ORIGINAL RESEARCH

To compare the intrathecal dexmedetomedine and magnesium sulfate as adjuvants to bupivacaine in total hip replacement

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ABSTRACT

Aim: To compare the intrathecal dexmedetomedine and magnesium sulfate as adjuvants to bupivacaine in total hip replacement. Materials and Methods: This prospective, randomized, double-blinded study enrolled 90 ASA physical status I and II patients scheduled for total hip replacement surgery under spinal anaesthesia, aged 35 to 75 years, of either gender, height 159 to 189 cm, and weight 52 to 88 kg after receiving written informed consent and institutional ethical committee approval. Using computer-generated random numbers, patients were allocated into three groups: Group D received 15 mg hyperbaric bupivacaine and 0.1 ml (10 µg) DXM, Group M received 15 mg hyperbaric bupivacaine and 0.1 ml (50 mg) Mg and Group C received 15 mg hyperbaric bupivacaine and 0.1 ml normal saline as control. Results: In compared to the control group C (5.01± 1.36 and 5.36± 1.21), the start time of block, both sensory up to T10 dermatome and motor to Bromage 3 scale, was quick in the DXM group D (3.01±0.98 and 4.11±1.04) and delayed in the Mg group M (7.11± 1.36 and 7.82± 1.55). One-way ANOVA with post tests revealed statistically significant differences between the groups in both the sensory (F=94.33, P<0.001) and the motor (F=61.58, P<0.001). When compared to the control group C (204±11.47 and 157±10.11), the regression time of block, both sensory up to T10 dermatome and motor to bromage 3 scale, was delayed in the DXM group D (361±12.85 and 336±11.44) and the Mg group M (275±11.74 and 252±10.26). However, out of the three groups, the DXM group's duration was the longest. One-way ANOVA with post tests revealed statistically significant differences between the groups in both the sensory (F=59.88, P<0.001) and the motor (F=174, P<0.001). Conclusion: In contrast to intrathecal Mg, intrathecal DXM augmentation of spinal block seems to be a promising option since it causes sensory and motor block to begin and last longer while causing no appreciable hemodynamic changes. In lengthy surgical operations, ten micrograms of DXM as an adjuvant to spinal bupivacaine produces low adverse effects and offers good postoperative analgesia.

Keywords: Intrathecal dexmedetomedine, magnesium sulfate, bupivacaine

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INTRODUCTION

Local, regional (spinal or epidural), or general anaesthesia may be used for total hip replacement procedures, although neuraxial blockade is the recommended kind of anaesthesia. Spinal anaesthesia, also known as a sub-arachnoid block, was developed by J. Leonard Corning in 1885 and is now the most adaptable and widely used regional block globally. It is most often used as a central block in surgical settings.[1] The spinal approach is simple to use and has a very high percentage of success. It has been shown that spinal anaesthesia may reduce

intraoperative blood loss, the stress response to surgery, and the likelihood of postoperative thromboembolic events.[4] It may be used to prolong analgesia into the post-operative period, when it has been shown that doing so results in greater analgesia than parenteral opioids can. Studies have indicated that total blood loss during total hip replacement procedures performed under spinal anaesthetic is reduced by 30% to 50%.[5] The main benefit of localised anaesthesia is most obvious after surgery. The need for opioids is decreased because to residual block, which shields the patient from the first round of

postoperative pain.[6] In a comprehensive patient care plan for an anaesthesia, post-operative pain control is a critical issue to be focused on in total hip replacement. A intrathecal adjuvant to these local anaesthetic agents is necessary for a fully satisfying duration of anaesthesia and patient satisfaction due to the limitations of hyperbaric bupivacaine and ropivacaine intrathecaly for sub arachnoid block, including their prolonged onset of sensory and motor blockage effect, short duration of action, and early postoperative requirement for an analgesic agent. Adjuvants to local anaesthetics are studied, including opioids (morphine, fentanyl, and sufentanyl) and other medications such dexmedetomidine (DXM), clonidine, magnesium sulphate (MgSO4), neostigmine, ketamine, and midazolam. The role of magnesium sulphate as an adjuvant to local anaesthetics in spinal anaesthesia is based on magnesium's potential anti-nociceptive effect through its voltage-dependent regulation of calcium influx into the cell and non-competitive antagonistic activity against N-methyl-D-aspartate (NMDA) receptors, which has the potential to prevent central sensitization from peripheral nociceptive stimulation. Dexmedetomidine is a sedative and co-analgesic administered intravenously. It enhances the impact of local anaesthetics, enables a lower dosage without depressing the respiratory system or causing hemodynamic instability, and is used to extend the duration of sensory, motor blockade, and analgesic effect.[7]

