ORIGINAL ARTICLE

A comparative study to evaluate the efficacy of intrathecal dexmedetomidine and intrathecal clonidine as an adjuvant to hyperbaric 0.75% ropivacaine in patients undergoing orthopedic surgery under spinal anaesthesia

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Abstract

Background: Spinal anaesthesia is a widely preferred technique for lower limb surgeries. Various adjuvants are used to prolong the duration and provide good postoperative analgesia. In our study, we compared the efficacy of intrathecal α-2-adrenergic receptor agonist, clonidine, and dexmedetomidine as an adjuvant to hyperbaric ropivacaine 0.75%. *Aim and Objectives:* To evaluate the efficacy of intrathecal dexmedetomidine and clonidine with hyperbaric ropivacaine 0.75% in orthopaedic surgeries. *Material and Methods:* The study was conducted in 100 patients of ASA I and II, randomly allocated in two groups (50 each) – 1) Group RC: 3.2 ml of intrathecal ropivacaine 0.75% heavy + 30 mcg of injectionclonidine, and; 2) Group RD: 3.2 ml of intrathecal ropivacaine 0.75% heavy + 3 mcg of injection dexmedetomidine. The drugs were compared based on the block characteristics, analgesic duration, 24-hour analgesic requirement, hemodynamic parameters, and side effects, if any. *Results:* Duration of sensory blockade (RC 268.98 ± 39.44 mins vs RD 288.62 ± 43.01 mins, $p = 0.015$) and motor blockade (RC 240.64 ± 39.93 mins vs RD 260.24 ± 39.30 mins, $p = 0.011$) was significantly prolonged in Group RD. The duration of analgesia was statistically significantly prolonged in Group RD 374.3 ± 30.06 mins vs Group RC 354.9 ± 32.21 mins, $p = 0.005$. There was significantly a greater 24-hour analgesia requirement in patients from Group RC (2.24 ± 0.59, $p = 0.033$). The side effects were comparable between the two groups. *Conclusion:* We recommend the use of intrathecal dexmedetomidine as an adjuvant in spinal anaesthesia as it prolongs the duration of sensory and motor blockade and provides good postoperative analgesia with hemodynamic stability and minimal side effects.

Keywords: ropivacaine, dexmedetomidine, clonidine, α2-adrenergic receptor, spinal anaesthesia

Introduction

The subarachnoid block is an age-old technique for providing anaesthesia in lower limb orthopaedic procedures because of its reliability, low cost, less blood loss, and good surgical field with decreased incidence of deep vein thrombosis and pulmonary complications in comparison with general anaesthesia [1]. Ropivacaine, a long-acting local anaesthetic, since its clinical introduction in 1996, has been used for various indications and has shown a better safety profile than bupivacaine with a comparatively decreased neurotoxic and cardiotoxic potential [2-3]. However, its drawbacks are a shorter duration of subarachnoid block and lesser duration of postoperative pain relief. Various neuraxial adjuvants like opioids, ketamine, and α-adrenergic agonist clonidine have been shown to enhance the quality of subarachnoid blockade when added to local anaesthetic drugs. This study was conducted to study α-adrenergic agonists dexmedetomidine and clonidine when added intrathecally as an

adjuvant and their effect on subarachnoid block characteristics and postoperative pain relief. Our primary aim was to compare the intrathecal dexmedetomidine and intrathecal clonidine as an adjuvant to 0.75% ropivacaine heavy in onset time, duration of sensory and motor blockade, and postoperative analgesic duration. Our secondary aim was to compare the incidence of side effects seen with the use of dexmedetomidine and clonidine as intrathecal adjuvants.

