

**EFFECT OF DEXMEDETOMIDINE ADDED TO SPINAL BUPIVACAINE FOR TOTAL ABDOMINAL HYSTERECTOMY**C. N. Vinod<sup>1</sup>, Narendra Babu<sup>2</sup>, Dwideep Chandra<sup>3</sup>**HOW TO CITE THIS ARTICLE:**

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**ABSTRACT: BACKGROUND:** Various adjuvants are being used with local anaesthetics in spinal anaesthesia for prolongation of intraoperative and post-operative analgesia. Dexmedetomidine, a highly selective  $\alpha_2$  adrenergic agonist is a new neuroaxial adjuvant gaining popularity. **AIMS:** To evaluate the onset and duration of sensory and motor block, hemodynamic effect, post-operative analgesia and adverse effects of dexmedetomidine given intrathecally with hyperbaric 0.5% bupivacaine. **METHODOLOGY:** A study was carried out in 30 adult female patients aged 18-55 yrs of ASA grade I and II in each group scheduled for Total abdominal hysterectomy under spinal anaesthesia. Group B received 2.5ml of 0.5% hyperbaric bupivacaine with 0.5ml of normal saline. Group D received 2.5ml of 0.5% hyperbaric bupivacaine with 10 $\mu$ g of dexmedetomidine in 0.5ml of normal saline. The onset time to reach peak sensory and motor level, regression time of sensory and motor block, rescue analgesia, hemodynamic changes and side effects were recorded. **STATISTICAL ANALYSIS USED:** Data obtained were tabulated and analyzed using statistical package for social science (SPSS 16.0 evaluation version) to calculate the sample size. Descriptive data are presented as Mean  $\pm$  SD and Continuous data are analyzed by unpaired 't' test.  $P < 0.05$  was considered statistically significant. **RESULTS:** Patients in dexmedetomidine group (group D) had a significantly longer sensory and motor block than patients in bupivacaine group (group B). The mean time of sensory regression to S1 was (323  $\pm$  31 min) in group D and (191  $\pm$  15min) in group B. The regression time of motor block to reach Bromage 0 was (314  $\pm$  30 min) in group D and (163  $\pm$  15 min) in group B. The time to rescue analgesia was significantly longer in group D (383  $\pm$  38 min) as compared to group B (228.6  $\pm$  15 min). **CONCLUSION:** Intrathecal dexmedetomidine as adjuvant to spinal bupivacaine is associated with prolonged sensory and motor block, hemodynamic stability and reduced demand of rescue analgesia in twenty four hours.

**KEYWORDS:** Bupivacaine, dexmedetomidine, spinal anaesthesia.

**INTRODUCTION:** Subarachnoid blockade is the most commonly used regional anaesthetic technique for lower abdominal surgery. Spinal block is easy to perform, economical and produces rapid onset of anaesthesia. One of the main disadvantages of spinal anaesthesia is its limited duration of action and hence lack of post-op analgesia.

A number of adjuvants such as clonidine, fentanyl and others have been studied to prolong the effect of spinal anaesthesia.<sup>1,2</sup> Dexmedetomidine, a highly selective  $\alpha_2$  adrenergic agonist, as an adjuvant to hyperbaric bupivacaine in spinal anaesthesia provides good quality of intraoperative and prolonged post-operative analgesia with minimal side effects.<sup>3,4,5</sup>

**MATERIALS AND METHODS:** After obtaining institutional ethical committee approval and written informed consent, 60 adult female patients belonging to ASA class I and II aged 18-55years scheduled

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for total abdominal hysterectomy under spinal anaesthesia were enrolled in this prospective randomized study. Study was carried out at Kempegowda Institute of Medical Science and Hospital, Bangalore from Aug 2013 to July 2014. Patients were randomly divided into two groups with 30 patients in each group. Patients with h/o uncontrolled hypertension, allergy to the study drug, heart block/dysrhythmias, contraindication for spinal anaesthesia and failure of spinal block were excluded from the study.

All patients were examined and investigated a day prior to surgery and were familiarized with visual analogue scale (VAS).<sup>6</sup> They were advised fasting for 6 hours and received alprazolam 0.5mg the night before surgery.

In the operation theatre, pulse oximetry, electro cardiogram and noninvasive blood pressure were attached and baseline parameters were recorded and monitoring was initiated.

Intravenous access was secured and all patients were preloaded with ringer lactate 10ml/kg. Under all aseptic precautions patients in sitting position lumbar puncture was performed at L<sub>3</sub>-L<sub>4</sub> interspace using 26G Quincke spinal needle. Patients were randomly divided into 2 groups with 30 patients in each group.

