International Journal of Science and Research Archive
eISSN: 2582-8185
Cross Ref DOI: 10.30574/ijsra
Journal homepage: https://ijsra.net/

(RESEARCH ARTICLE)

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Effect of dexmedetomidine infusion on characteristics of spinal anesthesia with hyperbaric bupivacaine: A randomized controlled trial

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International Journal of Science and Research Archive, 2024, 12(01), 386–393

Publication history: Received on 30 March 2024; revised on 06 May 2024; accepted on 09 May 2024

Article DOI: https://doi.org/10.30574/ijsra.2024.12.1.0816

Abstract

Dexmedetomidine is a highly selective and potent α2 agonist having both sedative and analgesic properties. This study was aimed to assess the effects of intravenous dexmedetomidine on sensory, motor, haemodynamic parameters and sedation during subarachnoid block (SAB) with 0.5% hyperbaric bupivacaine. Sixty patients undergoing infraumbilical and lower limb surgeries were allocated into 2 groups, group D (n=30) received dexmedetomidine infusion at the rate of 0.5 µg/kg/h over an hour prior to SAB and continued till the end of surgery whereas patients in group C (n=30) received similar volume of normal saline infusion for the same duration. Onset of sensory block was 63.77 ± 7.42 s in group D compared with 126.2 ± 12.83s in group C. Two segment regression time was 177.03 ± 11.64 min in group D and 85.67 ± 10.32 min in group C and analgesia duration was 287.67 ± 14.84 min in group D and 149 ± 13.16 min in group C. Onset of motor block was 3.95 ± 0.65 min in group D and 4.5 ± 0.62 min in group C. Motor blockade duration was prolonged in group D compared with group C. There was significant difference between the groups in respect to block characteristics. Administration of intravenous dexmedetomidine during SAB hastens the onset and prolongs the duration of sensory and motor block. It also provides conscious sedation and additional analgesia.

Keywords: Dexmedetomidine; Bupivacaine; Subarachnoid Block

1. Introduction

Regional anaesthesia offers several advantages over general anaesthesia for infraumbilical and lower limb surgeries like decrease incidence of deep vein thrombosis (DVT) and amount of operative blood loss[1]. Among regional anaesthesia, spinal anaesthesia is a frequently used technique in infraumbilical and lower limb surgeries. Regional anaesthesia, however, may promote some type of discomfort caused by the procedure itself or by a prolonged perioperative period, requiring the simultaneous administration of hypnotic, sedative and amnestic drugs. Many techniques and drug regimens, with partial or greater success, have been tried from time to time to eliminate the anxiety component and to prolong postoperative analgesia during regional anaesthesia. Different adjuvants[2] have been used to prolong subarachnoid block, to delay onset of postoperative pain and to reduce analgesic requirements. Uses of opioids as adjuvant have some adverse effects like nausea and vomiting, urinary retention, constipation and depression of ventilation[3]. So other adjuvants like tramadol[4], and midazolam[5] were also tried in this respect but these are not devoid of adverse effects. Oral pregabalin 300mg resulted in 50% reduction in 24-hour postoperative morphine requirements. However, combining pregabalin and dexamethasone provided no additional effects on pain or opioid requirement postoperatively nor did it reduced nausea or vomiting.[6] Many clinical studies have been carried out using intrathecal alpha-2 agonist such as clonidine and dexmedetomidine as adjuvants to local anaesthetics.[7]

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Dexmedetomidine is a highly selective and potent alpha-2 agonist and is seven to ten times more selective for alpha-2 receptor compared to clonidine and has a half-life of 2 to 3 hours.[8] It has sedative, analgesic, and anaesthetic sparing effect, has also been used for premedication in general anaesthesia.[9] It is known to induce sedation, decrease anaesthetic drug requirement and improve perioperative haemodynamics by attenuating blood pressure and heart rate responses to surgical stimulation. It has been used safely as premedicant or as a sedative in patients undergoing surgical procedures under regional anaesthesia.[10,11,12] There are very few data regarding the effect of intravenous dexmedetomidine on spinal anaesthesia and all published studies have used 1 μg/kg bolus followed by infusion.[12]

Hence the present study was conducted to assess the effects of intravenous (IV) dexmedetomidine on sensory and motor block characteristics, haemodynamic changes and sedation during subarachnoid block in patients undergoing infraumbilical and lower limb surgeries.

