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To Compare the Effects of Three Different Doses of Dexmedetomidine as an Adjuvant to Spinal Anaesthesia in Patients Undergoing Infraumbilical Surgeries: A Prospective, Randomized, Double-blind Clinical Study

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Abstract

Background: Spinal anesthesia is preferred technique of choice in infraumbilical surgeries. Limitation of this technique is shorter duration of analgesia, so various adjuvants have been used with intrathecal bupivacaine such as fentanyl, clonidine, and dexmedetomidine. Dexmedetomidine is a highly selective alpha 2 adrenergic agonist. The aim of our study was to know the effect of different doses of dexmedetomidine on intrathecal bupivacaine. **Subjects and Methods:** The prospective, randomized, double-blind study was conducted in tertiary health care center, on ninety patients of the American Society of Anesthesiology Class I and II, of age group 18–60 years of either sex. They were randomly allocated into three groups. Group BD5 (n = 30): intrathecal bupivacaine 12.5 mg (2.5 ml) + dexmedetomidine 5 μg (0.5 ml), Group BD10 (n = 30): intrathecal bupivacaine 12.5 mg (2.5 ml) + dexmedetomidine 10 μg (0.5 ml), Group BD15 (n = 30): intrathecal bupivacaine 12.5 mg (2.5 ml) + dexmedetomidine 15 μg (0.5 ml) administered intarthecally. The onset and maximum level of sensory block, time to reach maximum level of sensory block, time of two-segment sensory regression, the total duration analgesia, time of rescue analgesia, onset and duration of motor block and heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure, respiratory rate, and oxygen saturation were recorded at various intervals. Moreover, any adverse effects such as bradycardia, hypotension, nausea, vomiting, and sedation were recorded. **Results:** The onset time of sensory block in Group D5-2.76 $\pm$ 1.32, Group D10-2.45 $\pm$ 1.50, and Group D15-1.86$\pm$0.93, which is statistically significant (P = 0.025). The time taken for two-segment sensory regression Group D5-96.66 $\pm$ 33.67, Group D10-116.80 $\pm$ 36.27, and Group D15 120.96 $\pm$ 30.24, (P = 0.014). The time taken for complete sensory recovery in Group D5-319.83 $\pm$ 61.41, Group D10-336.13 $\pm$ 61.38, and Group D15-415.20 $\pm$ 96.6, which is statistically highly significant (P = 0.000). Time for rescue analgesia in Group D5-377.46 $\pm$ 60.05, in Group D10-401.60 $\pm$ 61.11, and in Group D15-517.96 $\pm$ 97.30, which is statistically highly significant (P < 0.000). **Conclusion:** We concluded that there was decrease in onset of sensory and motor blockade with the prolongation of duration of anesthesia and analgesia in a dose-dependent manner.

Keywords: Bupivacaine, Dexmedetomidine, Spinal Anesthesia.

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Introduction

Spinal anesthesia is the preferred mode of anaesthesia for lower abdominal surgeries as it is very economical and easy to administer.[1] Postoperative pain is a major problem in infraumbilical surgeries because of shorter duration of spinal anesthesia using only local anesthetics, and thus, early analgesic intervention is needed in postoperative period.Over the years many drugs have been used as an additive to spinal anaesthesia in order to hasten its onset of action, decrease the time to surgical incision, prolong the duration of action and to provide adequate postoperative analgesia. These drugs include midazolam, ketamine, fentanyl, clonidine,[2,3] many opioids and non opioids. Use of opioids is associated with its side effects like pruritis, nausea, vomiting, constipation, and respiratory depression which can be distressing for the patient.[4]

Dexmedetomidine, a highly selective α2 agonist is rapidly emerging as the choice of additive to spinal anaesthesia in view of its property to provide analgesia and awake sedation without respiratory depression along with stable haemodynamics.[5]

