

EFFECT OF DEXMEDETOMIDINE AS AN ADJUVANT TO 0.75% ROPIVACAINE VERSUS CLONIDINE AS A ADJUVANT TO 0.75% ROPIVACAINE IN INTERSCALENE BLOCK -A CLINICAL COMPARISON

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Abstract

Background: To determine the analgesic efficacy of dexmedetomidine and clonidine as an adjuvant to ropivacaine in interscalene block in patients undergoing elective upper limb surgeries. The current study was conducted as a prospective randomised controlled clinical trial after receiving institutional ethical committee approval and informed consent from patients. Onset of sensory block, Onset of motor block, Duration of sensory block, Duration of motor block, and Patient satisfaction score are the variables evaluated. **Materials and Methods:** Using local anesthetic agents with injection ropivacaine 0.75% and adjuvants clonidine and dexmedetomidine, the interscalene brachial plexus block was performed on 90 patients of either sex from June 2019 to December 2021 in the Department of Anaesthesiology and Critical Care at Kurnool Medical College, Kurnool. The study was started after receiving approval from the ethical committee. **Results:** Group 1 (plain ropivacaine) patients had sensory block at 13.5 ± 1.50 minutes, group 2 (ropivacaine plus clonidine) at 12.9 ± 1.57 minutes, and group 3 (ropivacaine plus dexmedetomidine) at 10.3 ± 1.60 minutes. Mean time of start of motor block of the patients in group 1 was 16.9 ± 1.27 minutes, group 2 was 16.4 ± 1.22 minutes and group 3 was 14.9 ± 1.47 minutes. Group 1 had 409.2 ± 26.03 minutes of analgesia, group 2 598.5 ± 21.10 minutes, and group 3 720.3 ± 34.41 minutes. In group 1, 21 patients (70%) have taken 3 rescue analgesics and 9 (30%) have taken 2. In group 2, 22 (73.3%) patients took 2 doses of rescue analgesia, 6 (20%) took 1, and 2 (6.7%) took 3. Group 1 patients took 3 rescue analgesic dosages more often ($p < 0.05$). **Conclusion:** The current investigation it was discovered that combination of clonidine and dexmedetomidine to 0.75% ropivacaine produced early start of sensory and motor block in interscalene brachial plexus block. Dexmedetomidine acted faster and longer than clonidine. Dexmedetomidine provides early onset and persistent sensory and motor block and surgical analgesia is better than that with clonidine.

INTRODUCTION

Perhaps the most dreaded sign of a disease, pain is something that man has tried to eliminate and master throughout history. By definition, according to the International Association for the Study of Pain, pain is "an unpleasant sensory and emotional experience related to actual or potential tissue damage or described in terms of such damage." In

order to provide both prolonged postoperative analgesia and intraoperative anesthesia, the peripheral nerve block,^[1] has grown in popularity. Additionally, it provides patients with ease by shortening their stay in the hospital, easing their financial burden, and helping them avoid the negative effects of general anesthesia.^[1] Regional anesthesia has entered a brand-new era of advanced techniques over the last few decades. With

the aid of long-acting local anesthetics, more modern adjuvants, nerve locators, and USG guidance.^[2], the block is made safe and effective. Brachial plexus block is a safe and dependable anesthetic technique that offers a satisfactory surgical condition with a full motor and sensory block.^[3]

The insertion of a peripheral nerve catheter or the addition of an adjuvant to local anesthetics are just two examples of the many techniques used to enhance the quality of the block and lengthen the duration of analgesia. We prefer alpha2 agonists as an adjuvant to ropivacaine for interscalene block since there are more complications with perineural catheter insertion.^[4] The sedative, analgesic, antihypertensive, and antiemetic properties of alpha2 agonists.^[5], as well as their reduced need for local anesthetic medications, led to their selection as an adjuvant. In peripheral nerve blocks, the alpha2 agonist clonidine.^[6] has been shown to extend the time of anesthesia and analgesia.

It has also been demonstrated that dexmedetomidine.^[7], a selective alpha2 agonist with an alpha2 affinity eight times greater than that of clonidine, can lengthen the time that sensory and motor blockade lasts when added as an adjuvant to local anesthetic in peripheral nerve blocks. Dexmedetomidine and clonidine were added to ropivacaine for interscalene brachial plexus block, and the results of this controlled, randomised, double-blind clinical study were compared.

