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# A Comparison of the Sensory and Motor Blockade Duration of Intrathecal Lidocaine 5%, Lidocaine 5% Plus Epinephrine and Lidocaine 5% Plus Dexamthasone: A Double Blind Randomized Clinical Trials Study

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**Abstract:** In the present study we conducted a randomized, prospective, double-blind, placebo-controlled clinical trial to evaluate the prolongation of lidocaine spinal anesthesia by intrathecal administration of dexamethasone. Ninety male patients scheduled for orthopedic surgery under spinal anesthesia were enrolled in the study and were randomly allocated to one of three groups and received their treatments intrathecaly; Group 1: 75 mg 5% lidocaine + 2 mL 0.9% NaCl; Group 2: 75 mg 5% lidocaine + 0.2 mg epinephrine (0.2 mL-BP) +1.8 mL 0.9% NaCl and Group 3: 75 mg 5% lidocaine + 8 mg dexamethasone (2 mL-BP). After performance of the block patients were kept in supine position and the pinprick level were kept between $T_6$ to $T_8$ in all patients. Block regression was estimated by pinprick every 5 min until a 4 sensory level regression from highest level. The duration of motor block was the time needed until the block returned to level 0 from level 3 on the Bromage scale. There were no significant differences in demographic data, duration of surgery, ASA classes (I/II), the maximal cephaled level and onset time of sensory and motor block among the groups. The duration of sensory block was significantly longer in the lidocaine-epinephrine and lidocaine-dexamthasone groups than the lidocaine group (respectively 85.7 and 82.1 min vs. 55.9 min for sensory block and 112.8 and 118.9 min vs. 79.2 min for motor block, $p<0.001$). The incidence of complications and the need for treatments were not different among groups. After one month follow up, no neurological or infectioneuos disorder was found in patients. We have shown that the addition of dexamethasone (8 mg-BP) intrathecaly to lidocaine spinal anesthesia prolongs the duration of intrathecal lidocaine sensory and motor blocks.

**Key words:** Dexamethasone, epinephrine, intrathecal injection, spinal anesthesia

## INTRODUCTION

Different additives have been used to prolong spinal anesthesia. Vasoconstrictors were originally added in spinal anesthesia to produce vasoconstriction of the spinal vessels, there by reducing vascular absorption of the local anesthetic[1]. Epinephrine is commonly added to lidocaine in an attempt to achieve a spinal anesthetic of intermediate duration. Some physicians have been concerned that the use of vasoconstrictors may be risky[2]. So, it seems necessary to find a substitute drug for vasoconstrictors when there is a contraindication to their use. A few studies have demonstrated the analgesic effect of corticosteroids[3,4]. Previously, it was found that dexamethasone can prolonged the blockade duration in the periphery[5,6]. The aim of this study was to evaluate the prolongation of lidocaine spinal anesthesia by intrathecal administration of dexamethasone.

## MATERIALS AND METHODS

After the Institutional Review Board approval and informed consent were obtain, 90 ASA physical status I and II male patients aged 25-45 years, height 160-180 cm scheduled for short orthopedic surgery(<60 min) under spinal anesthesia were admitted to the study. No patient had neurologic disorders and any contraindication for spinal anesthesia or dexamethasone administration. The patients were randomized into one of three groups (30 patients in each group) by A computer-generated randomization list that was drawn up by the statistician. Diazepam 5 mg intravenous for sedation were given to all patients in the operating room, ECG, NIBP and pulse oximeter monitoring were started. Base line Heart rate and blood pressure were measured and recorded every minute for 15 min after spinal blockade and then every 5 min until the end of surgery. After an intravenous injection of

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10 mL $kg^{-1}$ lactated Ringers solution, a mid line lumbar puncture was performed in the $L_{3-4}$ interspaces using 25 Quinck needle with the patients in lateral decubitus position and one of the following drug combinations administered:

Group 1: 75 mg 5% lidocaine + 2 mL 0.9% NaCl
Group 2: 75 mg 5% lidocaine + 0.2 mg epinephrine (0.2 mL-BP) + 1.8 mL 0.9% NaCl
Group 3: 75 mg 5% lidocaine + 8 mg dexamethasone (2 mL-BP)

Except Dr Movafegh and statistician, other study personnel and participants were blinded to treatment.