Various studies conducted by different authors have used dexmedetomidine in doses of 3 μg, 5 μg, 10 μg and 15 μg and there may be dose related prolongation of duration of motor blockade along with increase in the incidence of side effects of dexmedetomidine namely hypotension and bradycardia.[1,2,6] Hence, there seems to be no clear consensus on the dose of dexmedetomidine to be used as an additive to hyperbaric bupivacaine in spinal anaesthesia for daily practice. Avoidance of side effects of dexmedetomidinewhile ensuring a pain free perioperative period is vital for successful outcome of any surgical procedure.[7–9]

In this study, we aim to compare the efficacy of three different doses (5 μg,10μg and 15μg) of dexmedetomidine given in combination with 0.5% hyperbaric bupivacaine via intrathecal route in patients undergoing infraumbilical surgeries with regards to the haemodynamic stability, incidence of side effects (hypotension and bradycardia) and postoperative analgesia.[10,11]

Subjects and Methods

After obtaining the Institutional Ethical Committee approval, ninety patients of either sex of 18–60 years of age were scheduled for lower abdominal surgeries. All patients, who belong to the American Society of Anesthesiology Class (ASA) I and II, were enrolled for this prospective randomized, double-blinded study. After obtaining written informed consent, all patients were examined and investigated a day before surgery. Patients were advised fasting for 6 h before surgery; they were advised to take tablet alprazolam 0.5 mg and tablet ranitidine 150 mg night before surgery.

On the day of surgery, the patient was preloaded with 15 ml/kg of Ringer lactate solution, after obtaining IV line, half an hour before the procedure. The patient was shifted to operation theatre, connected to multiparameter monitor. Patients were randomly allocated using sealed envelope technique into three groups in a double-blinded manner (both attending anesthesiologist and patient were blinded).

The three groups are

Group BD5: Intrathecal bupivacaine 12 5 mg (2 5 ml + dexmedetomidine 5 μg (0 5 ml

Group BD10: Intrathecal bupivacaine 12 5 mg (2 5 ml + dexmedetomidine 10 μg (0 5 ml

Group BD15: Intrathecal bupivacaine 12 5mg (2 5 ml + dexmedetomidine 15μg (0 5 ml

Subarachnoid block was performed at L_{3-4} level with 25-gauge Quincke spinal needle with the patient in the left lateral position under aseptic precaution. Patients turned to the supine immediately after the block. The anesthesiologist who performed the block recorded the intraoperative data. O_2 inhalation at 2 L/min were kept for all the patients.

The onset and maximum level of sensory block, time to reach maximum level of sensory block, and time of two-segment sensory regression were recorded using 25-gauge hypodermic needle by pinprick. The total duration of analgesia, time to rescue analgesia, onset, and duration of motor block were recorded. Vitals monitored were heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure(DBP), mean arterial pressure (MAP), respiratory rate (RR), oxygen saturation (SpO_2) for every 2 min in the first 10 min, for every 5 min in next 15 min, thereafter every 15 min till the end of surgery.

The sensory onset was defined as the time from intrathecal injection to the loss of pinprick sensation at T_{10} dermatome. The maximum level of sensory block was defined as loss of sensation to pinprick in midclavicular line anteriorly, which was checked every 5 mins for the first 20 min. The time of two-segment regression was defined as time from intrathecal injection to regression of sensory loss to two-segments from highest level. The total duration of analgesia was defined as time from intrathecal injection to time of sensory regression to S1 dermatome. The duration of rescue analgesia was defined as the time from intrathecal injection to time when patient demanded for analgesics; then, the patient will be administered Inj diclofenac 75 mg intramuscularly.

The time of onset of motor block was defined as time from injection of intrathecal drug to the onset of modified Bromage 1 level motor block. The total duration of motor block was defined as the time of intrathecal injection to complete motor regression. The motor level was assessed according to modified Bromage score.[12]

- Bromage 0: The patient can move the hip, knee, and ankle
- Bromage 1: The patient is unable to move the hip, but able to move the knee and ankle
- Bromage 2: The patient is unable to move hip and knee, but able to move the ankle
- Bromage 3: The patient is unable to move the hip, knee, and ankle.

