Case Report

An unusually prolonged duration of spinal anaesthesia following 0.5% Levobupivacaine

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Abstract

Spinal anaesthesia is the method of choice for elective caesarean delivery. Levobupivacaine may produce a sensory and motor block that is different from that produced by bupivacaine, the most popular local anaesthetic for parturients undergoing caesarean section (CS). We present a case of unexpectedly prolonged spinal anaesthesia following a successful spinal block with levobupivacaine. There was no evidence of any neurological injury in this patient during injection.

Keywords: Anaesthesia: spinal, Anaesthetics: local.

Introduction

Various techniques of central neuraxial blockade have been successfully used for Caesarion Section (CS) surgery. Single-shot spinal anaesthesia (SPA) seems to be the most common procedure as it provides a dense block with rapid and predictable onset.[1]

Levobupivacaine, the pure S(-)-enantiomer of racemic bupivacaine, has recently been introduced for routine obstetric and nonobstetric spinal and epidural anaesthesia, peripheral nerve blocks, and infiltration analgesia.[2] In a study by Vercauteren et al., intrathecal levobupivacaine had a similar clinical profile to that of racemic bupivacaine for intrathecal labour analgesia but caused less motor block at similar doses.[3] The longer-lasting sensory block and the rapid mobilization of patients due to the faster recovery from motor block and reduced hypotension associated with levobupivacaine may be particularly advantageous for obstetric patients.[4,5] The lowest local anaesthetic dose of levobupivacaine for CS anaesthesia was reported to be 11.1 mg, with increased

concentrations allowing for more intense motor block.[2]

We present a case with unexpectedly prolonged motor and sensory block following anaesthesia with isobaric 0.5% levobupivacaine for CS.

Case Report

A 41-year old patient (body weight 68 kg, height 170 cm) with 38-week gestation was planned to have an elective caesarean section under spinal anaesthesia. The patient had a past history of uncomplicated caesarean section under general anaesthesia and no accompanying systemic conditions. Preoperative routine laboratory analyses revealed normal complete blood count and biochemical parameters.

The patient was placed on the operating room table and following 750 mL 0.9% NaCl infusion, her heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and peripheral oxygen saturation (SpO_2) were monitored, and preoperative measures were recorded (HR 92 beats/min, SBP 130mmHg, DBP 80 mmHg, SpO_2 99%). In addition, she was administered oxygen (O_2) at a rate of 4 L/min through a nasal catheter.

With the patient lying on the left lateral, side a 27-gauge Quincke spinal needle was used to access the subarachnoid space through the L3-4 interval with a single puncture. After free flow of the cerebrospinal fluid (CSF) was visualized, 14 mg isobaric levobupivacaine + 10 mcg fentanyl solution was injected in the CSF within 20 seconds. Then the patient was placed in the supine position, and the uterus was deviated to the left by elevating the right hip with a pillow. Vital signs were monitored. For the assessment of spinal anaesthesia, sensory block was measured with the hot-cold test, and the Bromage scale was used for motor block assessment. In regular assessments carried out following the drug administration to spinal gap, it was observed that the sensory block at the 15th minute was at T4 level and Bromage scale score was 3, and surgery was started.

Four minutes after the start of the operation, a live male baby was delivered. Intraoperative monitoring of haemodynamic and respiratory parameters revealed HR 60-107 beats/min, SBP 100-120 mmHg, DBP 50-65 mmHg, and SpO_2 98-99%. The patient did not develop hypotension and did not require ephedrine or atropine during the operation. The intervention was completed after 40 minutes without any complication. After the patient left the recovery room, we evaluated the patient for the second time. However, in the third evaluation, it was decided to carry out these evaluations more frequently, or hourly after observing that the block had still not regressed (at the 5th hour following spinal anaesthesia). In the postoperative visit five hours after the spinal anaesthesia, the sensory block was still at T7-8 level, and the motor block according to the Bromage scale remained at grade 3. Thus, to avoid missing any neurological complications, sensory and motor blocks were evaluated hourly (Table). Complete recovery of the sensory and motor blocks was observed at nine hours after spinal injection of the drug. The patient required an analgesic at seven hours after anaesthesia. She was mobilized 24 hours later. No neurological symptoms or signs such as urinary or anal incontinence, dysesthesia, and lumbar or leg pain developed in conjunction with the prolonged sensory and motor block. The patient was discharged from the hospital at postoperative day two without any complications. She did not consult a neurologist, and no tests were ordered because she had total recovery of the motor and sensory block with no remanant neurological symptoms or signs.