Material and Methods

We conducted this prospective, randomized, double-blind study after approval was taken from the Institutional Ethics Committee over a duration of one year and six months. One hundred patients in the age group of 18 to 65 years, with American Society of Anesthesiologists (ASA) Class I and II, of either sex who were planned for elective orthopaedic lower limb surgeries under subarachnoid block were selected. Randomization of patients into two groups with 50 patients in either group was done using a computer-generated table of random numbers method. We took informed written consent from all the willing patients who gave consent for the study. Patients with infection at location of subarachnoid block, any coagulation disorders, bleeding diathesis, sinus bradycardia, patients on treatment with α-adrenergic antagonists, beta-blockers, severe stenotic valvular heart disease, hypovolemia, obesity (BMI > 30 kg/m^2), allergic to study drugs, spine deformity, central or peripheral neuropathies, pregnant or lactating women, chronic opioid user, or refusal to participate were excluded. The preoperative anaesthetic assessment included clinical examination and laboratory investigation. Preoperatively, nil-by-mouth orders were given for 8 hours before surgery. The Visual Analog Scale (VAS) for grading the pain with VAS 0 indicating no pain and VAS 10 indicating worst possible pain was explained to patients. We calculated the sample size for our study by taking the mean time to reach T10 sensory block from the study done by Kanazi *et al*. [4]. Taking these values at a confidence limit of 95%, and a power of 80%, a sample size of 50 cases was allotted in both groups. Patients were randomly divided using a computer-generated table of random numbers into Group RC and Group RD with 50 patients in both groups. In Group RC, 50 patients were given intrathecal ropivacaine 0.75% heavy 3.2 ml added with injectionclonidine 30mcg (normal saline added up to 0.3 ml) to make the final volume 3.5 ml. In Group RD, 50 patients were given intrathecal ropivacaine 0.75% heavy 3.2 ml added with injection dexmedetomidine 3 mcg (normal saline added up to 0.3 ml) to make the final volume 3.5 ml. An anaesthesiologist who was not participating in the study made the study solution under sterile conditions. The anaesthesiologist giving the subarachnoid block and the patient were both blinded.

After confirming nil by mouth status, the patient was shifted to Operation Theatre (OT). In OT, ASA standard monitors were attached, and baseline values of Pulse Rate (PR), Oxygen Saturation (SpO_2), and Mean Arterial Pressure (MAP) of the patient were noted and monitored. A wide-bore 18-gauge intravenous line was established, and preloading was done with lactated ringer solution (15 ml/kg). Subarachnoid block with the study drug was performed in a sitting position with a 26-gauge Quincke needle at L3-L4 space with a midline approach under all aseptic precautions.

Then patients were given a supine position. Fluid maintenance was given with ringer lactate at 10 ml/kg/hour. An anaesthesiologist, who administered and assessed the subarachnoid block and hemodynamic parameters was not involved in the study.

Hemodynamic parameters were noted at 2.5 minutes initially for 15 minutes, then every 15-minute interval for 1 hour, and then hourly for the next 6 hours. The sensory block was evaluated by checking for loss of pinprick sensation. The sensory blockade onset was taken as the time from intrathecal injection to the loss of pinprick sensation at T10 level. The highest dermatomal level of sensory block was noted. Sensory blockade duration was taken as the time taken for sensory regression from the highest dermatomal level to S1. Motor blockade was evaluated by modified Bromage criteria which was graded as 0- No motor block,1- Inability to raise straight leg; able to move knees and feet, 2- able to move feet only, 3- Complete motor block. The motor blockade onset time was time taken from intrathecal injection to grade 3 motor blockade. The motor blockade duration was noted as a time interval taken from the time of intrathecal injection administration to the full recovery of motor power (grade-0). After the completion of the surgery, pain scores were assessed using VAS. The time from intrathecal injection of the study drug to the first demand for rescue analgesia from the patient for pain relief or VAS > 3 was noted as the duration of analgesia. Injection paracetamol 1 g intravenous was given for pain relief. Side effects like hypotension, bradycardia, nausea, vomiting, shivering, respiratory depression, or any other side effects within 24 hours were recorded. Hypotension was considered as systolic blood pressure below 90 mmHg or if decreased by more than 20% of the baseline value was managed with intravenous increments of 5 mg ephedrine.

Bradycardia was recorded as a pulse rate of less than 60 beats per minute and was treated by injection atropine intravenously. Any episodes of nausea or vomiting were managed by giving injection ondansetron 4 mg intravenously. Ramsay sedation scale was used to evaluate sedation in patients, and it was graded as: 1) patient anxious, agitated, or restless; 2) patient cooperative, oriented, tranquil; 3) patient responds to commands; 4) asleep but with brisk response to stimuli; 5) asleep, sluggish response to stimuli; and 6) asleep, no response to stimuli. Onset and duration of sensory blockade & highest dermatomal level achieved, onset and duration of motor blockade, duration of analgesia, and occurrence of any side effects were recorded and compared in both groups.

Statistical analysis

All the data entry was done in Microsoft excel data sheet and statistical analysis was done using Statistical Package for Social Sciences (software version 25.0) for Windows package (SPSS Science, Chicago, IL, USA). All data were represented as mean with standard deviation for quantitative data and as percentages for qualitative data. The two groups were compared using Independent t-test for quantitative data, and the Chi-square test was used for qualitative data. In the results, $p < 0.05$ was taken as statistically significant.