Hypotension was defined as a decrease in SBP by 30% from baseline, and it was treated by incremental dosage of Injection Mephentermine 6 mg and crystalloid fluids.

Bradycardia was defined as HR <50 beats/min, which was corrected using 0.6 mg of IV atropine sulfate. Other adverse effects such as sedation, nausea, and vomiting were recorded and treated accordingly. Sedation was assessed using modified Ramsay sedation scale.[13]

Modified Ramsay sedation scale

- Score 1 Anxious, agitated, restless
- Score 2 Cooperative, oriented, tranquil
- Score 3 Responds to commands only

- Score 4 Brisk response to light glabellar tap or loud noise
- Score 5 Sluggish response to light glabelar tap or loud noise
- Score 6 No response.

Statistical analysis

The statistical analysis of the data was done using Statistical Package for Social Sciences evaluation version 20 (IBM SPSS Statistics for Windows, Armonk, NY: IBM Corp). The data were expressed as either mean or standard deviation for number and percentages. The demographic data of patients were studied for each of the three groups.

Level of significance

- P is level of significance
- P > 0.05; not significant
- P < 0.05; significant
- P < 0.01; highly significant
- P < 0.001; very highly significant.

Result

Patient demographic characteristics were comparable among the groups with respect to age, sex, weight, height, and body mass index, there was no statistically significant difference (P > 0.000) [Figure 1].

	Group D_5	Group D_{10}	Group D_{15}	*P*
Age	37.26±13.70	36.26±11.75	36.96±12.00	0.951
Height	161.80±6.67	164.16±6.73	163.73±8.61	0.422
Weight	60.43±8.75	61.86±8.64	61.80±8.13	0.764
BMI	23.05±2.81	22.91±2.74	23.04±2.50	0.975

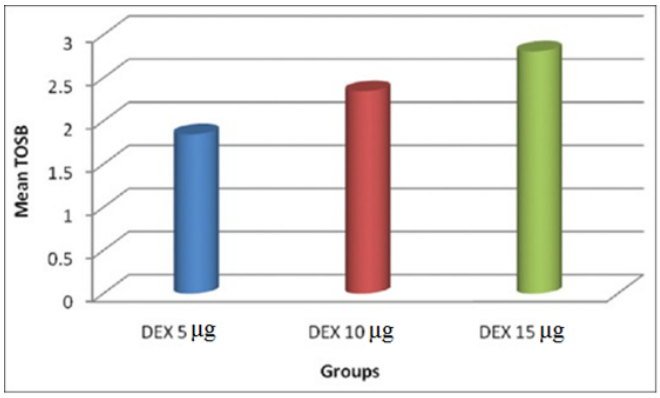
BMI=Body mass index

Figure 1: Demography

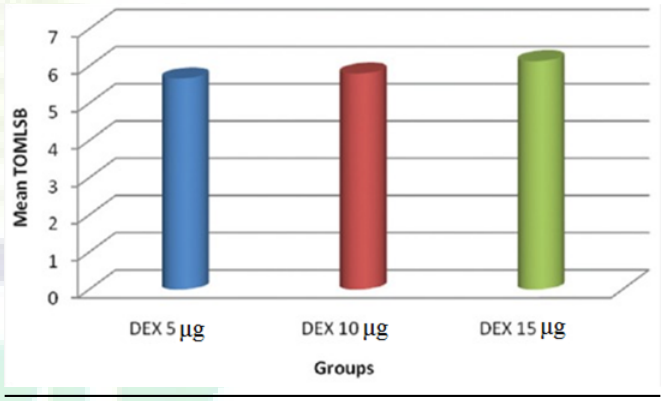
The onset time of sensory block [Graph 1] in Group D_5-2.76 ± 1.32, Group D_{10}-2.45 ± 1.50, and Group D_{15}-1.86 ± 0.93, which is statistically significant (P = 0.025) as the dosage of dexmedetomidine increased the onset time of sensory block is significantly decreased.

