To Compare the Effects of Intrathecal Dexmedetomidine versus Magnesium Sulphate as Adjuvants to Hyperbaric Bupivacaine for Lower Limb Orthopedic Surgeries

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INTRODUCTION: Various intrathecal adjuvants are used for analgesia, sedation and prolongation of subarachnoid block. AIM: The present study was undertaken with the aim to compare the onset and duration of sensory and motor block, maximum sensory level, bromage grade achieved and duration of analgesia induced by dexmedetomidine and magnesium sulfate along with 0.5% hyperbaric bupivacaine. Materials and Methods: After obtaining institutional ethics committee approval, written informed consent, 60 patients of ASA I and II, age 20-60 years, scheduled for elective surgeries were randomly assigned into two groups of 30 each. Group D received 3ml(15mg) of hyperbaric bupivacaine +0.1ml(10ug) of dexmedetomidine. Group M received 3ml(15mg) of hyperbaric bupivacaine +0.1ml(50mg) of magnesium sulfate. The characteristics of blockage and postoperative analgesia were noted. Statistical analysis done with graphpad.com software using student ‘t’ test and chi square test. Data expressed as mean (+/-) SD. P<0.05 significant. Results: Demographic data was similar and comparable between two groups. Onset time for sensory and motor block was earlier and the mean duration of analgesia was higher in group D than group M. Conclusion: We conclude that intrathecal dexmedetomidine is more effective in producing early onset and prolonged duration of sensory and motor blockade, with minimum side effects in comparison with intrathecal magnesium sulfate.

Key Words: Bupivacaine, Dexmedetomidine, Magnesium sulfate, Spinal anaesthesia.
Dexmedetomidine, a second-generation alpha 2 adrenergic receptor-specific, pharmacologically active δ isomer of medetomidine. It produces a unique type of sedation analgesia with less ventilatory depression than most common used sedative-hypnotics. It produces analgesia by the central, spinal, and peripheral mechanisms.

Supra spinal level of analgesia and sedation is due to modulation of noradrenergic pathways in the noradrenergic nucleus or locus ceruleus.

Spinal level of antinociception is through substantia gelatinosa. It closes the dorsal horn to the stimulus of Adelta and C fibers, also blocking the release of nociceptive humoral transmitters like substantia gelatinosa. It produces hyperpolarisation of cell membrane causing suppression both neuronal firing and release of noradrenaline at nerve terminals. This antinociceptive effect explains sensory block when given in subarachnoid space.

Peripheral activity is due to activation of postsynaptic alpha 2 adrenoceptors. Activation of alpha 2 receptors leads to a dose-dependent reduction in the level of plasma catecholamine, bradycardia, and hypotension secondary to sympathetic inhibition of medial vasomotor center in a dose-dependent manner.

Magnesium is second abundant intracellular cell cation and 4th abundant cation in the body. The effect of its nociception is primarily based on the regulation of calcium influx into cell, i.e., natural physiological calcium antagonism and inhibit NMDA receptor blockers in a non-competitive way and prevents central sensitization from peripheral nociceptive stimulation. Magnesium accentuates neuromuscular blockers by itself and potentiates the neuromuscular blockade of both NMDR and DMRs.

IV. Materials And Methods

After obtaining informed consent and the institutional ethics committee approval, this study was conducted in Rangaraya medical college, Kakinada. The study included 50 patients of ASA grade I and II aged 20-60 years of either gender, height 150cms and above, and weight 50-90 kgs, scheduled for elective lower limb surgeries under spinal anesthesia. Patients with a history of hypertension, allergic to study drug, opioid addiction, sedative drug consumption, contraindicated for spinal anesthesia, failure of the spinal block and the need for GA were excluded from the study. They were randomly assigned according to the table of randomization into two groups of 25 each. Group D and Group M. Patients in Group D were given 3ml(15mg) of hyperbaric bupivacaine + 0.1 mg (10mcg) of dexmedetomidine. Patients in Group M were given 3ml(15mg) of hyperbaric bupivacaine + 0.1ml (50mg) of MgSO4. The complete pre-anesthetic check-up was done a day before surgery. All relevant investigations were done, and the patient was kept overnight fasting. Tab Pantop 40mg and Tab. Alprazolam 0.25mg was advised at bedtime on night before surgery.