Table: Postoperative assessment of sensory and motor block.

	5 hours	6 hours	7 hours	8 hours	9 hours
Sensory Block	T7-8	T9-10	L1-2	S1-2	-
Motor Block	3	3	2	1	0

Discussion

Spinal anaesthesia for CS has gained popularity over epidural techniques because of its easy placement and rapid onset. In pregnant women, engorgement of epidural veins from aortocaval compression with displacement of CSF may contribute to the undesired cephalad extensions of the block. Furthermore, CS is a relatively short-duration procedure that is often followed by early mobilization of the patient. In turn, this increases the potential for late extension of the block.[6] There is still some common belief that plain local anaesthetics give unpredictable blocks. Reports indicate that plain local anaesthetics may in fact be hypobaric and may show cephalad extensions as a late complication.[7]

Levobupivacaine, the pure S(-)-enantiomer of racemic bupivacaine, was developed to improve the cardiac safety profile of bupivacaine. Although the potency of levobupivacaine is similar to that of bupivacaine when in vivo, in vitro, and human pharmacodynamic studies regarding nerve blockades have been conducted, it has low cardiovascular and central nervous system toxicity. In addition, the anaesthetic-analgesic effects of the two drugs are comparable at similar doses. Sensory block by levobupivacaine may be more prolonged compared to bupivacaine. While this difference may be 25-30 minutes in epidural administration, it may last up to two hours in peripheral nerve blockades. With epidural administration, levobupivacaine produced less prolonged motor block than sensory block.[8]

Although it was reported that a spinal dose of 15 mg levobupivacaine provided sensory block for up to 6.5 hours,[8] sensory block levels change as suggested by newer studies in which spinal levobupivacaine was used in pregnant women and in our case, periods of time especially shorter than motor block time are reported. In their study that investigated spinal levobupivacaine median effective dose (E50) and MLAD for caesarean section, Celleno et al. established the MLAD of levobupivacaine for caesarean section as 11.10mg.[2]

Bouvet et al. reported that, when combined with intrathecal sufentanil and intrathecal morphine 100 µg, the ED95 of intrathecal levobupivacaine was 12.9 mg for caesarean delivery. If doses of levobupivacaine less than the ED95 particularly near the ED50 were used, the doses should be administered under a CSE technique, in order to be able to complete insufficient analgesia during surgery. In this study, reaching T6 level with levobupivacaine was reported to be 6.9 (3.1). A complete recovery from motor block (time for Bromage's scale=0) was reported to be 183(67) min.[9]

In their study that investigated influence of positioning on plain levobupivacaine spinal anaesthesia in cesarean section, Gori et al. used 12.5 mg levobupivacaine.[6]

When lower spinal local anaesthesia by combined spinal-epidural technique was used, Gunusen et al. found that levobupivacaine 10 mg combined with fentanyl 10µg provided fast and effective induction of surgical anaesthesia for cesarean section. A complete recovery from motor block with this dose was in 112.5 minutes (60-135); and regression to T10 level was reported to be in 125.2±12.9 minutes (13).[10]

The levobupivacaine dose used on our case (14 mg) was close to the ED95 dose reported for levobupivacaine (12.9 mg). As also reported in literature, if we were to use a combined technique we could have used a lower dose. However, since we adopted single shot spinal anaesthesia technique and considering the height of the patient, we believe that the spinal levobupivacaine dose administered was adequate for surgery. While, in our case, perfect surgical anaesthetic conditions were obtained, no side effects as hypotension or bradycardia were observed. The patient did not reveal any need for additional opioid or sedative agents. However, since sensory and motor block recovery times are longer, we believe that there is an extension due to levobupivacaine especially in the recovery from motor block. The addition of various doses of opioids may allow the dose of local anaesthetic to be reduced, producing a synergistic effect that enhances analgesia and prolong the duration of the sensory block without intensifying motor block.[11,12]

Central nervous system and cardiac toxicity are well-established complications of regional anaesthesia. Two cases of grand mal seizures were reported after accidental intravenous injection of levobupivacaine.[13] Another report presented a patient who demonstrated generalized seizure activity after an injection of 30 mL of levobupivacaine 0.5% for interscalene brachial plexus block.[14] Although convulsions following the administration of racemic bupivacaine are a well-recognized complication, a case was reported with self-limiting convulsions following the institution of an axillary brachial plexus block with levobupivacaine.[15]