Results

All patients (n = 100) completed the study with 50 patients in each group. There was no statistical difference in patient demographic data in terms of age, height, weight, ASA grading, and duration of surgery between Group RC and Group RD as shown in Table 1. The onset time of sensory blockade up to T10 was similar in the two groups RC and RD (6.00 ± 0.83 mins vs 5.72 ± 0.62 mins) with p-value not significant. The onset time to motor block was also similar in the two groups RC and RD (7.10 mins ± 0.86 vs. 6.95 ± 0.76 mins, $p >$ 0.05) and statistically not significant (Table 2). The duration of sensory blockade (Group RC 268.98 ± 39.44 mins vs Group RD 288.62 ± 43.01 mins, p = 0.015) and motor blockade (Group RC 240.64 ± 39.93 mins vs Group RD 260.24 ± 39.30 mins, p = 0.011) were significantly prolonged in Group RD as compared to Group RC (Table 2). The mean values of Heart Rate (HR) and Mean Arterial Pressure (MAP) of either group were comparable (Figures 1, 2). All patients always had oxygen saturation (SpO_2) greater than 96% throughout the study duration and was comparable between the two groups. A VAS score of less than 3 was observed in both groups during the whole duration of the surgery and there was no additional requirement of analgesics intraoperatively. VAS score was comparable between both groups. However, at the 5th and 6th hour, there was an increase in VAS score between both groups which was not statistically significant (Figure 3). The duration of analgesia was statistically significantly longer in Group RD 374.3 ± 30.06 mins vs Group RC 354.9 ± 32.21 mins with p = 0.005. There was a greater 24-hour analgesia requirement in patients from Group RC (2.24 ± 0.59) compared with Group RD (2.02 ± 0.37) which is statistically significant (p = 0.033) (Table 3). T10 sensory level was achieved in all patients. However, there were more patients with dermatomal levels progressing to T6 as the highest sensory level in the Group RC group (30%) when compared to Group RD (20%) (p = 0.51) (Table 4). Hypotension was seen in 3 patients (6%) in Group RC and 1 (2%) patient in Group RD. In Group RC, 2 patients (4%) had nausea and 3 patients (6%) in Group RD had nausea with no episode of vomiting. Bradycardia was seen in 3 (6%) patients in group RC, of which 2 patients were treated with injection atropine 0.6 mg IV while group RD had only 1 (2%) case of bradycardia. Ramsay sedation scores were comparable in both groups (Figure 4). Patients of either group did not complain of dyspnoea or showed any signs of respiratory distress during the study and did not require additional oxygen in post-anaesthesia care unit. No other side effects were documented. Side effects between both groups were comparable and not statistically significant ($p >$ 0.05) (Table 5).

Table 1: Comparision of demographic data between Groups RC & RD

Parameters	Group RC (n=50) Mean ± SD	Group RD (n=50) Mean ± SD	*p*
Age (years)	39.98 ± 13.60	40.28±13.22	0.91 (NS)
Gender (M: F)	26/24	27/23	0.84 (NS)
Height (centimetre)	165.70 ± 8.08	166.76 ± 6.95	0.49 (NS)
Weight (kilogram)	56.08 ± 8.96	54.44 ± 8.70	0.34 (NS)

Values expressed in Mean ± SD, NS-Non-significant

Table 2: Comparision of duration and onset between Groups RC & RD

	Group RC	Group RD	*p*
Sensory onset (minutes)	6.00 ± 0.83	5.72 ± 0.62	0.09(NS)
Motor onset (minutes)	7.10 ± 0.86	6.95 ± 0.76	0.31(NS)
Duration of sensory blockade (minutes)	268.98 ± 39.44	288.62 ± 43.01	0.015(S)
Duration of motor blockade (minutes)	240.64 ± 39.93	260.24 ± 39.30	0.011(S)
Duration of Surgery (minutes)	103.22 ± 25.74	106.62± 23.02	0.48 (NS)
ASA (I: II)	22/28	27/23	0.32 (NS)

Values expressed in Mean ± SD, NS-Non-significant, S-significant (p <0.05)