MATERIALS AND METHODS

90 patients of either sex underwent interscalene brachial plexus block as part of the current study, which was conducted in the Department of Anaesthesiology and Critical Care at Kurnool Medical College, Kurnool, from June 2019 to December 2021. Local anesthetic agents with injection ropivacaine 0.75% and adjuvants clonidine

and dexmedetomidine were used. Obtaining approval from the ethical committee allowed the study to proceed.

Groups

All of the patients were divided into three groups at random, with 30 patients in each group.

GROUP-1: 29ml of 0.75% Inj. Ropivacaine hydrochloride with 1ml normal saline.

GROUP- 2: 29ml of 0.75% ropivacaine + 1microgram/kg of. clonidine

GROUP-3: 29ml of 0.75% ropivacaine+1 microgram/kg of dexmedetomidine

Inclusion Criteria

- ASA grade I and grade II patients undergoing orthopaedic upper limb surgeries between the ages of 18 and 60, of both sexes, will be included.
- Patients with signed consent forms.

Exclusion Criteria

- ASA Grade-III and IV of a patient in a high-risk group. disorders of bleeding.
- liver diseases, renal diseases, cardiovascular diseases, and respiratory diseases. irregular heartbeat.
- Patients with known local anesthetic sensitivity patients who are morbidly obese.
- Patients who have suffered damage to any of the upper limb's nerves, infection present at the block's location.
- Patients rejection.

RESULTS

The analgesic efficacy of dexmedetomidine and clonidine as an adjuvant to ropivacaine in the interscalene block in patients undergoing elective upper limb surgeries was evaluated in a total of 90 patients. Thirty patients in each of 3 group. The study's findings are.

Table 1: Distribution of the groups by mean age (in years)

Groups	Mean age	Standard deviation	p-value
Group 1	47.3	6.59	0.433; NS
Group 2	45.9	7.22	
Group 3	48.1	6.05	

NS = Not Significant

The mean age of the patients in group 1 was 47.3±6.59 years, patients in group 2 was 45.9±7.22 years and patients in group 3 was 48.1±6.05 years. The difference in the mean age between the groups is not significant statistically. (Anova value F=0.844; p>0.05; Not significant).

Table 2: Distribution of the groups by Gender

Groups	Male		Female		Total
	Number	Percentage	Number	Percentage	
Group 1	21	70%	9	30%	30 (100%)
Group 2	23	76.7%	7	23.3%	30 (100%)
Group 3	20	66.7%	10	33.3%	30 (100%)

□² = 0.757; df = 2; p = 0.685; Not significant

In group 1, around 21 (70%) patients are males and 9 (30%) patients are females. In group 2, around 23 (76.7%) patients are males and 7 (23.3%) patients are females. In group 3, around 20 (66.7%) patients are males and 10 (33.3%) patients are females. There is no significant difference between the groups with respect to proportion of male patients and female patients. ($p>0.05$; Not significant).

Table 3: Distribution of the groups by mean weight (in kilograms)

Groups	Mean weight	Standard deviation	p-value
Group 1	68.3	8.56	0.693; NS
Group 2	67.3	8.75	
Group 3	66.4	8.41	

The mean weight of the patients in group 1 was 68.3 ± 8.56 kilograms, patients in group 2 was 67.3 ± 8.75 kilograms and patients in group 3 was 66.4 ± 8.41 kilograms. The difference in the mean weight between the groups is not significant statistically. (Anova value $F=0.369$; $p>0.05$; Not significant).

Table 4: Distribution of the groups by ASA grading

Groups	ASA grade I		ASA grade II		Total
	Number	Percentage	Number	Percentage	
Group 1	17	56.7%	13	43.3%	30 (100%)
Group 2	19	63.3%	11	36.7%	30 (100%)
Group 3	15	50%	15	50%	30 (100%)

$\chi^2 = 1.086$; $df = 2$; $p = 0.581$; Not significant

In group 1, around 17 (56.7%) patients were in ASA grade I and 13 (43.3%) patients were in ASA grade II. In group 2, around 19 (63.3%) patients were in ASA grade I and 11 (36.7%) patients were in ASA grade II. In group 3, around 15 (50%) patients were in ASA grade I and 15 (50%) patients were in ASA grade II. There is no significant difference between the groups with respect to ASA grading. ($p>0.05$; Not significant).