Prolonged neurological deficit was reported after the institution of 5% bupivacain for interscalene block.[16] In another report presenting a patient who developed unexpected neurological deficit after injection of 0.75% bupivacaine, the authors suggested that psychiatric disorders, such as conversion, might be a rare cause of neurological deficit following successful regional anaesthesia procedures.[17]

Only a few studies have investigated obstetric spinal anaesthesia using plain levobupivacaine. Gautier et al. reported the duration of spinal anaesthesia (first demand for analgesics) with an intrathecal levobupivacaine and sufentanyl combination as 136 minutes.[18]

Gori et al. investigated the effects of intrathecal 12.5 mg 0.5% isobaric levobupivacaine administration for CS and reported that the regression time of anaesthesia by two dermatomes at the supine position was 76 minutes, and the regression time up to L1 was 158 minutes. The regression time up to S1 was 230 minutes. The mean time to complete motor recovery and the mean time to the first requirement of analgesics were 159 minutes and 131 minutes, respectively.[6]

The lower volume of CSF was suggested to be associated with prolonged sensory block, but the same mechanism would not be applicable for prolonged motor block.[19]

In a case report, prolonged motor and sensory block after low-dose spinal hyperbaric bupivacaine administration was attributed to low CSF volume. The authors reported that the prolonged effect might occur without any additional signs related to spinal bupivacaine administration.[20] Prolonged block due to low CSF volume cannot be only connected with the use of low dose spinal local anaesthesia. It was reported that lumbosacral CSF volume inversely correlated with peak sensory block level and with time required for regression of the sensory block using 3 ml plain 0.5% bupivacaine for spinal block.[21]

Epidural ropivacaine 1% administration following

spinal bupivacaine administration was reported to cause prolonged intense motor block in two cases. The prolonged block was attributed to the interaction between the two amid group local anaesthetics. The authors advised to be on the alert for unexpected motor block in cases that received epidural ropivacaine after spinal bupivacaine administration.[22]

Regarding the difference between motor and sensory block, in an experimental rat model, in contrast to clinical expectations, rat ventral (motor) nerve roots seemed to be more sensitive to low concentrations of bupivacaine than dorsal (sensory) nerve roots.[23]

The literature search about prolonged block after clinical administration of levobupivacaine revealed no cases other than prolonged axillary block after axillary administration in a uraemic patient. Motor block ended at thirty hours in that case, and the authors attributed this to uraemic neuropathy.[24]

In our case, the effect of spinal isobaric levobupivacaine lasted almost two times the reported duration in the literature. Therefore, this is the first reported case of prolonged sensory and motor block with a longer recovery time for motor block compared to the recovery for sensory block.

In cases with prolonged block, the differential diagnosis should include haematoma formation secondary to spinal intervention, cauda equina syndrome, transient radicular irritation (TRI), and anterior spinal artery syndrome.[20] There was no pain, paresthesia, or haemorrhage during the insertion of the spinal needle, and spinal puncture was successful at the first attempt in our case. Thus, development of haematoma or nerve damage due to needle insertion was not likely. Moreover, the patient did not develop low blood pressure that would decrease blood flow, nor was she administered intravenous vasopressor drugs. Considering the height of the patient, the levobupivacaine and opioid doses used were in accordance with the suggested doses in the literature . No other neurological symptom or sign developed in conjunction with prolonged sensory and motor block in the postoperative period in our patient.

Transient neurological symptoms (TNS) and TRI are terms used to describe bilateral pain in the lower back or buttocks and/or radiating down the lower extremities which develop after recovery from spinal anaesthesia. They are not accompanied by any motor, sensory, or sphincter function loss or signs of meningeal irritation.[25] Cauda equina syndrome and TRI are associated with accompanying vascular conditions, old age, epinephrine or hyperbaric bupivacaine use, and the lithotomy position in cases undergoing spinal anaesthesia. The causative mechanism is suggested to be nerve cell membrane damage and neuronal injury due to the high concentrations of local anaesthetics.[20]

We did not detect any additional neurological symptom or sign in our case that could be associated with the above mentioned pathologies, and we did not perform any laboratory tests since the patient recovered completely after nine hours.

In conclusion, although local anaesthetics are usually held responsible for prolonged sensory block rather than motor block, we suggest that the low CSF volume may account for the prolonged block in our case. The possibility of an unexpectedly prolonged block should be considered in pregnant patients who receive spinal levobupivacaine administration. Such patients should be closely monitored for additional neurological signs.

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