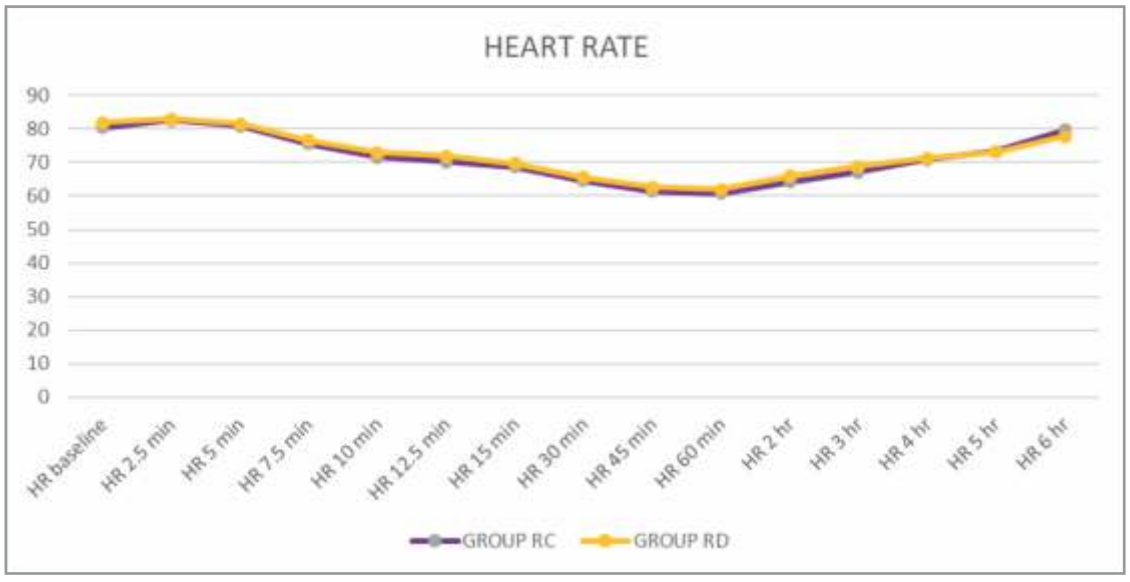

Figure 1: Comparison of heart rate between Group RC and Group RD

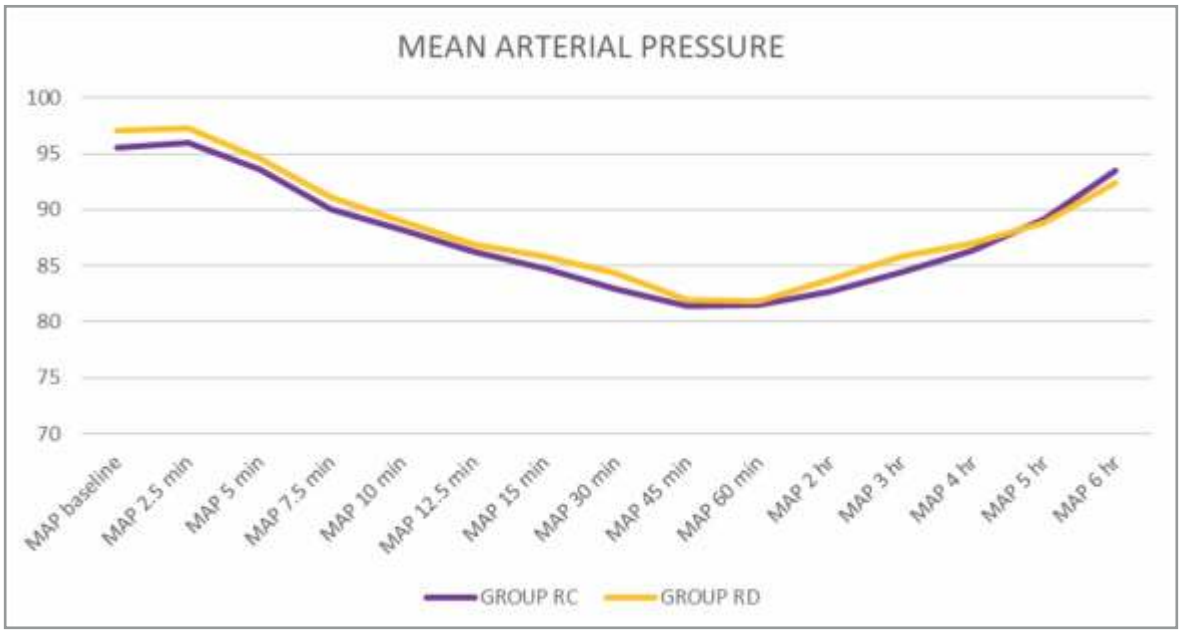

Figure 2: Comparison of mean arterial pressure between Group RC & Group RD

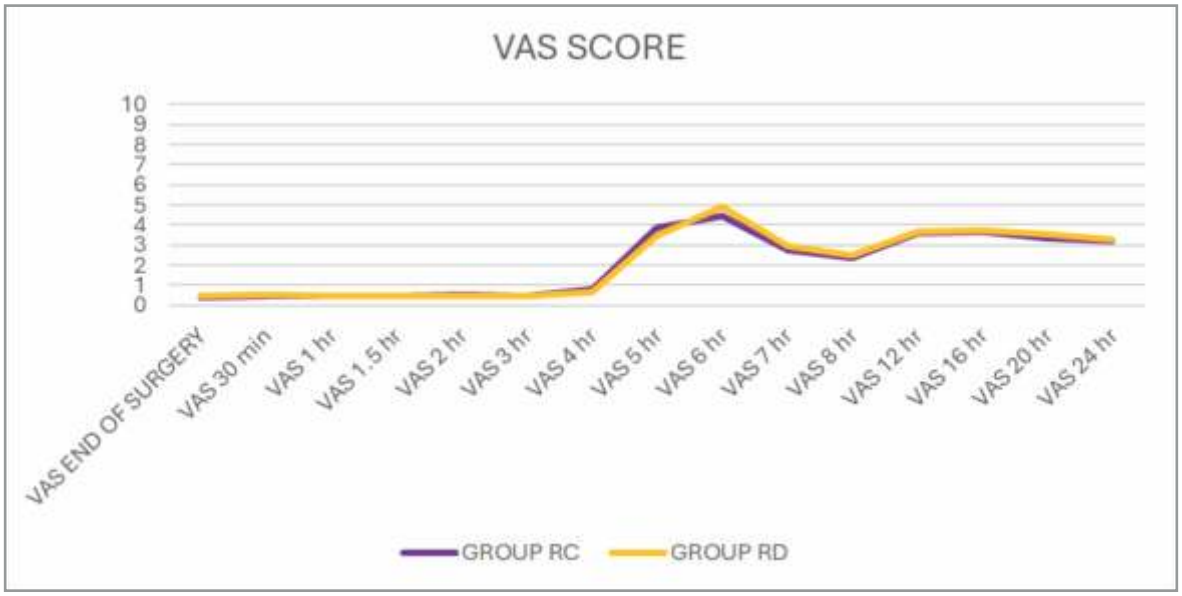

Figure 3: Comparison of visual analogue score between Group RC and Group RD

Table 3: Duration of analgesia and 24-hour analgesia requirement

	Group RC	Group RD	*p*
Duration of analgesia (minutes)	354.9 ± 32.21	374.3 ± 30.06	0.005 (S)
24- hour analgesia requirement (grams)	2.24 ± 0.59	2.02 ± 0.37	0.033 (S)

Values expressed in Mean ± SD, S-significant (p <0.05)

Table 4: Highest sensory block achieved (expressed in percentage)

Highest sensory block	Group RC	Group RD	*p*
T6	13 (26 %)	12 (24 %)	0.33 (NS)
T8	17 (34 %)	19 (38 %)	0.16 (NS)
T10	20 (40 %)	19 (38 %)	0.33 (NS)