Table 5: Distribution of groups by mean heart rate

Mean heart rate at	Group 1	Group 2	Group 3	p-value
Baseline	87.8 ± 6.66	89 ± 6.2	91 ± 6.4	0.156 (NS)
5 mins	95.9 ± 7.05	95.1 ± 6.19	97.3 ± 6.42	0.425 (NS)
10 mins	86.1 ± 7.81	85.1 ± 6.19	87.3 ± 6.42	0.463 (NS)
15 mins	82.5 ± 6.63	83.1 ± 6.19	85.3 ± 6.42	0.211 (NS)
20 mins	78.3 ± 6.42	79.1 ± 6.19	81 ± 6.4	0.243 (NS)
25 mins	77.2 ± 7.15	76.1 ± 6.19	78 ± 6.4	0.536 (NS)
30 mins	73.2 ± 6.32	74 ± 6.2	76 ± 6.4	0.214 (NS)
45 mins	84.1 ± 7.21	85 ± 6.2	87 ± 6.4	0.227 (NS)
60 mins	86.3 ± 6.34	87 ± 6.2	89.3 ± 6.42	0.163 (NS)
90 mins	89.1 ± 6.33	90 ± 6.2	92 ± 6.4	0.196 (NS)
120 mins	95.9 ± 6.99	97.1 ± 6.19	99.3 ± 6.42	0.131 (NS)
150 mins	97.4 ± 6.57	98.1 ± 6.19	100 ± 6.42	0.270 (NS)

The average heart rate varies over time equally in all three groups. The difference between the groups was statistically insignificant at any time ($p>0.05$).

Table 6: Distribution of the groups by mean systolic blood pressure (SBP)

Mean SBP	Group 1	Group 2	Group 3	p-value
Baseline	123 ± 4.57	122 ± 5.14	120 ± 7.4	0.134 (NS)
5 mins	125 ± 5.12	125 ± 5.6	124 ± 7.6	0.772 (NS)
10 mins	112 ± 7.1	111 ± 6.6	110 ± 7.4	0.548 (NS)
15 mins	111 ± 6.4	109 ± 6.6	108 ± 7.2	0.220 (NS)
20 mins	104 ± 6.39	103 ± 6.7	102 ± 7.4	0.529 (NS)
25 mins	99 ± 7.4	98 ± 8.8	96 ± 7.1	0.321 (NS)
30 mins	94 ± 7.3	93 ± 8.9	92 ± 7.4	0.620 (NS)
45 mins	96 ± 7.00	95 ± 8.3	94 ± 7.4	0.596 (NS)
60 mins	102 ± 7.35	101 ± 8.0	99 ± 7.3	0.299 (NS)
90 mins	111 ± 6.6	110 ± 7.4	109 ± 7.4	0.558 (NS)
120 mins	113 ± 6.4	112 ± 7	111 ± 6.4	0.506 (NS)
150 mins	116 ± 6.8	115 ± 6.43	113 ± 7.4	0.234 (NS)

Mean systolic blood pressure fluctuates over time similarly in each of the three groups. The statistical differences between the groups were insignificant at all times. ($p>0.05$).

Table 7: Distribution of the groups by mean diastolic blood pressure (DBP)

Mean DBP	Group 1	Group 2	Group 3	p-value
Baseline	84±8.8	83±6.1	82±7.92	0.604 (NS)
5 mins	86±8.9	85±7.9	84±6.5	0.615 (NS)
10 mins	77±7.9	78±8.1	76±7.9	0.625 (NS)
15 mins	75±7.6	76±7.4	73±5.9	0.246 (NS)
20 mins	69±8	70±7.8	66±7.9	0.131 (NS)
25 mins	65±8.4	66±7.9	63±7.3	0.329 (NS)
30 mins	63±7.9	64±8.1	61±7.8	0.334 (NS)
45 mins	65±7.9	66±7.9	63±7.2	0.310 (NS)
60 mins	70±8.1	71±8.0	68±7.2	0.319 (NS)
90 mins	75±7.9	74±8.1	73±7.3	0.610 (NS)
120 mins	77±7.9	76±7.4	75±5.9	0.555 (NS)
150 mins	78±8.1	77±8.0	75±7.6	0.331 (NS)

Mean diastolic blood pressure varies over time similarly in all three groups. The difference between the groups was never statistically significant ($p > 0.05$) at any point in time.