2. Methodology

After approval from Institutional Ethics Committee and written informed consent from patients, the study was carried out, after taking detailed history and thorough physical examination, assessment of spine, airway examination and routine preoperative investigations in sixty (60) adult patients. Patients of ASA grade I and II, aged 18-64 years undergoing elective infraumbilical and lower limb surgeries were enrolled for this study. As per the previous study (Effect of supplementation of low dose intravenous dexmedetomidine on characteristics of spinal anaesthesia with hyperbaric Bupivacaine- SS Harsoor et al),with SD of 120 mins in the duration of analgesia at 80% of minimum study power and at 5% level of significance, the required sample size was calculated to be 56 (28 in each group), rounding up we kept the sample size as thirty (30) in each group. Subjects were randomly allocated into 2 groups using sealed envelope system for randomisation, Group D (n=30) received Dexmedetomidine infusion and Group C (n=30), the control group, received similar volume of saline infusion using computer generated random numbers.

All patients were kept fasting overnight and they were blinded to the group allocation. An IV line was established with 18-gauge cannula and all patients were preloaded with Ringer lactate solution 10 ml/kg after arriving at the preanaesthetic care room. Standard monitors were attached and baseline haemodynamic parameters were recorded. Group D patients received IV Dexmedetomidine at the rate of 0.5 μg/kg/hour started 1 hour prior to subarachnoid blockade and continued till end of operation. Group C received similar volume of normal saline. On arrival in the operation theatre standard monitors were re -attached to note the physiological parameters like ECG, non-invasive blood pressure, heart rate and SpO2. Disposable BIS (Bispectral Index) sensor electrodes were applied to the patient's forehead. After proper antiseptic dressing and draping, lumbar puncture was done at L 3-4 interspace using a 25-gauge Quincke needle, 12.5 mg of 0.5% hyperbaric Bupivacaine was deposited in the subarachnoid space after confirming the free flow of CSF.

Sensory blockade was checked by pin prick sensation. Time to attainment of T10 blockade was considered as time of onset of sensory blockade. Recovery time for sensory blockade was defined as two dermatome regression of anaesthesia from maximum level.

Motor blockade was assessed immediately after sensory block assessment using a Modified Bromage scale. Motor block onset is the time to attain a Modified Bromage Scale of 3. Motor block duration is the time for return to Modified Bromage Scale1.

The highest sensory block level and recovery time of both sensory and motor block were recorded. The levels of sedation were evaluated throughout the operation using BIS Monitor.

After completion of surgery the patients were sent to the postoperative recovery room. Postoperative pain was assessed by VAS (Visual Analogue Scale) at 0, 4, 8 and 12 postoperative hours where 0 hour is the time when the patient is being attended/assessed first by the fixed blinded case recorder at the post operative care room. Rescue analgesia in the form of injection Diclofenac 75mg intravenous was administered postoperatively when VAS score ≥ 40 or on demand. Time of administration of first rescue analgesia was noted. Total requirement of Diclofenac in first 12 postoperative hours was also recorded. Degree of consciousness was monitored by BIS monitor. All the parameters were recorded by a prefixed blinded single case recorder.

2.1. Statistical analysis

Numerical variables were compared between groups by student's independent samples t-test if normally distributed or by Mann-Witney U test if otherwise. Chi-square test or Fisher's exact test were employed for intergroup comparison of categorical variables. All analysis were two tailed and $p<0.05$ was considered statistically significant

3. Results

Data were summarized by descriptive statistics namely mean and standard deviation (SD) for numerical variables and counts and percentages for categorical variables

Table 1 Demographic Parameters

Parameter	Group D (n=30)	Group C (n=30)	p value
Age(yrs)	41.1±12.90	42.23±11.74	0.723
Sex ratio(F:M)	12:18	14:16	0.797
BMI	23.04±2.34	23.15±1.87	0.837
ASA status (I: II)	19:11	20:10	1.000
Duration of surgery(mins)	110±26.78	110±24.90	0.921

Demographically both the groups were comparable. There was no significant difference in demographic data, surgical characteristics, duration of surgery between the two groups.

Table 2 Sensory and Motor Block Parameters

Parameter	Group D (n=30)	Group C (n=30)	p Value/Remarks
Time of onset of sensory block(s)	63.77±7.426	126.20±12.834	<0.001
Peak sensory block height (PSBH)	PSBHT8:PSBHT10 15:15	PSBHT8:PSBHT10 14:16	Not significant
Two segment regression of sensory block(mins)	177.03±11.637	85±10.317	<0.001
Time of onset of motor block(mins)	3.95±0.648	4.50±0.616	<0.001
Regression of motor block to Bromage 1(mins)	219.83±19.497	130.17±13.162	<0.001
Timing of first rescue analgesic(mins)	287.67±14.84	149.00±13.157	<0.001
Total Diclofenac requirement in first 12 hour period(mgs)	112.50(75-150)	225.00(150-225)	<0.001

There was statistically significant difference between two groups in all parameters except peak sensory block height. Time of onset of sensory block was significantly faster in group D compared to group C with $p<0.001$.Two segment regression time for sensory block was significantly prolonged in group D compared to group C($p<0.001$).Time of onset of motor block was also significantly faster in group D compared to group C, $p<0.001$.Regression of motor block to Bromage 1 took longer time in group D compared to group C,$p<0.001$.Timing of first rescue analgesic was significantly prolonged in group D compared to group C with $p<0.001$.[Table 2].