The time taken to achieve the maximum level sensory block is not statistically significant among the groups (P = 0.402) [Graph 2].

The time taken for two-segment sensory regression [Graph 3] Group D_5-96.66 ± 33.67, Group D_{10}-116.80 ± 36.27, and Group D_{15}-120.96 ± 30.24, (P = 0.014), which is statistically significant, which is earlier in Group D_5 when compared to Group D_{10} and Group D_{15} (Group D_5 < Group D_{10} < Group D_{15}) [Figure 2].



Graph 1: **The onset time of sensory block in Group D_5-2.76 ± 1.32, Group D_{10}-2.45 ± 1.50, Group D_{15} -1.86 ± 0.93, which is statistically significant (P = 0.025). As the dosage of dexmedetomidine increased, the onset time of sensory block is significantly decreased**



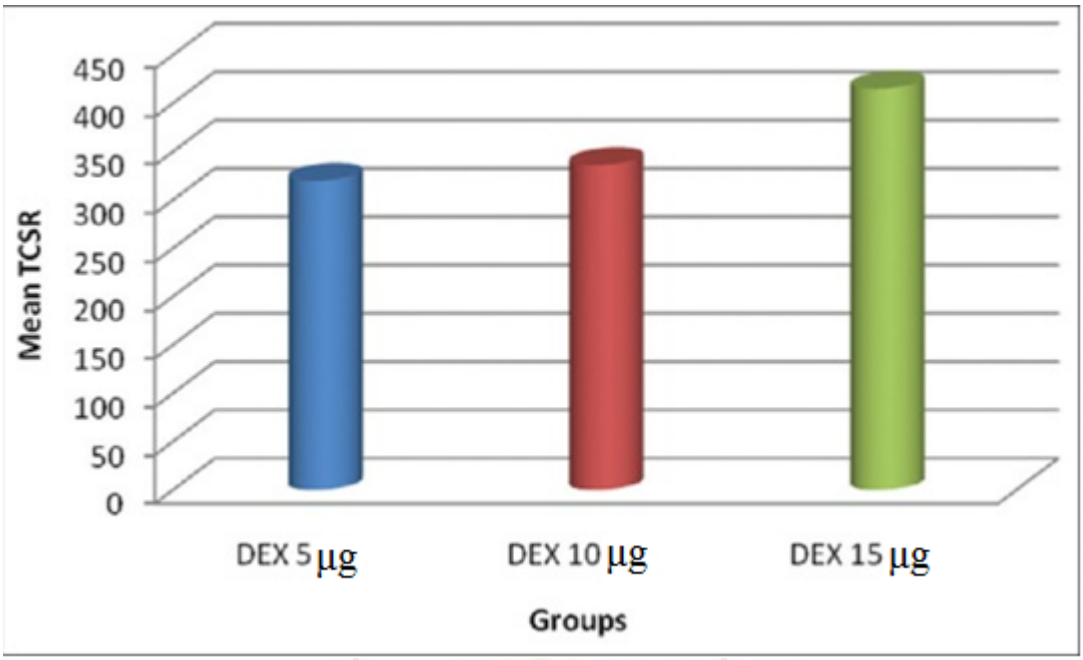
Graph 2: **The time taken to achieve the maximum level sensory block is not statistically significant among the groups (P = 0.402)**

The time taken for complete sensory recovery [Graph 4] in Group D_5-319.83 ± 61.41, Group D_{10}-336.13 ± 61.38, and Group D_{15}-415.20 ± 96.6 which is statistically highly significant (P = 0.000; Group D_{15} > Group D_{10} > Group D_5).