**Group B:** Received 2.5ml volume of 0.5% hyperbaric bupivacaine and 0.5ml of normal saline.

**Group D:** Received 2.5ml volume of 0.5% hyperbaric bupivacaine and 10µg of dexmedetomidine in 0.5ml of normal saline.

Patients were made supine following the block and oxygen 5l/min were given through a face mask. The onset and the duration of sensory block, highest level of sensory block, time to reach the highest dermatome level of sensory block, motor block onset, time to complete motor block recovery and duration of spinal anaesthesia were recorded.

The onset of sensory block was defined as the time between injection of drug and the absence of pain at T10 dermatome assessed by sterile pinprick every 2 min till T10. On achieving T7 sensory blockade surgery were allowed. Then testing were conducted every 10min until the point of 2 segment regression of the block was observed. The motor level was assessed according to modified Bromage scale:

**Grade 0:** The patient is able to move hip knee and ankle.

**Grade 1:** Patient is unable to move the hip, but is able to move the knee and ankle.

**Grade 2:** Patient is unable to move the hip and knee, but is able to move the ankle.

**Grade 3:** Patient is unable to move the hip, knee and ankle.

Vitals were recorded at 1, 2, 3, 4, 5, 10, 15, 20, 25, 30 min and subsequently every 30min. Hypotension defined as a decrease of systolic blood pressure more than 30% from baseline and was treated with IV bolus of 6mg Ephedrine and IV fluids as required. Bradycardia defined as heart rate <50bpm was treated with IV atropine 0.6mg. The incidence of nausea and vomiting and sedation were recorded. Sedation was assessed by modified Ramsay sedations scale:

1. Patient anxious and agitated or restless.
2. Patient co-operative oriented and tranquil.
3. Responds to verbal commands while sleeping.
4. Exhibits brisk response to light glabellar tap or loud noise while sleeping.
5. Sluggish response to light glabellar tap or loud noise while sleeping.
6. No response to light glabellar tap or loud noise while sleeping.

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Post operatively the regression time for sensory and motor block were recorded in a post anaesthesia care unit along with the vital signs and Visual analogue scale (VAS) scores. Any patients showing VAS more than or equal to 3 was given diclofenac intramuscularly as rescue analgesia. All duration were calculated considering the time of spinal injection as time zero. Patients were discharged from PACU after sensory regression to S<sub>1</sub> dermatome and Bromage 0.

Data obtained were tabulated and analyzed using statistical package for social science (SPSS 16.0 evaluation version) to calculate the sample size. Descriptive data are presented as Mean  $\pm$  SD and Continuous data are analyzed by paired/unpaired 't' tests. The comparison was studied using the chi square test or fisher's test as appropriate with a p value reported at the 95% confidence interval. P<0.05 was considered statistically significant.

**RESULTS:** The groups were comparable with respect to age, height, weight and ASA physical status (Table1). The characteristics of block and regression time are summarized in (Table2).

| Variables   |       | Group B | Group D |
|-------------|-------|---------|---------|
| Age (years) | Mean  | 43.37   | 41.93   |
|             | SD    | 5.149   | 7.643   |
|             | Range | 30-56   | 28-60   |
| Height (cm) | Mean  | 159.9   | 160.7   |
|             | SD    | 5.252   | 5.563   |
|             | Range | 146-170 | 148-170 |
| Weight (kg) | Mean  | 60.40   | 61.13   |
|             | SD    | 7.587   | 10.187  |
|             | Range | 43-75   | 43-90   |

**Table 1: Demography**

| Variables                                    | Group B<br>(Mean $\pm$ SD) | Group D<br>(Mean $\pm$ SD) | p-value |
|----------------------------------------------|----------------------------|----------------------------|---------|
| Onset of sensory block (min)                 | 6.30 $\pm$ 1.0             | 2.50 $\pm$ 0.7             | <0.001  |
| Onset of motor block to Bromage -3 (min)     | 10.67 $\pm$ 1.0            | 7.40 $\pm$ 1.3             | <0.001  |
| Time from injection to highest sensory level | 12.53 $\pm$ 1.2            | 10.97 $\pm$ 1.2            | <0.001  |
| Time for two segment regression              | 87.33 $\pm$ 10.4           | 111.33 $\pm$ 20.8          | <0.001  |
| Time for sensory regression to S1 segment    | 191.0 $\pm$ 15.1           | 323.33 $\pm$ 31.4          | <0.001  |
| Time for motor block regression to Bromage 0 | 163.67 $\pm$ 15.1          | 314.0 $\pm$ 30.1           | <0.001  |
| Rescue analgesia (min)                       | 228.67 $\pm$ 15.2          | 383.67 $\pm$ 38.9          | <0.001  |

**Table 2: Characteristics of Block**

The onset time of block, both sensory up to T10 dermatome and motor to Bromage 3 scale was rapid in the group D (2.50  $\pm$  0.7 min and 7.40  $\pm$  1.3 min) in comparison with group B (6.30  $\pm$  1.0 min and 10.67 $\pm$  1.0 min) p-value <0.05. There was no difference between group D and B in the highest level of block achieved.