Concerning the perioperative haemodynamic parameters, systolic blood pressure, diastolic blood pressure and mean arterial blood pressure were significantly lower in group D than in group C.

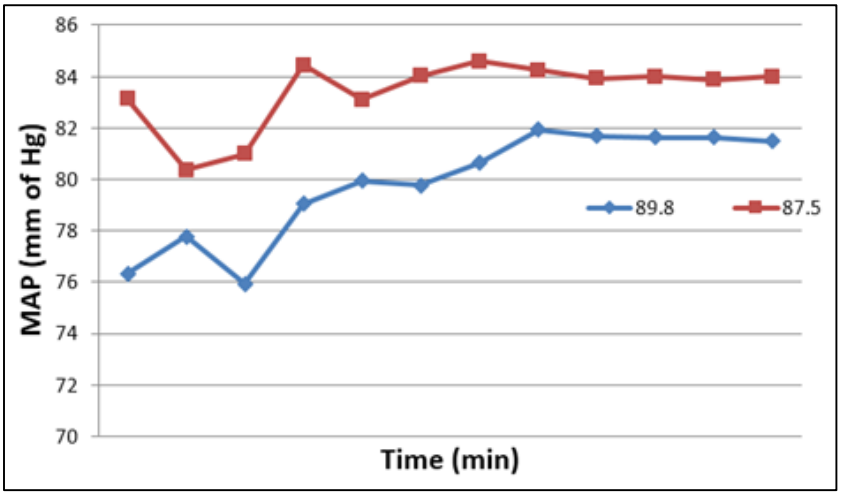

Figure 1 Mean Arterial Pressure v/s Time

MAP from baseline noted throughout intraoperative period and a statistically significant difference noted ($p < 0.05$) at 5, 15, 20, 25, 30 and 50 min between groups.

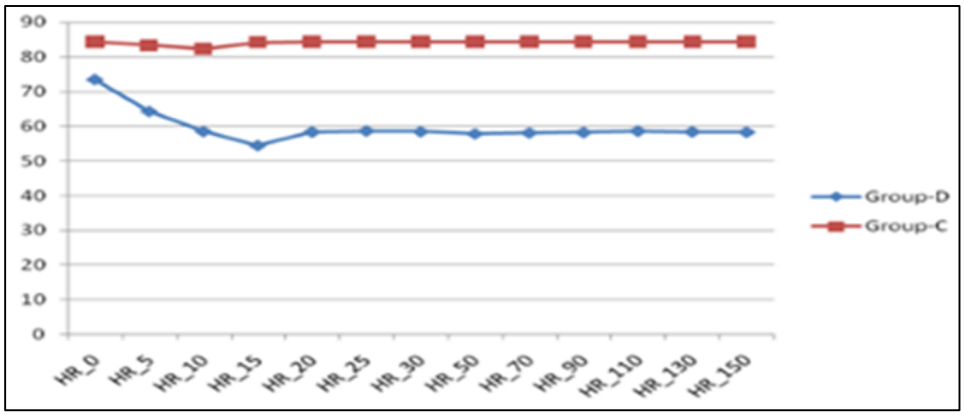

Figure 2 Heart Rate v/s Time

Fall in HR noted throughout intraoperative period in Group D and a statistically significant difference in p value (<0.05) noted between groups.

Respiratory rate (RR) in both groups was found to be comparable at 0,5,10,15,30 min($p>0.05$). RR at 20,25,50,70,90,110,130,150 min in group D was significantly lower than group C, but oxygen saturation was comparable between both the groups.