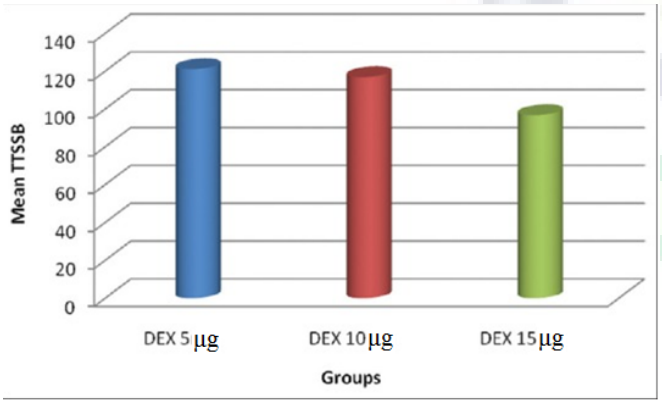
The sensory recovery time is maximum in Group D_{15} compared to Group D_{10} and Group D_5 (Group D_{15}> Group D_{10}> Group D_5).

Time of onset of motor blockade in Group D_{15}-2.26 ± 1.048, Group D_{10}-2.70 ± 2.08, Group D_5-3.76 ± 7.38 though the onset of motor block is early in Group D_{15} when compared to D_{10} and D_5 (Group D_{15} < Group D_{10} < Group D_5), which is statistically not significant (P = 0.413).

The total duration of motor blockade in Group D_{15}-401.23 ± 114.49 is prolonged when compared to Group D_{10}-375.23



Graph 4: **The time taken for complete sensory recovery in Group D_5-319.83 ± 61.41, Group D_{10}-336.13 ± 61.38, Group D_{15}-415.20 ± 96.6. Which is statistically highly significant (P = 0.000) (Group D_{15} > Group D_{10} > Group D_5)**



Graph 3: **The time taken for two-segment sensory regression Group D_5-96.66 ± 33.67, Group D_{10}-116.80 ± 36.27, Group D_{15}-120.96 ± 30.24 (P = 0.014), which is statistically significant, which is earlier in Group D_5 when compared to Group D_{10} and Group D_{15} (Group D_5 < Group D_{10} < Group D_{15})**

	Group D_5	Group D_{10}	Group D_{15}	*P*
Time of onset sensory block	2.76±1.32	2.45±1.50	1.86±0.93	0.025
Time taken to achieve maximum level of sensory block	5.76±0.72	5.50±1.16	5.43±1.07	0.402
TTSSR	96.66±33.67	116.80±36.27	120.96±30.24	0.014
TCSR time taken for complete sensory recovery	319.83±61.41	336.13±61.38	415.20±96.6	<0.001
Total duration analgesia	322.50±71.87	358.70±73.89	458.33±95.21	<0.001
Time for rescue analgesia	377.46±60.05	401.60±61.11	517.96±97.30	<0.001
Time of onset of motor blockade	3.76±7.38	2.70±2.08	2.26±1.048	0.413
Total duration of motor blockade	340.93±86.67	375.23±72.39	401.23±114.49	0.046

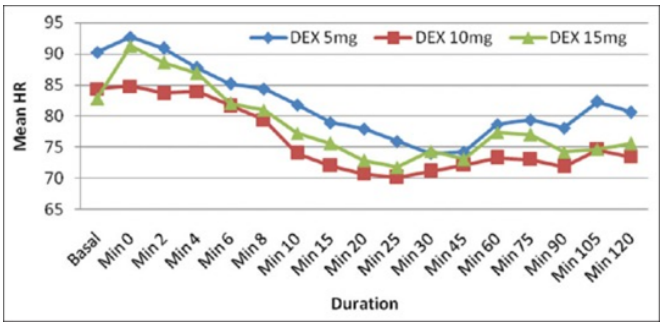
TCSR=Time to complete sensory recovery, TTSSR=Time taken for two segment sensory regression

