthesia 1985; 40: 1009-12.
6. Agarwal D, Mohta M, Tyagi A, Sethi AK. Subdural block and the anaesthetist. Anaesth Intensive Care 2010; 38: 20-6.
7. Tsen LC. Anesthesia for obstetric care and gynecology surgery. In: Anesthesiology. 2nd ed. Edited by Longnecker DE, Brown DL, Newman MF, Zapol WM: New York, McGraw-Hill. 2012, pp 1148-68.
8. Birnbach DJ, Ranasinghe JS. Anesthesia complications in the birthplace: is the neuraxial block always to blame? Clin Perinatol 2008; 35: 35-52.
9. Clark RB, Brown MA, Lattin DL. Neostigmine, atropine, and glycopyrrrolate: does neostigmine cross the placenta? Anesthesiology 1996; 84: 450-2.
10. Parer JT. The effect of atropine on heart rate and oxygen consumption of the hypoxic fetus. Am J Obstet Gynecol 1984; 148: 1118-22.
11. Naughton NN, Cohen SE. Nonobstetric surgery during pregnancy. In: Obstetric anesthesia. 3rd ed. Edited by Chesnut DH: Philadelpia, Mosby, 2004, p 264.
12. Kanto J, Virtanen R, lisalo E, Mienpää K, Liukko P. Placental transfer and pharmacokinetics of atropine after a single maternal intravenous and intramuscular administration. Acta Anaesthesiol Scand 1981; 25: 85-8.
13. Pan PH, Moore CH, Ross VH. Severe maternal bradycardia and asystole after combined spinal-epidural labor analgesia in a morbidly obese parturient. J Clin Anesth 2004; 16: 461-4.
14. Thurlow JA, Kinsella SM. Intrauterine resuscitation: active management of fetal distress. Int J Obstet Anesth 2002; 11: 105-16.
15. Morgan GE, Mikhail MS, Murray MJ. Clinical anesthesiology. 4th ed. New York, Lange Medical Books. 2006, pp 890-921.