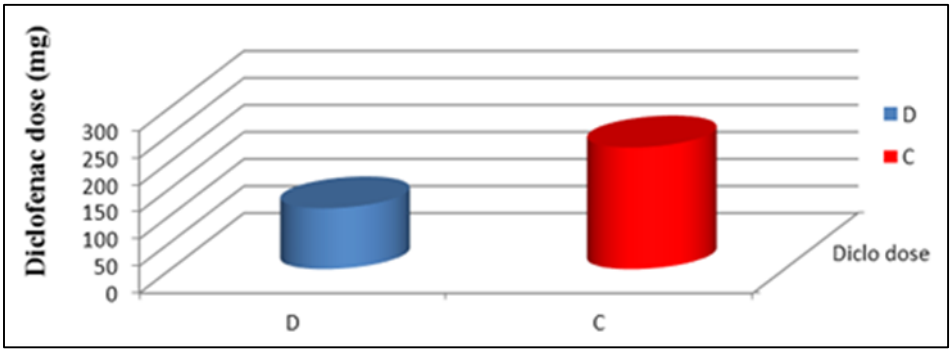

Figure 3 Comparison of Total Diclofenac Requirement in Postoperative 12 hr. Period

The analgesic requirement in Group D (112.50 mg) was significantly less than in Group C (225.00 mg) with p<0.001[Fig 3 & Table 2].

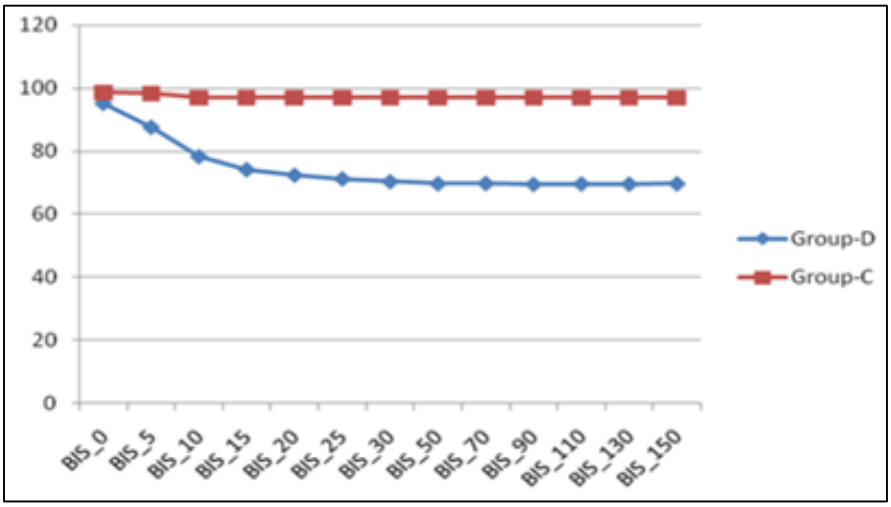

Figure 4 BIS% v/s Time

The linear graph shows a decreasing trend of BIS in group D.

4. Discussion

Dexmedetomidine is a more selective α2-A receptor agonist than clonidine, with more sedative and analgesic effects. Recent studies have shown the efficacy of both intrathecal and IV dexmedetomidine in prolonging spinal anesthesia. Prolongation of spinal anesthesia after IV dexmedetomidine is by its supra-spinal action at locus ceruleus and dorsal raphe nucleus. Dexmedetomidine has a role in modulating pain and inhibiting the transmission and perception of pain as well as anxiety. It causes bradycardia, hypotension and even transient hypertension as hemodynamic side effects.

In the present study, Group D patients received infusion of dexmedetomidine at the rate of 0.5 µg/kg/hr 1 hour prior to subarachnoid block and continued till the end of surgery and group C received similar volume of normal saline infusion. The duration of analgesia was significantly prolonged in the dexmedetomidine group (287.67 ± 14.84 min) compared to the control group (149.00 ± 13.16) and total requirement of analgesics was significantly less in the dexmedetomidine group compared to the control group in our study. Similarly, Hong *et al*.[12] Noticed that postoperative pain intensity was lower and the mean time to first request for postoperative analgesia was longer in the dexmedetomidine group compared to the control group (6.6 h vs. 2.1 h). Kaya *et al*.[13] in their study observed that dexmedetomidine increased the time to first request for postoperative analgesia and decreased the analgesic requirements.

In another study observing the effect of dexmedetomidine infusion on spinal anaesthesia with ropivacaine by Elcicek K et al.[11], it was observed that dexmedetomidine bolus of 1 µg/kg followed by infusion at 0.4 µg/kg/h prolonged the duration of sensory and motor regression. Recently, administration of a single bolus of 1 µg/kg and 0.5 µg/kg, also were reported by Hong et al.[12] and Kaya et al.[13] respectively to prolong the duration of analgesia and sensory blockade. The duration of sensory block and analgesia in our study were similar with above studies despite using a lower initial loading dose of 0.5 µg/kg, compared with 1 µg/kg. Furthermore, by Jaakola ML et al.[14] an evaluation of the analgesic effect of different doses of IV dexmedetomidine (0.25, 0.5 and 1 µg/kg) on ischemic pain in healthy volunteers demonstrated moderate analgesia with a ceiling effect at 0.5 µg/kg.