NS-Non-significant

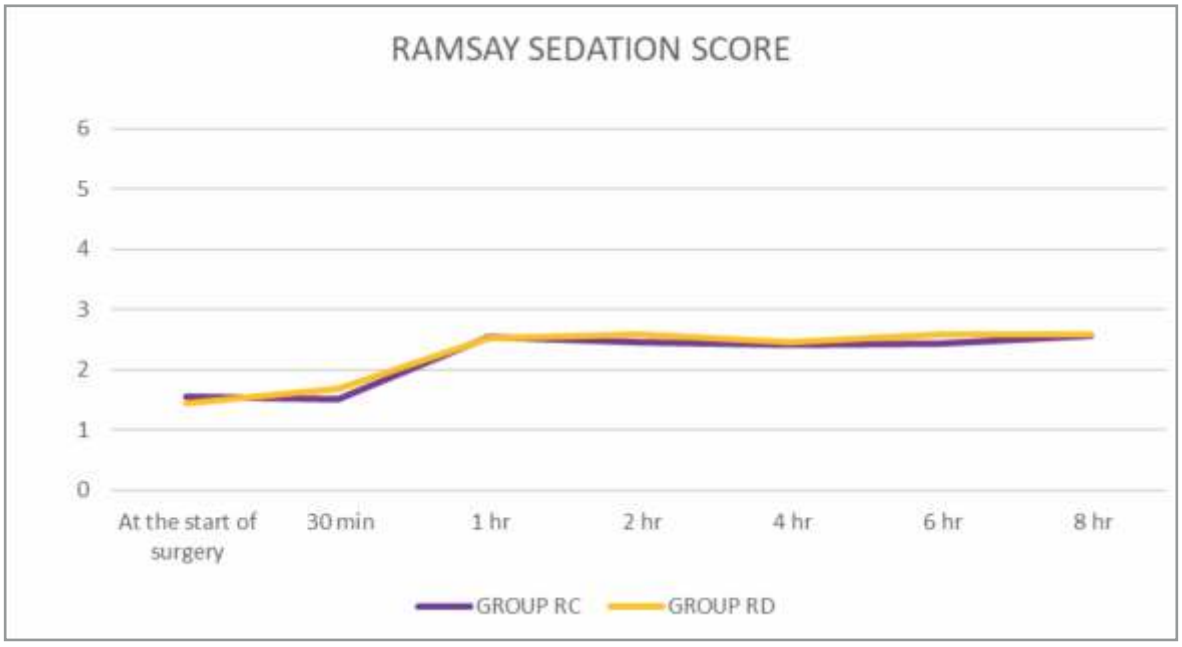

Figure 4: Comparison of Ramsay sedation score between Group RC & Group RD

Table 5: Side effects (expressed in percentage)

Side effects	Group RC	Group RD	*p*
Bradycardia	3 (6%)	1 (2%)	0.18 (NS)
Hypotension	3 (6%)	1 (2%)	0.18 (NS)
Nausea	2 (4%)	3 (6%)	0.42 (NS)
Vomiting	No	No	-
Shivering	No	No	-
Respiratory depression	No	No	-
Pruritis	No	No	-
PDPH	No	No	-
Urinary retention	No	No	-
Backache	No	No	-

NS-Non-significant

Discussion

Ropivacaine is a long-acting aminoamide that has been used as a local anaesthetic drug for local infiltration, peripheral nerve blocks, and other clinical uses since its introduction in 1996. It was introduced for intrathecal use for providing spinal anaesthesia in the European Union in February 2004 [5]. Ropivacaine has consistently demonstrated its safety, and reliability in providing spinal anaesthesia in lower limb orthopaedic surgeries [6-7]. Ropivacaine produces sufficient surgical anaesthesia with a lower incidence of side effects. However, it has a slower onset of sensory action, with a shorter extent of sensory and, motor action, including a shorter duration of pain relief after surgery in comparison to bupivacaine [8-10]. Over many years, several adjuvants have been used with local anaesthetics drugs intrathecally to prolong their analgesic effects to provide better pain relief in patients after surgery resulting in better patient satisfaction and recovery [11]. Opioids when used intrathecally as adjuvants in neuraxial blocks result in the prolongation of the effect of spinal anaesthesia and also, provide good analgesia postoperatively. However, they are associated with clinically relevant non-nociceptive dose-dependent side effects like pruritus, nausea, vomiting, mental status changes, hyperalgesia, urinary retention, and early or delayed onset of respiratory depression [12]. This limitation has resulted in renewed interest in the use of non-opioid drug adjuvants like α-2 adrenergic drugs which include clonidine and recently introduced dexmedetomidine that have comparatively fewer side effects.

