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A case of transient neurologic symptoms following epidural mepivacaine and spinal tetracaine

Tamie Takenami, Yoo Kondou, Hiroshi Kimotsuki, Hirotsugu Okamoto, and Sumio Hoka

Department of Anesthesiology, Kitasato University, 1-15-1 Kitasato, Sagamihara, Kanagawa 228-8555, Japan

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Introduction

Several reports suggest that transient neurologic symptoms (TNS), characterized by postoperative development of pain or dysesthesia radiating into the buttocks or lower extremities, represent a mild manifestation of possible neural damage, although the etiology of the symptoms remains unclear. The symptoms have been reported after spinal anesthesia with local anesthetics such as lidocaine [1], tetracaine [1], bupivacaine [1], and mepivacaine [2], but seldom after epidural anesthesia [3,4]. We describe here a case of TNS that occurred after recommended doses of epidural mepivacaine and spinal tetracaine.

Clinical report

A 46-year-old woman (height 149 cm, weight 47 kg) was scheduled for curettage of the endometrium by hysteroscopy to confirm the diagnosis of a suspected uterine cancer. Her physical status was ASA I without any neurological symptoms, including lumbago. She received diazepam (10 mg) as oral premedication 40 min before arrival at the operating room. With the patient in the left lateral position, epidural puncture was made with an 18-gauge needle at the L4–L5 interspace with a loss-of-resistance technique. The 20-gauge catheter was advanced 3 cm into the epidural space. After a test dose of 2 ml of 1% mepivacaine, 14 ml of 2% mepivacaine was injected. Cold sensation was lost from T10 to S3 on the left side and from T10 to L1 on the right side 15 min after epidural injection. Because the level of hypesthesia was considered insufficient for the curettage procedure, especially on the right side, 20 min after the epidural injection, spinal puncture was performed using a 25-gauge Sprotte needle after removal of the epidural catheter. Two milliliter of 0.5% tetracaine dissolved in 5% glucose solution was intrathecally injected at the L4–L5 interspace with the patient in the left lateral position. This resulted in the spreading of hypesthesia bilaterally from the T4 to the S5 level that was considered sufficient for the operation. Both anesthetic procedures were performed easily, without any bleeding, and no paresthesia occurred during insertion of the needle or catheter or during drug injection. The patient was subsequently placed in the lithotomy position for approximately 30 min. The curettage procedure was uneventful and lasted for 10 min. Vital signs were stable during curettage. Six hours after intrathecal injection, sensory and motor functions were completely recovered in both lower extremities.

On the first postoperative day, the patient complained of numbness on the posterior side of the left lower extremity, and the numbness radiated to the ankle. The patient could not walk because of severe pain in the left leg that occurred when her feet touched the floor. At rest, she felt numbness rather than pain. On the second postoperative day, the numbness spread bilaterally to the lower back, buttocks, and thighs. She did not complain of bowel or bladder dysfunction. There was no significant hyperalgesia or sensory loss according to the cold and pinprick tests. Motor nerve function was intact in both extremities, and there was no impairment of deep tendon reflexes. There were no abnormalities on lumbar radiographic examination. The symptoms gradually disappeared 4 days after surgery, and the patient was discharged 5 days after surgery without any symptoms.

Address correspondence to: T. Takenami
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Discussion

In the present case, the patient had numbness in the lower back and buttocks radiating to the dorsolateral sides of the bilateral thighs and calves toward the ankle within 24 h after spinal anesthesia, without any objective sensory or motor deficits. These symptoms are most likely to reflect neurologic damage in the L5-S1 radicular territory, but not limited to the sciatic nerves. The symptoms spontaneously resolved within 5 days. These findings are compatible with TNS.

TNS are defined only by subjective symptoms and cannot be detected by objective neurological tests [5]. The pathophysiological mechanisms of TNS have not been clearly identified. Several clinical studies examined the difference in incidence of TNS caused by various local anesthetics [1]. These studies suggest that TNS may be related to the neurotoxic properties of the local anesthetics themselves. However, several factors may participate in the development of neurotoxic effects. In our patient, TNS developed after the use of the recommended dose of 10 mg of 0.5% tetracaine. A case of TNS with the same dose of tetracaine was also reported previously [6], where 1 mg of phenylephrine was added to the intrathecal tetracaine. The same investigators compared the incidence of TNS after spinal anesthesia with 0.5% tetracaine(11.4–11.9 mg) dissolved in 7.5% or 0.75% glucose solution, with or without phenylephrine [7], and demonstrated that phenylephrine significantly increased the incidence of TNS [12.5% (10/80) vs 1.3% (1/80)], but additional glucose was not relevant to the incidence. However, although the intrathecal solution that our patient received did not contain phenylephrine, it is still possible that spinal tetracaine was responsible for the symptoms in our patient.

It is possible that trauma to spinal nerves occurred during placement of the spinal needle, because the puncture was performed 15 min after epidural injection of 2% mepivacaine. However, because touch sensation remained intact at the time of spinal needle insertion, it is unlikely that the spinal needle damaged the nerve roots without eliciting paresthesia.

The patient was positioned in the lithotomy position, which may predispose the patient to the development of radicular symptoms [1,8]. It is suggested that the lithotomy position stretches the nerve roots, especially the dorsal roots of L5 and S1, which may reduce blood flow and increase the vulnerability of the nerve to local anesthetics [8]. Our patient developed symptoms related to L5 and S1 radicular territory. Although the period spent in this position was relatively short, a similar short period in the lithotomy position has been demonstrated previously as a risk factor for TNS [2].

Another possible factor is the injection of 14 ml of 2% mepivacaine into the epidural space prior to intrathecal tetracaine. Development of TNS after epidural analgesia was recently documented in three separate reports [3,4,9]. Freedman and Rudow [4] suggested that the low cerebrospinal fluid (CSF) concentration of lidocaine, even in epidural anesthesia, might cause TNS. A number of laboratory studies suggested that all local anesthetics are potentially neurotoxic [9]. In our previous histological studies using rats, the primary location of the neurotoxic lesion caused by relatively high doses of tetracaine [10], lidocaine [11], and mepivacaine [11] was commonly at the posterior root. Since our patient received both spinal and epidural anesthesia at the same interspace, epidural mepivacaine may synergistically enhance the neurotoxic effect of intrathecal tetracaine. Although the epidural anesthetic effect was insufficient, especially on the right side, the concentration of mepivacaine in the CSF after epidural injection could have reached near-neurotoxic levels. It has been reported that a small dose of epidural bupivacaine (5 ml of 0.25% solution), which is considered less neurotoxic than mepivacaine, caused TNS [3]. In our case, epidural anesthesia caused a more pronounced effect on the left side than on the right side. This may account for the onset and prolonged symptoms on the left side.

In summary, we have reported a patient who developed symptoms compatible with TNS after spinal anesthesia with a clinically recommended dose of tetracaine. The combination of the lithotomy position and epidural mepivacaine prior to spinal tetracaine may have had a synergistic effect on the symptoms. Careful consideration of the total local anesthetic dose at relatively short intervals may be needed during combined spinal and epidural anesthesia.

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