Table 8: Distribution of the groups by mean oxygen saturation (SpO₂)

SpO ₂	Group 1	Group 2	Group 3	p-value
Baseline	97.5±0.97	97.7±1.06	97.7±0.92	0.663 (NS)
5 mins	97.8±0.97	97.7±0.92	97.7±0.94	0.894 (NS)
10 mins	97.6±0.93	97.5±1.01	97.5±0.97	0.899 (NS)
15 mins	97.7±0.92	97.9±0.97	97.8±0.97	0.720 (NS)
20 mins	97.7±0.94	97.7±1.03	97.6±0.96	0.901 (NS)
25 mins	97.6±0.96	97.6±0.93	97.5±1.01	0.899 (NS)
30 mins	97.5±1.01	97.7±0.92	97.7±1.06	0.671 (NS)
45 mins	97.7±0.92	97.7±0.94	97.7±0.92	1.000 (NS)
60 mins	97.5±1.01	97.5±0.97	97.5±1.01	1.000 (NS)
90 mins	97.9±0.97	97.8±0.97	97.9±0.97	0.899 (NS)
120 mins	97.7±1.03	97.6±0.96	97.7±1.03	0.906 (NS)
150 mins	97.7±1.06	97.5±1.01	97.6±0.93	0.742 (NS)

In all the three groups mean oxygen saturation changes equally with time. At any point of time the difference between the groups were not significant statistically. ($p > 0.05$).

Table 9: Distribution of the groups by onset of sensory block (minutes)

Time of onset of sensory block (minutes)				
Groups	Mean	Standard deviation	p-value	
Group 1	13.5	1.50	<0.001 (S)	
Group 2	12.9	1.57		
Group 3	10.3	1.60		

The mean time for the onset of sensory block in group 1 patients was 13.5 minutes, group 2 patients were 12.9 minutes, and group 3 patients were 10.3 minutes. In comparison to group 1 and group 2, patients in group 3 experienced sensory block significantly sooner (ANOVA value $F=35.794$; $p < 0.05$; Significant).

Between groups 1 and 2, there was no statistically significant difference in the mean time for the onset of sensory block. Not significantly ($p=0.136$) However, the difference between groups 1 and 3 as well as groups 2 and 3 was statistically significant ($p < 0.001$; Significant).

Table 10: Distribution of the groups by onset of motor block (minutes)

Time of onset of motor block (minutes)				
Groups	Mean	Standard deviation	p-value	
Group 1	16.9	1.27	<0.001 (S)	
Group 2	16.4	1.22		
Group 3	14.9	1.47		

The patients in groups 1, 2 and 3 had a mean time of onset of motor block of 16.9 (standard deviation 1.27 minutes), 16.4 (standard deviation 1.22 minutes), and 14.9 (standard deviation 1.47 minutes), respectively. When compared to patients in groups 1 and 2, group 3 patients had a noticeably quicker onset of motor block. $F=18.528$ in the ANOVA; $p < 0.05$; Significant.

Between groups 1 and 2, there was no statistically significant difference in the mean time for the onset of motor block. Unimportant ($p=0.125$) But there was a statistically significant difference between groups 1 and 3, as well as between groups 2 and 3. Significant at ($p < 0.001$).

Table 11: Distribution of the groups by duration of sensory block (minutes)

Duration of sensory block (minutes)				
Groups	Mean	Standard deviation	p-value	
Group 1	388.5	32.46	<0.001 (S)	
Group 2	515.3	35.89		
Group 3	685.3	36.27		

Group 1 patients' sensory block lasted, on average, 388.5 32.46 minutes; group 2 patients' lasted, on average, 515.3 35.89 minutes; and group 3 patients lasted, on average, 685.3 36.27 minutes. The group 3 patients had the longest sensory block duration, followed by group 2 patients, and group 1 patients had the shortest. F=545.77 in the ANOVA; p 0.05; Significant.