Dexmedetomidine group didn't have higher level of sensory block compared to the control group in our study, contrary to the study results of Kaya *et al*.[13] Higher level of sensory-motor block height was also found in study done by Reddy VS et al.[15] whereas, in study done by Magalhaes E et al.[16] using low dose intravenous dexmedetomidine as sedative agent after spinal anaesthesia reported no difference in block characteristic by dexmedetomidine. In our study, the mean time for two-dermatomal regression of sensory blockade was significantly prolonged in the dexmedetomidine group (177.03 ± 11.64 min) compared to the control group (85.67 ± 10.31). Hong *et al*.[12] reported that the mean time to two-segment regression was prolonged in the dexmedetomidine group (78 min vs. 39 min for cold and 61 min vs. 41 min for pinprick for dexmedetomidine group and control group, respectively). Similar observations were noted by Kaya *et al.*[13], Tekin *et al.*[17] and Harsoor et al.[18] in the dexmedetomidine and control groups, respectively. In our study, motor block was

significantly prolonged in the dexmedetomidine group. Similarly, Elcicek *et al*.[14] and Hong *et al*.[15] also found that the complete resolution of motor blockade was significantly prolonged in the dexmedetomidine group. Contrary to the above studies, Kaya *et al*.[13] reported no significant prolongation in the duration of motor block in the dexmedetomidine group compared to the control group.

Twenty percent of patients in the dexmedetomidine group had bradycardia requiring atropine compared to the control group in which none of the patients developed bradycardia, which is similar to the findings of Harsoor et al.[18] where they found 16% vs 0% bradycardia in dexmedetomidine group vs control group. Studies by Elcicek et al.[11] and by Bajwa SJ et al.[19] on intravenous dexmedetomidine with loading dose of 1 μg/kg over 5-10 min had bradycardia as one of the prominent side effect with incidence up to 30-40%. The reported bradycardia in all these studies was transient and were easily reversed with intravenous atropine.

In the present study, intraoperative and postoperative systolic, diastolic, and mean arterial blood pressures were significantly lower in the dexmedetomidine group at different time points as compared to the control group and hypotention was treated with iv mephentermine. Eliceck *et al*.[11] reported significant decrease in mean arterial pressure in the dexmedetomidine group as compared to the control group. Contrary to the above observations, Al Mustafa *et al.*[10] and Tekin *et al*.[17] reported no significant difference in mean arterial pressures in the dexmedetomidine and control groups.

Dexmedetomidine does not cause significant respiratory depression despite providing good sedation resulting in wide safety margins. In the present study, oxygen saturation was maintained equally well in both the groups during surgery and in the postoperative period, similar to the study results of Harsoor et al.[18]. The aim of sedation in regional anaesthesia technique include general patient comfort, freedom from specific discomfort and some amnesia for entire procedure. Proper sedation has shown to improve the patient satisfaction during regional anaesthesia and may be considered as a mean to increase the patient acceptance for regional anaesthesia technique. It sometimes covers up some inadequate or insufficient block and can help to reduce the requirement of opioid analgesic and indirectly contribute to reduction in postoperative nausea vomiting[20]. The sedative action of dexmedetomidine differs from benzodiazepine and propofol that act through GABA receptor and produces clouding of consciousness and at times patient co-operation may be lost whereas the sedation produced by dexmedetomidine which acts on the locus ceruleus of the brain, which induces sedation resembling natural sleep by means of sleep modulation and maintaining respiratory control[21] with minimal effect on respiratory rate and tidal volume[22]. Excessive sedation has been reported when intravenous dexmedetomidine (1 μg/kg) when given as bolus dose[23] whereas dexmedetomidine when administerd in lower dose of (0.5 μg/kg with or without infusion), adequate sedation has been reported. Most of patients receiving dexmedetomidine were sedated, but easily arousable in the present study. There was statistically significant difference in the distribution of BIS among the population of study groups (p value <0.001). This significance existed over entire time during intraoperative period.

Postoperative shivering, postoperative nausea and vomiting, headache was not observed in any patient

5. Conclusion

It can be concluded that infusion of dexmedetomidine at the rate of 0.5 μg/ kg/ hour, one hour prior to hyperbaric bupivacaine spinal anaesthesia and intraoperatively, hastens the onset of sensory and motor block and prolongs the total duration of analgesia and sensory and motor block. Further, IV dexmedetomidine supplementation during SAB produces satisfactory arousable sedation without causing respiratory depression.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

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