MATERIALS AND METHODS

This prospective, randomized, double-blinded study enrolled 90 ASA physical status I and II patients scheduled for total hip replacement surgery under spinal anaesthesia, aged 35 to 75 years, of either gender, height 159 to 189 cm, and weight 52 to 88 kg after receiving written informed consent and institutional ethical committee approval. Exclusion criteria for the trial included individuals having a history of uncontrolled, labile hypertension, allergies to the study medicines, opium addiction, sedative drug use, contraindications for spinal anaesthesia, failed spinal blocks, and the requirement for general **Patients** were anaesthesia. not given premedication, and upon entering the operation room, noninvasive blood pressure (NIBP), ECG, and pulse oximetry (SpO2) were all monitored. Following infusion of 500 mL lactated Ringer's solution and with the patient in the sitting position, lumbar puncture was performed at the L3-L4 level through a midline approach using a 25G Quincke spinal needle (Spinocan, B Braun Medical, Melsungen, Germany). Using computer-generated random numbers, patients were allocated into three groups:

- 1. Group D received 15 mg hyperbaric bupivacaine and 0.1 ml (10 μ g) DXM
- 2. Group M received 15 mg hyperbaric bupivacaine and 0.1 ml (50 mg) Mg

3. Group C received 15 mg hyperbaric bupivacaine and 0.1 ml normal saline as control

Patients were placed in the supine posture after receiving an intrathecal injection, and 2 L/min of oxygen was administered via a face mask. The intraoperative data were documented by the anesthesiologist providing the block while keeping them blind to the study medication. By utilising analgesia to pin prick with a small hypodermic needle in the midclavicular line, sensory block was evaluated bilaterally. The modified Bromage scale was used to evaluate motor blockade[8]. Prior to surgery, peak sensory level, Bromage 3 motor block, and time to attain T10 dermatome sensory block were all noted. In a postanesthesia care unit (PACU), the regression time for sensory and motor block was noted. Calculations for all times used the timing of the spinal injection as the starting point. After sensory regression to the S1 dermatome and Bromage 0, patients were released from the PACU. Heart rate, NIBP, and SpO2 levels in the three groups were monitored before to surgery, during surgery, and while they were shifting. Systolic blood pressure 90 mmHg or a >30% drop from baseline readings was considered hypotension. The definitions of tachycardia and bradycardia were heart rates more than 100 and 60, respectively. Any adverse effects during the procedure, including nausea, vomiting, pruritus, sedation, and additive analgesia, were noted.

RESULTS

Age, height, body weight, and body mass index (BMI) did not vary across the groups. The greatest dermatome height attained was comparable across these groups. There was no discernible difference between the groups in terms of oxygen saturation, heart rate, systolic or diastolic arterial blood pressure, or heart rate variability.

Table 1: Onset times of sensory blocks for sample groups

| Group | Mean±Sd | P value |
|---------------------|-----------|---------|
| Dexmedetomidine (D) | 3.01±0.98 | 0.001 |
| Magnesium (M) | 7.11±1.36 | |
| Control (C) | 5.01±1.36 | |

Table 2: Onset times of motor blocks for sample groups

| Group | Mean±Sd | P value |
|---------------------|-----------|---------|
| Dexmedetomidine (D) | 4.11±1.04 | 0.001 |
| Magnesium (M) | 7.82±1.55 | |
| Control (C) | 5.36±1.21 | |

Table 3: Regression times of sensory blocks for sample groups

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|---------------------|-----------|---------|
| Group | Mean±Sd | P value |
| Dexmedetomidine (D) | 361±12.85 | 0.001 |
| Magnesium (M) | 275±11.74 | |
| Control (C) | 204±11.47 | |

Table 4: Regression times of motor blocks for sample groups

| Group | Mean±Sd | P value |
|---------------------|-----------|---------|
| Dexmedetomidine (D) | 336±11.44 | 0.001 |
| Magnesium (M) | 252±10.26 | |
| Control (C) | 157±10.11 | |

In compared to the control group C (5.01±1.36 and 5.36±1.21), the start time of block, both sensory up to T10 dermatome and motor to Bromage 3 scale, was quick in the DXM group D (3.01±0.98 and 4.11±1.04) and delayed in the Mg group M (7.11± 1.36 and 7.82± 1.55). One-way ANOVA with post tests revealed statistically significant differences between the groups in both the sensory (F=94.33, P<0.001) and the motor (F=61.58, P<0.001). When compared to the control group C (204 ± 11.47 and 157 ± 10.11), the regression time of block, both sensory up to T10 dermatome and motor to bromage 3 scale, was delayed in the DXM group D (361±12.85 and 336±11.44) and the Mg group M (275±11.74 and 252±10.26). However, out of the three groups, the DXM group's duration was the longest. One-way ANOVA with post tests revealed statistically significant differences between the groups in both the sensory (F=59.88, P<0.001) and the motor (F=174, P<0.001). In the first hour after the administration of the spinal anaesthesia and in the first hour in the PACU, there was no discernible difference between the three groups in the mean values of heart rate and mean arterial pressure. All patients in the three groups had SpO 2 levels that were greater than 95%, whether during surgery or while in the PACU. Follow-up tests performed 24 hours and 2 weeks after discharge revealed no neurological deficits symptoms of spinal anesthesia-related back, buttock, or leg discomfort.