Over the years various studies have been done to

study the effectiveness and safety profile of intrathecal clonidine, α-2 adrenoreceptor agonist when added to local anaesthetic drugs in neuraxial blocks [13-14]. Clonidine when given intrathecally as an adjuvant has been shown to increase the effect of sensory, and motor blockade of subarachnoid block and provides effective pain relief postoperatively. The mechanism of pain relief by intrathecal administration of clonidine is through the activation of descending noradrenergic pathways at the spinal level. It acts on postsynaptic α2 receptors, hyperpolarizes dorsal horn neurons, inhibits substance P release, and produces analgesia. Clonidine produces generalized inhibition of sympathetic nervous system activity and autonomic reflexes [15]. Clonidine, in addition, causes local vasoconstriction resulting in decreased vascular uptake of local anaesthetic around neural structures. This action results in the prolongation of local anaesthetic drug action. Neuraxial administration of clonidine also has a local effect on sympathetic nerves in the spinal cord [11]. Dexmedetomidine, a relatively new α2-adrenoreceptor agonist, is clinically used to provide intravenous sedation and as a co-analgesic drug. The anti-nociceptive action of intrathecal dexmedetomidine is due to the stimulation of α2-adrenergic receptors in the substantia gelatinosa of the dorsal horn of spinal cord which inhibit the nociceptive neurons and release of nociceptive neurotransmitters such as substance P [16].

Dexmedetomidine is considered a potent drug due to its higher α2:α1 receptors selectivity ratio of 1620:1 when compared to clonidine which has α2:α1 receptors selectivity ratio of 220:1. This high selectivity ratio for α2/α1 receptor of dexmedetomidine provide a better clinical profile like longer duration of action and lesser side effects when compared to that of clonidine [17].

Intrathecal dexmedetomidine in various studies, has been shown to provide stable hemodynamic conditions, good quality intraoperative analgesia, increased postoperative analgesic duration, decrease in 24-hour pain score, associated with decreased incidence of shivering, and minimal side effects [18]. Many researchers have used various doses of clonidine with local anaesthetics for intrathecal administration ranging from 15 μg to 300 μg and intrathecal dexmedetomidine doses ranging from 3 μg to 15 μg. Dexmedetomidine, a highly lipophilic drug has a higher binding affinity to spinal α2 adrenoreceptors around 8 to 10 times when compared with clonidine [4, 19]. Hence, considering the above binding affinity to spinal alpha receptors of both drugs, we considered the equipotent dose of intrathecal dexmedetomidine as 3 μg and intrathecal clonidine dose as 30 μg for comparison. We chose hyperbaric ropivacaine 0.75% for our study due to its better safety profile than bupivacaine. Our results showed that the duration of sensory blockade (Group RC 268.98 ± 39.44 mins vs Group RD 288.62 ± 43.01 mins) and motor blockade (Group RC 240.64 ± 39.93 mins vs Group RD 260.24 ± 39.30 mins) was significantly increased in Group RD as compared to Group RC. We also observed that the time interval of postoperative pain relief was statistically significantly longer in Group RD 374.3 ± 30.06 mins vs Group RC 354.9 ± 32.21 mins ($p < 0.05$). We observed that the VAS score was comparable in both groups. However, due to the significant prolongation of postoperative analgesia in Group RD, there was a significantly lesser 24-hour analgesia requirement in patients from Group RD (2.02 ± 0.37) when compared with Group RC (2.24 ± 0.59) with significant $p < 0.05$. The side effects in both groups were not statistically significant and comparable

with no episode of significant sedation or respiratory depression.

Ganesh *et al*. [20] compared the effect of intrathecal dexmedetomidine (3 μg) and clonidine (30 μg) added to hyperbaric bupivacaine in 150 patients operated under spinal anaesthesia. The time for the first analgesia demand by patients in the dexmedetomidine group was 366.6 ± 37.5 min which was significantly longer than clonidine group which was 344.4 ± 28.9 min. They found that sensory blockade and motor blockade duration were statistically significantly longer in the dexmedetomidine group. They concluded that using α_2-agonists as an adjuvant with hyperbaric bupivacaine intrathecally resulted in a faster onset time of both motor and sensory block along with prolongation of the postoperative analgesic effect. Kanazi *et al*. [4] added dexmedetomidine (3 mcg) or clonidine (30 mcg) to intrathecal bupivacaine and found that they provide prolongation of sensory and motor blockade, stable hemodynamic, and without significant sedation. The findings from the studies mentioned above are in concurrence with the results of our study. Our study also showed that the time interval of sensory blockade, motor blockade, and time to first rescue analgesia was statistically significantly increased with dexmedetomidine when compared to clonidine.

