J Anesth (2001) 15:106–107

Journal of
Anesthesia

A case of cauda equina syndrome following spinal anesthesia with hyperbaric dibucaine

Ken-ichiro Akioka, Kazunori Torigoe, Hajime Maruta, Nobutaka Shimizu, Yuko Kobayashi, Yoshiro Kaneko, and Rinji Shiratori

Department of Anesthesia, Nihon University, Nerima Hikarigaoka Hospital, 2-11-1 Hikarigaoka, Nerima-ku, Tokyo 179-0072, Japan

Key words Cauda equina syndrome · Dibucaine

Introduction

More than 400000 ampules of dibucaine chloride (Neoperucamine S), a hyperbaric local anesthetic for spinal anesthesia, are used every year in Japan. Among recent reports of adverse events caused by this drug, there have been only a few reports of persistent sensory [1] or urinary [2] disorders. Most neuropathies caused by spinal anesthesia are mild, and patients complain of nondescript symptoms that usually disappear in a few days. Hence, only a small percentage of neuropathies are reported to the anesthesia department.

This case report describes a patient who had dysuria and decreased perineal sensation for a few weeks following spinal anesthesia.

Case report

A 51-year-old ASA Class I woman (150 cm, 52 kg) diagnosed with right chondrosteoma was hospitalized to undergo surgery on the right first toe.

Anesthesia

When a 22 G needle was inserted into the subarachnoid space at the L4-5 interspace, the patient experienced radiating pain in her right leg, so the needle was removed immediately. Once the needle was withdrawn, the patient experienced pain no longer. Consequently, the needle was reinserted at the same interspace in another direction, and 2.0 ml of 0.24% dibucaine chloride containing 66 μg of epinephrine was gradually injected. Following drug injection, the patient did not have any complaints. The patient was placed in a supine position during the surgery. The analgesic level at the start of surgery was L1. The vital signs during anesthesia and surgery were stable, and the postoperative analgesic level remained at L1.

Three days after anesthesia, the patient complained of enuresis, and desensitization was confirmed in the S2–S5 area. However, the patient did not have motor disturbance of the lower extremities or bowel dysfunction. The area of desensitization gradually decreased and had almost disappeared at the time of discharge. Although the patient could urinate, she was unable to empty her bladder completely, so self-catheterization was required. On the same day, administration of the following drugs was initiated: 1.0 mg of distigmine bromide, 1.5 mg of mecobabalamin, and 3 mg of prazosin hydrochloride. On days 3 and 4, 1 g of sodium methylprednisolone succinate was drip infused. Also, on day 6, 0.5 mg of neostigmine was administered intramuscularly. As spontaneous urination increased gradually, the feeling of urinary retention was alleviated. On day 12, the patient was able to urinate naturally. On day 19, the desensitization completely disappeared, and the patient was discharged on day 20.

Discussion

One of the most common factors of neuropathies following spinal anesthesia is nerve damage caused by the needle used for spinal anesthesia. The present patient experienced transient radiating pain in the leg during needle insertion. The pain disappeared when the injection needle was removed, and the patient did not complain of pain or numbness after injection of the local anesthetics. Furthermore, the location of the radiating

Address correspondence to: K. Akioka
Received: June 25, 1999 / Accepted: December 8, 2000

pain was different from that of the desensitization. Drasner et al. [3] recommend caution in attempting a second spinal injection. We must also pay special attention if the first attempt is unsuccessful. When performing spinal anesthesia, we use the following steps: (1) a narrow-gauge needle (23–25 G) is used; (2) when a patient complains of radiating pain, the injected needle is removed immediately without inserting it further or rotating it; and (3) when a needle is reinserted, the direction of insertion is altered, and the internal needle is removed at a shallower depth to assess the backflow of the spinal fluid. It is important to keep in mind that the tips of current disposable needles are very sharp, thus making it easier to damage nerve fibers. Schneider et al. [4] investigated the cause of transient neurological symptoms (TNS), and reported that when the cauda equina is stretched during surgery (dorosacral position), the nerve is more likely to be damaged during spinal anesthesia.

In addition to direct nerve damage, the following factors may contribute to the onset of neuropathies following spinal anesthesia: delayed pharmacological action of local anesthetics, neurological ischemia caused by the vascular contraction induced by epinephrine, and miscellaneous physiological symptoms. Sakura et al. [5] reported that local anesthetics play the most important role in the onset of neuropathies following spinal anesthesia. There have been several reports of TNS caused by currently available anesthetics [6–8]. At present, research is being conducted to investigate the effects of the concentration of local anesthetics [9], specific gravity [10], and vasoconstrictors [11]. Most neuropathies caused by these factors are reversible. However, it is necessary to examine patients closely to distinguish irreversible neurological damage from reversible damage.

In conclusion, we reported a patient with cauda equina syndrome following spinal anesthesia with hyperbaric dibucaine who had mild bladder dysfunction and perineal desensitization for a few weeks, without bowel or motor dysfunction of the legs. Based on the present findings, we believe that thorough explanation and early treatment are important in the treatment of neuropathies following spinal anesthesia.

References

1. Fukuda H, Ikeno S, Ishii R (1994) Meralgia paresthetica after spinal anesthesia. Masui (Anesthesia) 43:1073–1075
2. Osuga K, Hirabayashi Y, Fukuda H (1999) Severe lightning limb pain induced by spinal anesthesia. Masui (Anesthesia) 48:67–69
3. Drasner K, Ringler ML (1991) Repeat injection after a "failed spinal": at times, a potentially unsafe practice. Anesthesiology 75:713–714
4. Schneider M, Ettlin T, Kaufmann M (1993) Transient neurologic toxicity after hyperbaric subarachnoid anesthesia with 5% lidocaine. Anesth Analg 76:1154–1157
5. Sakura S, Sumi M (1996) Transient neurologic toxicity? Anesth Analg 83:887–888
6. Sumi M, Sakura S, Kosaka Y (1996) Intrathecal hyperbaric 0.5% tetracaine as a possible cause of transient neurologic toxicity. Anesth Analg 82:1076–1077
7. Lynch J, zur Nieden M, Kasper SM (1997) Transient radicular irritation after spinal anesthesia with hyperbaric 4% mepivacaine. Anesth Analg 85:872–873
8. Casati A, Fanelli G, Aldegheri G (1998) A transient neurological deficit following intrathecal injection of 1% hyperbaric bupivacaine for untilateral spinal anesthesia. Eur J Anesthesiol 15:112–113
9. Hampl KF, Schneider MC, Pargger H (1996) A similar incidence of transient neurologic symptoms after spinal anesthesia with 2% and 5% lidocaine. Anesth Analg 83:1051–1054
10. Hampl KF, Schneider MC, Thorin D (1995) Hyperosmolarity dose not contribute to transient radicular irritation after spinal anesthesia with hyperbaric 5% lidocaine. Reg Anesth 20:363–368
11. Sakura S, Sumi M, Sakaguchi Y (1997) The addition of phenylephrine contributes to the development of transient neurologic symptoms after spinal anesthesia with 0.5% tetracaine. Anesthesiology 87:771–778