After performance of the block and until the end of the surgery the patients were kept in supine position. If a $T_8$ pinprick level had not been achieved, subjects were placed in a 5 to-$10^0$ trendelenburg position after 2 min and if the pinprick level at 2 min was > $T_7$ they were placed in a 10 to-$20^0$ elevation and thus, the pinprick level were kept between $T_6$ to $T_8$ in all patients.

All patients had received oxygen from nasal cannula. If there was a more than 30% reduction of mean arterial pressure from the baseline value, ephedrine 5 mg intravenous as a bolus was given with additional injections if needed. If heart rate decreased to <50 bpm, one bolus of 0.5 mg atropine intravenous was administered.

The development of sensory block was followed by pinprick every 1 min until achievement of $T_6$ to $T_8$ level. Block regression was estimated by pinprick every 5 min until a 4 sensory level regression from highest level. Motor block was assessed at the same time points using a modified Bromage scale[7], was defined as: level 0:no block (the ability to flex the knee and feet), level 1:Partial block (the ability to flex the knee and stand with full movement of the feet), level 2: nearly complete block (the inability to flex the feet) and level 3:complete block (the inability to move the legs or feet). The duration of motor block was considered as the time needed for the block to return from level 3 to level 0 on the modified Bromage scale.

In the current study we tested the hypothesis that the dexamethasone can prolonged 5% lidocaine spinal anesthesia. All patients were visited just before discharge from hospital and one month later and asked about any complication or neurologic disorders.

Sample size was estimated for detection 15 min block duration differences between groups with $\alpha$=0.5 and $\beta$=0.1. Statistical analysis was performed with SPSS package (SPSS Inc. chicago, II, USA). Demographic data, onset time and time to achieve the highest and duration of sensory and motor block were analyzed using one-way ANOVA and post hoc test with Tukeys method. Chi-squre analysis were used to compare differences of maximal block level, nausea and vomiting and hypotension or bradycardia treatments. $p<0.05$ was considered to be significant.

## RESULTS AND DISCUSSION

All spinal blocks were successful and no additional perioperative analgesic was needed. There were no significant differences in demographic data, duration of surgery and ASA classes (I/II) between groups (Table 1).

There were no significant differences in the maximal cephalad level and onset time of sensory and motor block among the groups (Table 2). The duration of sensory and motor blockade were different between groups ($p<0.001$ ANNOVA). Post hoc test with Tukey method showed that this times were significantly longer in lidocaine-dexamethasone and lidocaine-epinephrine groups than lidocaine group ($p<0.001$ Tukey), but there were no significant differences between two treatment groups (Table 2).

Table 3 shows that the incidence of complications and need for treatments were not different among groups. No neurologic or infectioneuos disorder occurred in patients.

The present results indicate that the addition of dexamethasone (8 mg-BP) to 5% lidocaine for spinal anesthesia provided significant prolongation of sensory and motor block in comparison with plain lidocaine and there is no difference between dexamethasone -lidocaine 5% and epinephrine (0.2 mg-BP)-lidocaine 5% in sensory and motor block duration.So, the onset time of sensory and motor blockade are similar between this additives and saline. The incidence of intraoperative nausea, vomiting and the need for antiemetic, atropine and ephedrine were similar in all groups.

The analgesic effect of epidural and spinal steroids have been reported in animal and human studies[4,8-11]. Mirzaie *et al.*[4] reported that the combination of corticosteroids and bupivacaine diminishes postoperative back pain experienced by patients undergoing lumbar disectomy in the immediate postoperative period. Kotani *et al.*[8] reported that intrathecal injection of methylprednisolone with lidocaine induced excellent and long-lasting analgesia for burning pain,lancinating pain and allodynia in patients with post herpetic neuralgia in the early stage of herpes zoster. Also, Taguchi *et al.*[12] administered betamethasone intrathecaly in three cancer patients. They concluded that intrathecal injection of bethamethasone can be a useful approach in some patients with intractable cancer pain.