DISCUSSION

When administered intravenously during anaesthesia, DXM lowers the need for opioid and inhalational anaesthetics.[9] The affinity of DXM to a-2 receptors has been shown to be ten times greater than that of clonidine.Kalso et al.[11] Kanazi et al.[12] discovered that in urologic surgical patients, the duration of sensory and motor block was similarly extended with little adverse effects when 3 g DXM or 30 g clonidine were administered to 13 mg spinal bupivacaine. By inhibiting the release of C-fiber transmitters and causing post-synaptic dorsal horn neurons to become hyperpolarized, intrathecal DXM when paired with spinal bupivacaine prolongs the sensory block.[13] A-2 adrenoreceptor agonists may cause motor block prolongation by binding to motor neurons in the dorsal horn of the spinal cord.[14] A-2 receptor agonists that are injected intravenously have antinociceptive effects on both somatic and visceral pain.[15] In their investigation, Kanazi et al.[12] discovered that when bupivacaine (12 mg) spinal block is combined with a low-dose DXM (3 g), the result is a substantially longer sensory and motor block than when bupivacaine is used alone. The findings of Al-Mustafa et al.[16] and Al-Ghanem et al.[17] employed greater dosages of DXM (5 g and 10 g), and they discovered that its impact is dose-dependent and that using DXM shortened the time it took for sensory block to begin at the T10 dermatome. Similar results were obtained in our investigation. A greater volume of DXM was injected into the subarachnoid area at a higher dosage. Bradycardia and hypotension are the two main adverse effects associated with the usage of intrathecal a-2 adrenoreceptor agonists. These adverse effects were not substantial in the current trial, most likely because we administered the medication intrathecally rather than intravenously. However, it was shown that in the Mg group, both the start and resolution of motor blockage as well as the duration to reach the maximal sensory level were slower. When Mg was added intravenously in addition to fentanyl and isobaric bupivacaine (we used hyperbaric bupivacaine in our trial), Ozalevli et al. noticed a comparable delay in the onset of spinal anaesthesia.[18] They hypothesised that the pH and baricity of the solution containing Mg were different, which may have contributed to the delayed onset in the research by Malleeswaran et al. on patients with mild preeclampsia.[19] Although less than with intrathecal DXM, there was a prolonging of the motor and sensory block in our investigation. A further finding made by Arcioni et al. was that intrathecal and epidural Mg extended and potentiated the motor block.[20] These findings are in line with a prior research in which the duration of spinal anaesthesia was extended by adding intrathecal magnesium (50 mg) to bupivacaine (10 mg) and fentanyl (50 g) during lower extremity surgery while the patients were under spinal anaesthesia.[21] They showed that adding 50 mg of intrathecal Mg to intrathecal fentanyl improved analgesia during painless delivery. Intrathecal Mg was utilised to lengthen the analgesic duration of opioids in people. These outcomes were analogous to those of animal trials, in which intrathecal Mg lengthened the opioids' analgesic duration.[22]

Magnesium significantly reduces NMDA-induced currents and inhibits NMDA channels in a voltagedependent manner.[23] Neurotransmitters glutamate and aspartate are released in response to noxious stimulus, and they bind to the NMDA receptor. When these receptors are activated, calcium enters the cell and starts a sequence of processes that make cells more sensitive to sustained stimulation, including spinal cord processes called wind-up and long-term potentiation. The length of acute pain may be influenced by **NMDA** receptor signalling [24,25]. Calcium influx is blocked by magnesium, and NMDA receptor channels are noncompetitively antagonised.[26]

CONCLUSION

In contrast to intrathecal Mg, intrathecal DXM augmentation of spinal block seems to be a promising

option since it causes sensory and motor block to begin and last longer while causing no appreciable hemodynamic changes. In lengthy surgical operations, ten micrograms of DXM as an adjuvant to spinal bupivacaine produces low adverse effects and offers good postoperative analgesia. The duration of spinal analgesia is similarly prolonged by intrathecal Mg, but less so than by intrathecal DXM and with a later start.

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