In the recovery room, an appropriate IV cannula was secured and given 10ml/kg of Ringer's lactate before surgery. The noninvasive monitors were attached to the patient, and baseline vitals were noted. The patient was placed in a sitting position under aseptic precautions. The subarachnoid block is given using 25G Quincke spinal needle in L3-L4 intervertebral space. The anesthetist performing the block was blinded to study drug and recorded the intraoperative data. The following observations were made. The onset of sensory block assessed by pinprick sensation every 2 minutes until no sensation(grade 2) was achieved. (graded according to Gromley and Hill 1996.)0-normal sensation,1-blunted sensation,2-no sensation. Grade 2 was taken as the onset of sensory blockade.onset of motor block was assessed every 2 minutes till complete motor block grade 3 was achieved. (graded according to Modified Bromage scale.) Grade 0-no motor block, Grade 1-inability to raise extended legs, Grade 2-inability to flex knees, Grade 3-inability to flex ankle joints, and grade 3 was taken as a complete motor block. Time to reach the T10 dermatomal sensory level, peak sensory elevl, and Bromage 3 motor block were recorded before surgery. Regression time for sensory and motor block was recorded in PACU. All durations were calculated, assuming the time of spinal injection as time zero. Patients were discharged from PACU after sensory regression to S1 dermatoome and Bromage grade 0. Intraoperative noninvasive monitoring of vitals(HR, SBP, DBP &SpO2) was done every 2 minutes for 1st 10 minutes. Every 5 minutes for the next 15 minutes and every 15 minutes thereafter till completion of surgery. Side effects-hypotension(decrease in SBP to <90mm hg or decrease in MAP of >20% from baseline). Bradycardia (fall in HR >20% from baseline), nausea, vomiting, headache were recorded. Any additive analgesia was also recorded.
To Compare the Effects of Intrathecal Dexmedetomidine versus Magnesium Sulphate as Adjuvants..

V. Results

STATISTICAL ANALYSIS:
1. P value <0.05 was considered statistically significant.
2. Quantitative data (age, height, weight, hemodynamic parameters after spinal anesthesia, onset time of sensory and motor blockade, duration of sensory and motor blockade, time to reach maximum sensory blockade) was analyzed using student T-test.
3. Qualitative data (sex) was analyzed using the Chi-square test.

There was no significant difference in demographics data in 2 groups, P-value >0.05 (Tab1). The onset of sensory and motor blockade was rapid in group D in comparison with group M (Tab 2) (Fig 1), the results were statistically significant, with a P-value <0.05. The time to attain maximum sensory level is shorter in group D in comparison with group M with significant P-value < 0.05. Duration of sensory and motor blockade is higher in group D with statistically significant p-value 0.0001 (Fig 2).

**TABLE 1**

<table>
<thead>
<tr>
<th></th>
<th>Group D(n=30)</th>
<th>Group M(n=30)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age(years)</td>
<td>35.20±6.95</td>
<td>35.83±7.91</td>
<td>0.744</td>
</tr>
<tr>
<td>Height/cms</td>
<td>169.60±5.5</td>
<td>169.30±6.00</td>
<td>0.840</td>
</tr>
<tr>
<td>Weight(kgs)</td>
<td>39.0±8.7</td>
<td>38.6±9.4</td>
<td>0.670</td>
</tr>
<tr>
<td>Sex(M:F)</td>
<td>26:4</td>
<td>28:2</td>
<td>0.670</td>
</tr>
<tr>
<td>ASA (I / II)</td>
<td>26:4</td>
<td>27:3</td>
<td>0.706</td>
</tr>
<tr>
<td>Duration of surgery(minutes)</td>
<td>124.08±33.70</td>
<td>118.36±36.51</td>
<td>0.330</td>
</tr>
</tbody>
</table>