Ganesh *et al*. [20] and Kanazi *et al*. [4] found that the onset time of sensory and motor block was significantly faster in the dexmedetomidine (3 mcg) group in comparison with the clonidine (30 mcg) group when added to bupivacaine while Agrawal *et al*. [21] found that sensory onset in bupivacaine with dexmedetomidine 5 mcg group BD (4.87 ± 0.73), and bupivacaine with clonidine 50 mcg group BC (5.20 ± 1.40) was comparable and statistically not significant. Our study also showed that sensory onset in ropivacaine with dexmedetomidine group RD (5.72 ± 0.62 min), and ropivacaine with clonidine group RC (6.00 ± 0.83 mins) was comparable and not statistically significant. Dar *et al*. [9] observed that hyperbaric ropivacaine has a slower onset time of sensory and motor blockade in comparison with hyperbaric bupivacaine. In study done by Ganesh *et al*. [20] the sensory onset was faster in both groups (group dexmedetomidine -1.2 ± 0.4 min and group clonidine -1.4 ± 0.5 min) when compared to the time of sensory onset seen in our study. This may be attributed to their use of heavy bupivacaine along with a larger volume (4 ml) for giving spinal anaesthesia.

Sharan *et al*. [22] compared intrathecal clonidine (30 mcg) with intrathecal fentanyl (25 mcg) added to ropivacaine in patients undergoing lower abdominal surgeries under subarachnoid block. They found that clonidine significantly increased the duration of the subarachnoid block and the postoperative analgesia when compared with fentanyl. They also noted that the occurrence of episodes of bradycardia and hypotension in patients was more common in the clonidine group. In our study, we had similar observations where hypotension was observed in 3 patients (6%) in group RC and 1 patient (2%) in group RD while bradycardia was observed in 3 patients (6%) in group RC, group RD had only 1 case (2%) of bradycardia. These side effects among groups were comparable and statistically non-significant. In our study, we observed no significant sedation, or any episodes of respiratory depression seen in patients in either group. Dexmedetomidine provides sedation which is like normal physiological sleep with no suppression of respiratory function and the patients can wake up at any time.

This quality of sedation of dexmedetomidine may become advantageous over clonidine when higher doses are used. Bajwa *et al*. [23] in their study used clonidine (2 mcg/kg) and dexmedetomidine (1 mcg/kg) epidurally and found sedation scores with dexmedetomidine were better than clonidine and statistically significant ($p < 0.05$). The α2-agonist drugs when administered intrathecally have a dose-dependent sedative effect. We administered low doses of intrathecal dexmedetomidine and intrathecal clonidine which may have resulted in a comparable sedation score and no significant side effects.

Sharma *et al*. [24] studied the effect of intravenous dexmedetomidine on 120 patients undergoing infraumbilical surgeries under spinal anaesthesia with hyperbaric bupivacaine 0.5%. They concluded that a bolus of 1 mcg/kg over 10 minutes followed by an infusion of 0.5 mcg/kg/hr immediately after neuraxial anaesthesia accelerated the sensory onset, prolonged the duration of analgesia and increased the time for 2 segment regression.

Singh *et al*. [25] conducted a comparative study evaluating dexmedetomidine and clonidine as adjuvants to 0.5% ropivacaine in supraclavicular brachial plexus block. The study included a sample size of 30 patients per group, with clonidine administered at 150 mcg and dexmedetomidine at 100 mcg. The findings indicated that both adjuvants accelerated the onset of sensory and motor blockade while prolonging analgesia. However, dexmedetomidine was identified as the superior option due to its faster onset of sensory and motor blockade and a higher rate of complete blockade compared to clonidine.

The limitation of this study was in terms of the small sample size. We suggest a multicentre study with a larger sample size with a similar type of injury and pain scale/intensity to conclude an effective dose. Further studies can be done to include ASA III and IV patients for the efficacy of intrathecal alpha 2 agonists as adjuvants.

Conclusion

This study concludes that the addition of α-2-adrenergic receptor agonists intrathecally to hyperbaric ropivacaine significantly increased postoperative analgesia and duration of sensory and motor blockade. We recommend the use of intrathecal dexmedetomidine as an adjuvant to ropivacaine in subarachnoid block to provide better postoperative pain relief with minimal side effects.

Acknowledgements

We thank all the patients for their valuable support.

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How to cite this article:

Mehta S, Deshpande J, Singhi HB, Pathan A. A comparative study to evaluate the efficacy of intrathecal dexmedetomidine and intrathecal clonidine as an adjuvant to hyperbaric 0.75% ropivacaine in patients undergoing orthopedic surgery under spinal anaesthesia. *J Krishna Inst Med Sci Univ* 2025; 14(1):20-31.

***Submitted:** 28-Sep-2024 **Accepted:** 10-Dec-2024 **Published:** 01-Jan-2025*