Figure 2: The results of characteristics of spinal block

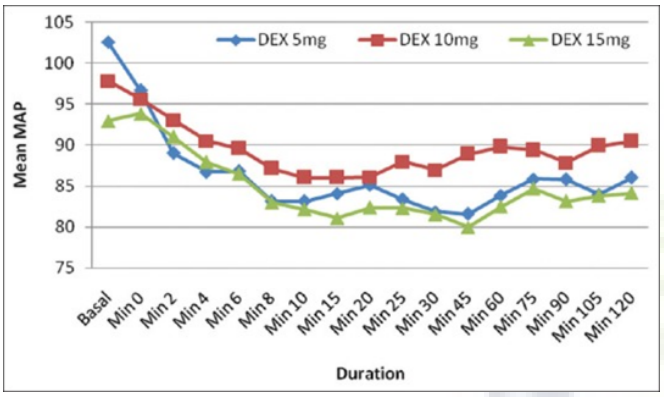
± 72.39 and Group D_5-340.93 ± 86.67 which is statistically significant (P = 0.046). The total duration of motor blockade depends on the dosage; more the dosage prolongs the block.

Hemodynamic and other parameters

The HR, MAP, RR, and SpO_2 were measured at various intervals and were comparable among the groups [Graph 5 and Graph 6].



Graph 5: **Mean heart rate**



Graph 6: **Mean of mean arterial pressure**

The HR fell significantly at 105 and 120 min from basal rate within groups, we noticed bradycardia in 3 (10.0%) patients among Group D_5, 4 (13.3%) patients in Group D_{10}, 10 (33.3%) patients in Group D_{15}, which is statistically significant (P = 0.044). MAP fell significantly at 30^{th}, 45^{th}, and 60^{th} min from basal rate within the groups, we noticed hypotension in 4 (13.3%) patients in Group D_5, 6 (20.0%) patients in Group D_{10}, 13 (43.3%) patients in Group D_{15}, which is statistically significant (P = 0.020) [Figure 3]. We did not notice any respiratory depression and desaturation among the groups at various interval.

Adverse effect	Group D_5, number of patients (%)	Group D_{10}, number of patients (%)	Group D_{15}, number of patients (%)	P
Bradycardia	3 (10.0)	4 (13.3)	10 (33.3)	0.044
Hypotension	4 (13.3)	6 (20.0)	13 (43.3)	0.020
Vomiting	0	0	1 (3.3)	0.364
Hypoventilation	0	0	0	
Desaturation	0	0	0	

Figure 3: Adverse effects

Discussion

Since its FDA approval for use in humans as a short term medication for sedation/analgesia in the intensive care unit, researchers have been exploring the prospect of using dexmedetomidine as an additive in spinal analgesia taking into advantage its highly selective agonistic action for intrathecal $\alpha 2$ receptors which have antinociceptive actions for both somatic and visceral pain. When used intrathecally as an adjuvant to local anesthetics, it prolongs the motor and sensory block of local anesthetics. It may have an additive or synergistic effect secondary to different mechanism of action of local anesthetics.

It acts by binding to the presynaptic C fibers and postsynaptic dorsal horn neurons. Their analgesic action is due to depression of release of neurotransmitters of C fibers and hyperpolarization of postsynaptic dorsal horn neurons.[14]

In this prospective, randomized, double-blind study, in patients scheduled for elective lower abdominal surgeries between age groups of 18 and 60 years of either sex. We have compared the dose-dependent effect of 5, 10, 15 μg of dexmedetomidine added to 12.5 mg of intrathecal bupivacaine. We have studied onset time and duration of motor and sensory block, as well as postoperative rescue analgesia, hemodynamic response, and associated adverse effects such as bradycardia, hypotension, sedation, respiratory depression, nausea and vomiting.

The demographic profile in all the three groups was comparable and statistically insignificant.