**TABLE 2**

<table>
<thead>
<tr>
<th>Outcomes</th>
<th>Group D</th>
<th>Group M</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset time of sensory block(minutes)</td>
<td>3.50±0.44</td>
<td>4.16±0.96</td>
<td>0.001</td>
</tr>
<tr>
<td>Onset time of motor block(minutes)</td>
<td>6.47±0.48</td>
<td>7.28±1.23</td>
<td>0.001</td>
</tr>
<tr>
<td>Time to attain maximum sensory level(minutes)</td>
<td>10.20±2.70</td>
<td>12.84±3.33</td>
<td>0.001</td>
</tr>
<tr>
<td>Duration of sensory block(minutes)</td>
<td>146.7±21.5</td>
<td>119.5±19.01</td>
<td>0.0001</td>
</tr>
<tr>
<td>Duration of motor block(minutes)</td>
<td>196.01±24.6</td>
<td>161.4±19.8</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

Fig1
VI. Discussion

In recent years intrathecal adjuvants gained popularity with the aim of prolonging the duration of the block, decreased resource utilization compared with GA, and faster recovery. This study aims to compare the effects of Dexmedetomidine and MgSO4.

No significant differences were found among age, height, weight, sex, ASA grade I and II and duration of surgery among two groups with P-value >0.05, which is insignificant.

Onset time of sensory blockade in group D (3.50±0.44) minutes compared to Group M.

AL-Mustafa et al. (1) and al-Ghanem (2) used dexmedetomidine 5mcg, and 10mcg added to 12.5mg hyperbaric bupivacaine found its effect in a dose-dependent manner and that the onset of sensory block to reach T10 dermatome level was shorter with use of dexmedetomidine.

Onset of motor blockade was rapid (6.47±0.48) minutes and duration of motor blockade was (96.01±24.6 minutes) higher in group D at the time of onset of motor block.

Similar results were found by Shukla et al. (3) in their study in which they found that the onset of a motor blockade is rapid and of prolonged duration in group D.

Level of the sensory block on an average was high in group D compared to group M.

Ozaleni et al. (4) found that with the addition of MgSO4 to bupivacaine and fentanyl during subarachnoid blockade using 25mcg fentanyl with 50mg MgSO4 onset of both sensory and motor blockade was delayed but the duration was prolonged without significant side effects.

Intrathecal dexmedetomidine when compared with spinal bupivacaine prolonged sensory blockade by depressing the relation of c fiber transmitters and by hyperpolarisation of postsynaptic dorsal horn neurons (5).

Kanazi et al. (6) found in their study that the supplementation of bupivacaine 12mg spinal block with a low dose of dexmedetomidine 3mcg produces significant shorter onset of motor blockade and a significantly longer sensory and motor block than bupivacaine alone.
In the case study by Malleswaran et al. (7) on mild pre-eclampsia patients shows that MgSO4 contributed to delayed onset suggested that the difference in pH and baricity of solution containing MgSO4 contributed to late-onset.

Arcioni et al. (8) also observed that intrathecal and epidural Magnesium potentiates and prolongs motor block. In our study also there was a prolongation of motor and sensory block, although less than that with intrathecal dexmedetomidine.

Buvanendran et al. (9) study conducted in patients undergoing lower extremity surgery during spinal anesthesia, in which addition of intrathecal Mg (50mg) to 10mg bupivacaine + 50mcg fentanyl prolongs period of spinal anesthesia.

Magnesium blocks NMDA channels in a voltage-dependent way and produces a dramatic reduction of NMDA induced currents(10).

VII. Conclusion

Intrathecal dexmedetomidine supplementation to spinal anesthesia is an excellent alternative to intrathecal mgso4 as it produces earlier onset and prolonged duration of sensory and motor blockade without associated significant hemodynamic changes.10mcg of dexmedetomidine as an adjuvant to bupivacaine in spinal anesthesia in lower limb orthopedic surgeries of the long period has minimum side effects with excellent postoperative analgesia. Intrathecal Magnesium Sulphate8 also prolongs the duration of spinal analgesia but less than intrathecal dexmedetomidine and is with a delayed onset.

References