Table 1: Demographic data, duration of surgery and ASA classes between groups

| | Lidocaine (n=30) | Lidocaine-Epinephrine (n=30) | Lidocaine-Dexamethasone (n=30) |
|---|---|---|---|
| Age (years) (Range)* | 33.50±9.06 (22-41) | 33.26±9.62 (20-43) | 29.80±8.01 (21-40) |
| Weight (kg)* | 71.63±8.74 | 75.24±11.59 | 76.83±13.71 |
| Height (cm)* | 165.2±6 | 171.8±7 | 173.4±8 |
| Duration of surgery (min)* | 46.6±3 | 48.2±2 | 47.8±3 |
| ASA classes (I/II) | 16/12 | 14/16 | 15/14 |

*Values are mean±SD, There were no significant differences between groups

Table 2: Duration of onset time and time to reach maximal sensory and motor blockade among diffferent groups

| | Lidocaine (n=30) | Lidocaine-Epinephrine (n=30) | Lidocaine-Dexamethasone (n=30) |
|---|---|---|---|
| Onset time for sensory block (sec) | 43.33±29.16 | 57.50±33.65 | 53.33±33.74 |
| Time to reach maximal sensory blockade level (min) | 10.63±4.31 | 11.26±4.81 | 12.36±4.28 |
| Duration of sensory blockade (min)** | 55.90±9.30 | 82.10±17.42* | 85.66±16.90* |
| Onset time for motor blockade (sec) | 49.52±16.24 | 62.41±21.45 | 69.65±23.51 |
| Time to reach maximal motor blockade level (min) | 14.41±5.57 | 15.66±17.78 | 16.23±4.37 |
| Duration of motor blockade (min)** | 79.23±7.42 | 112.75±17.78* | 118.89±16.35* |

*Values are mean±SD, **p<0.001 (ANOVA), * p<0.001 (Tukey post hoc test)

Table 3: Incidence of complications and need for treatment among different groups

| | Lidocaine (n=30) | Lidocaine-Epinephrine (n=30) | Lidocaine-Dexamethasone (n=30) |
|---|---|---|---|
| Nausea and vomiting | 4(14.3%) | 3(10.0%) | 3(10.0%) |
| Need to antiemetic | 2(6.7%) | 1(3.3%) | 2(6.7%) |
| Need to ephedrine | 4(14.3%) | 3(10.0%) | 3(10.3%) |
| Need to Atropine | 3(10.0%) | 2(6.7%) | 2(6.7%) |

Previous works demonstrated that addition of dexamethasone to local anesthetics prolonged duration of blockade of the peripheral nerves[5,6]. Castillo *et al.*[5] characterized a prolonged percutaneous blockade of the sciatic nerve in rats using bupivacaine-dexamethasone micro spheres[5]. Other study, demonstrated that incorporation of dexamethasone into bupivacaine micro spheres significantly prolongs intercostals nerve block in sheep[6].

Although corticosteroids have been studied for postoperative pain relief in oral, general and orthopedic surgery[13,14], other studies have not corroborated these reports[15,16].

The mechanism of the analgesia induced by corticosteroids is not fully understood. This effect is suspected to be mediated by their anti-inflammatory or immune-suppressive effects. Prostaglandins may play an important role in mediating various forms of spinal sensitization[17,18] and corticosteroids may modulate pain perception through their inhibitory effects on spinal prostaglandin production.

According to the traditional theory of steroid action, steroids bind to intracellular receptors and modulate nuclear transcription. But like the intrathecal betamethasone in Taguchi *et al.*[12] report, in this study intrathecal dexamethasone produced rapid effect. It appears that the mechanism for the analgesic effect of intrathecal steroid treatment described above doesn't explain this acute effect but it may be transmitted by specific membrane bind receptors[19,20].

However, there are several arguments about the safety of intrathecal steroids[21]. In animal experiments, repeated intrathecal injection of low-dose betamethasone[22] and triamecilonon acetate[10] did not induce spinal neurotoxicity.Intratechal steroids have been frequently used in the treatment of multiple sclerosis, mumps meningitis, central nervous system involvement in systemic lupus erythematosos and in the management of sciatica[22-24]. In the study reported by Kotani *et al.*[8] no complications were found in the 89 patients with post herpetic neuralgia who received four dose of intrathecal methylprednisolone acetate. In another study, after approximately 2000 intrathecal injections of dexamethasone (8 mg-BP) in 200 patients for treatment of post-traumatic visual disturbance, no serious complications or neurological disorder were found in one month follow up[25]. Like this study, we follow up our patients for one months and no infectioneous, neurological or other complications were found.

In conclusion, we have shown that like epinephrine (0.2 mg-BP), the addition of dexamethasone (8 mg-BP) intrathecaly to lidocaine spinal anesthesia prolongs duration of intrathecal lidocaine sensory and motor blocks.

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