The onset of sensory block to T10 is dose dependent, Group D_5-2.76 $\pm$ 1.32, Group D_{10}-2.45 $\pm$ 1.50, Group D_{15}-1.86$\pm$0.93, which is statistically significant (P = 0.025), our results can be compared with Shaikh and Dattatri and Al-Mustafa et al, who also noticed similar results.[15,16]

We did not notice any statistically significant difference in the time to achieve the maximum level of sensory blockade in all the three groups.

The onset of motor block to Bromage 1 was dose dependent in Group D_{15}-2.26 $\pm$ 1.048, Group D_{10}-2.70 $\pm$ 2.08, and Group D_5-3.76 $\pm$ 7.38, but which is statistically not significant (P = 0.413). Our study is comparable with that of Chaudhry et al,[17] they used 5 and 10 μg of dexmedetomidine with 12.5 mg of 5% hyperbaric bupivacaine in femur surgeries, they also did not notice anystatistically significant difference in motor onset time.

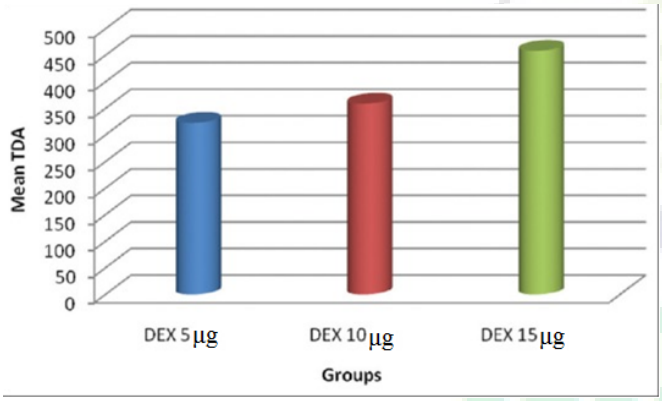
Our study is also comparable favourable with that of Gupta et al,[18] they have also not noticed any significant difference in the motor onset, but they have compared only 5 μg of dexmedetomidine with fentanyl alone with hyperbaric bupivacaine.

The time taken for two-segment sensory regression is dose-dependent Group D_5-96.66 $\pm$ 33.67, Group D_{10}-116.80 $\pm$ 36.27, Group D_{15}-120.96 $\pm$ 30.24 (P = 0.014), which is statistically significant. Our study is comparable with that of

Shaikh and Dattatri,[15] they have also noticed the similar results. Our study is also comparable with Eid et al,[11] study who also observed a statistically significant in the time to two segment regression.

The time taken for complete sensory recovery is dose-dependent Group D_5-319.83 ± 61.41, Group D_{10}-336.13 ± 61.38, Group D_{15}-415.20 ± 96.6, which is statistically highly significant (P = 0.000). Our study is comparable with studies by Eid et al,[11] and Bansal et al,[14] wherein they also observed a highly significant difference.

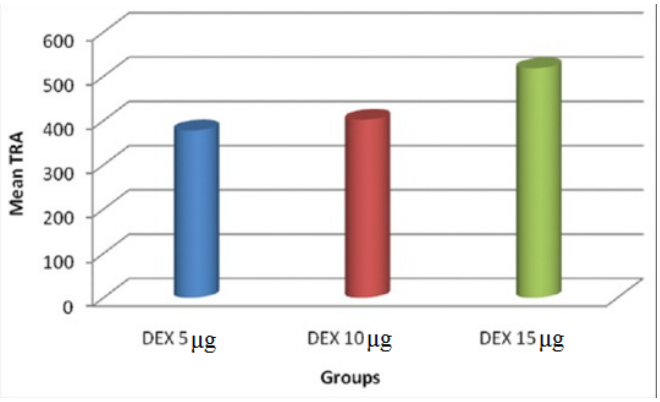
The total duration of analgesia [Graph 7] is dose dependent in Group D_5-322.50 ± 71.87, Group D_{10}-358.70 ± 73.89, Group D_{15}-458.33 ± 95.21. (P = 0.000) which is statistically highly significant. Our results are comparable with Bansal et al,[14] and Gupta et al,[7] they have also noticed a significant difference among the groups in a dose-dependent manner. Our study is also comparable with Gupta et al,[7] and Gupta et al,[18] who also noticed the prolonged duration of analgesia in their studies.