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Block regression was significantly slower with the addition of intrathecal dexmedetomidine (group D) as compared with group B, as both time to two segment regressions and time to S1 segment regression were significantly slower with intrathecal dexmedetomidine. P-value <0.05. The regression of motor block to bromage 0 was significantly slower in group D ( $314 \pm 30$  min) compare to group B ( $163 \pm 15.1$  min) p-value <0.05. The time to rescue analgesia was significantly longer in group D ( $383 \pm 38$  min) as compared to group B ( $228.6 \pm 15$  min).

There was no significant difference in the mean value of heart rate and mean arterial pressure in the first hour after performing the spinal anaesthesia and the first hour in the PACU between the two groups the sedation score was more in group D patients ( $3.4 \pm 0.3$ ) which was statistically significant. The SPO<sub>2</sub> was higher than 95% in all patients either in the intraoperative or in the PACU time. 24 hours and 2 weeks follow up did not show neurological impairment related to spinal anaesthesia such as back, buttock or leg pain, headache or any neurological deficit.

**DISCUSSION:** Different agents such as magnesium sulphate, phenylephrine, clonidine has been used as adjuvant for prolonging the duration of spinal anaesthesia. Kanazi et al<sup>7</sup> found in their study that supplementation of bupivacaine (12mg) spinal block with a low dose of dexmedetomidine (3µg) produces a significantly shorter onset of motor block and a significantly longer sensory and motor block than bupivacaine alone as found in our study also.

Dexmedetomidine is an  $\alpha_2$  adreno receptor agonist which has 10 times higher affinity for  $\alpha_2$  adreno receptor than clonidine.<sup>8,9,10</sup> Intrathecal dexmedetomidine when combined with spinal bupivacaine prolongs the sensory block by depressing the release of C-fiber transmitters by hyper polarization of post synaptic dorsal horn neurons.<sup>11</sup>

Motor block prolongation by  $\alpha_2$  adreno receptor agonist may result from binding these agonists to motor neuron in the dorsal horn of the spinal cord.<sup>12</sup> Intrathecal  $\alpha_2$  adreno receptor agonist have antinociceptive action for both somatic and visceral pain.<sup>13</sup> A number of animal studies conducted using intrathecal dexmedetomidine at a dose range of 2.5 – 100 µg did not report any neurological deficit with its use.<sup>14-17</sup>

Al-Mustafa et al<sup>5</sup> studied effect of dexmedetomidine 5 and 10 µg with bupivacaine in urological procedures and found that dexmedetomidine prolongs the duration of spinal anaesthesia in the dose dependent manner. We had the same result in our study.

Rajni Gupta et al<sup>18</sup> studied the effect of adding dexmedetomidine v/s fentanyl to intrathecal bupivacaine and concluded that dexmedetomidine produces more prolonged motor and sensory block as compared with fentanyl. In our study in the dexmedetomidine group we found longer duration of both sensory and motor blockade, stable hemodynamic conditions and good patients satisfaction.

The most significant side effect reported about the use of intrathecal  $\alpha_2$  adreno receptor agonist are bradycardia and hypotension. In our study hypotension was more in the dexmedetomidine group than in the bupivacaine group, but it was not statistically significant.

Hala EA et al<sup>19</sup> observed dose dependent prolongation of motor and sensory blockade with intrathecal dexmedetomidine and desirable sedation level may be beneficial in patients undergoing lengthy complex surgery. We had Ramsay sedation score of three in the dexmedetomidine group compared to bupivacaine group and it did not affect the level of consciousness.

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**CONCLUSION:** Intrathecal dexmedetomidine supplementation of spinal block produces earlier onset and prolonged duration of sensory and motor block without associated significant hemodynamic alterations. 10 µg of dexmedetomidine as adjuvant to spinal bupivacaine in Total abdominal hysterectomy (TAH) has minimal side effect and provides excellent quality of post-operative analgesia.

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