Graph 7: **Total duration of analgesia in Group D_5-322.50 ± 71.87, Group D_{10}-358.70 ± 73.89, Group D_{15}-458.33 ± 95.21, which is statistically highly significant (P = 0.0001) (Group D_{15} > Group D_{10} > Group D_5)**

The time for rescue analgesia [Figure 8] in Group D_5-377.46 ± 60.05, in Group D_{10}-401.60 ± 61.11, and in Group D_{15}-517.96 ± 97.30, which is statistically highly significant, P < 0.0001. Our study is comparable with Bansal et al,[14] (they have compared between dexmedetomidine 5 μg and 10 μg) and Eid et al,[11] (they have compared between dexmedetomidine 10 μg and 15 μg). They have also noticed the prolonged time for request of the first dose of analgesic in a dose-dependent manner, which is statistically significant.

Our study is also comparable with Gupta et al,[7] Nayagam et al,[19] Gupta et al,[18] who also noticed prolonged rescue analgesia with dexmedetomidine as compared to fentanyl and clonidine.



Graph 8: **Time for rescue analgesia Group D5-377.46 ± 60.05, in Group D10-401.60 ± 61.11, in Group D15-517.96 ± 97.30, time of rescue analgesia is early in Group D5 compared to Group D10 and Group D15 (Group D5 < Group D10 < Group D15) which is statistically highly significant P < 0.0001**

The total duration of motor blockade in Group D_{15}-401.23 ± 114.49 is prolonged when compared to Group D_{10}-375.23 ± 72.39 and Group D_5-340.93 ± 86.67 which is statistically significant (P = 0.046). The total duration of motor blockade depends on the dosage; more the dosage prolongs the block. Our study is comparable with Bansal et al,[8] Eid et al,[11] Gupta et al,[7] and Shaikh and Dattatri,[15] in which they have also observed the dose-dependent prolongation of motor blockade. Our study is also comparable with Singh et al,[20] who have also observed the similar results with dexmedetomidine in comparison with clonidine.

In our study, bradycardia was seen in 10% cases of Group D_5 and 13.3% cases in Group D_{10}, 33.3% cases in Group D_{15}, which is statistically significant. In our study, hypotension was observed in 4 (13.3%) cases in Group D_5, 6 (20%) cases in Group D_{10}, 13 (43.3%) cases in Group D_{15}, which is statistically significant. This is comparable with Al-Mustafa et al,[16] who in their study, found a dose-dependent decrease in MAP when compared to bupivacaine. Our study is also comparable with Khan et al,[21] in which they noticed a higher incidence of bradycardia and hypotension in dexmedetomidine group as compared to fentanyl group. Episodes of hypotension were treated with graded dose of IV injection mephentermine 6 mg, and bradycardia was treated with IV atropine 0.6 mg.

We observed 1 case of vomiting in Group D_{15} which was insignificant. There was no incidence of respiratory depression and desaturation in our study. Our study is comparable with Gupta et al,[7] Eid et al,[11] Caudhry et al,[17] and Shaikh and Dattatri,[15] with respect to these adverse effects.

In our study, we have noticed Grade 2 Ramsay sedation score in all three groups, which is statistically insignificant.

Conclusion

Dexmedetomidine is an effective additive to spinal anaesthesia which provides a stable haemodynamics and prolonged post-operative analgesia. The ideal intrathecal dose of dexmedetomidine is 10 μg with minimal side effects and hemodynamic response with prolonged postoperative analgesia.

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