There is a statistically significant difference in the mean duration of sensory block between groups 1 and 2, groups 1 and 3, and groups 2 and 3 (p0.001; Significant).

Table 12: Distribution of the groups by duration of motor block (minutes)

Duration of motor block (minutes)				
Groups	Mean	Standard deviation	p-value	
Group 1	301.5	28.38	<0.001 (S)	
Group 2	475.7	25.28		
Group 3	663.7	28.34		

The patients in groups 1, 2, and 3 experienced motor blocks for a mean of 301.5 minutes, (SD -28.38 minutes), 475.7 minutes, (SD-25.28 minutes) and 663.7 minutes (SD - 28.34min) respectively. After group 2 patients and group 1 patients, group 3 patients had the longest duration of motor block. Group 2 patients had the second-shortest duration. (F=1313.9; p 0.05; Significant; ANOVA value).

It is statistically significant (p 0.001; Significant) that group 3 have longer mean motor block durations than group 1 and group 2.

Table 13: Distribution of the groups by duration of analgesia (minutes)

Duration of analgesia (minutes)				
Groups	Mean	Standard deviation	p-value	
Group 1	409.2	26.03	<0.001 (S)	
Group 2	598.5	21.10		
Group 3	720.3	34.41		

Mean duration of analgesia in group 1 was 409.2 ± 26.03 minutes, group 2 was 598.5 ± 21.10 minutes and group 3 was 720.3 ± 34.41 minutes. Duration of analgesia was significantly higher in group 3 patients, followed by group 2 patients and least for group 1 patients. (ANOVA value F=958.8; p<0.05; Significant).

Significant statistically (p 0.001; Significant) is the difference in the mean duration of analgesia between groups 1, 2, and 3, as well as between groups 2 and 3.

Table 14: Distribution of the groups by number of doses of rescue analgesia

n number of doses of rescue analgesia	Group 1	Group 2	Group 3
0 Doses	0 (0%)	0 (0%)	4 (13.3%)
1 Dose	0 (0%)	6 (20%)	26 (86.7%)
2 Doses	9 (30%)	22 (73.3%)	0 (0%)
3 Doses	21 (70%)	2 (6.7%)	0 (0%)

² value with Yate's correction= 89.663; df = 6; p<0.001; Significant

In group 1, around 21 (70%) patients have taken 3 doses of rescue analgesia and remaining 9 (30%) patients have taken 2 doses of rescue analgesia. In group 2, around 22 (73.3%) patients have taken 2 doses of rescue analgesia, 6 (20%) patients have taken 1 dose of rescue analgesia and only 2 (6.7%) patients have taken 3 doses of rescue analgesia. In group 3, around 26 (86.7%) patients have taken 1 dose of rescue analgesia and remaining 4 (13.3%) didn't receive any dose of rescue analgesia. Significantly higher number of patients in group 1 has taken 3 doses of rescue analgesia (p<0.05; Significant).

DISCUSSION

Most anesthesiologists base their use of regional anesthesia on brachial plexus blockade. Brachial plexus block results in anaesthesia that is restricted to a specific area of the body, does not interfere with the metabolism of the rest of the body, early patient ambulation, and early discharge. The procedures

around the shoulder, upper arm, and forearm are the typical indications for interscalene brachial plexus block.

Long-acting local anesthetics like ropivacaine or bupivacaine are used to block nerves, which is beneficial for postoperative pain management. However, the block's duration is still insufficient to prevent the need for opioids after surgery.

Alternatively, peripheral nerve catheters can be used to extend the duration of analgesia, but this is more time-consuming, expensive, and difficult than using single-shot blocks.

Numerous adjuvants have been added to local anesthetics when used in brachial plexus block to enhance the quality of the block and lengthen the duration of postoperative analgesia.^[8] These adjuvants include epinephrine, clonidine, dexmedetomidine, opioids, bicarbonate, neostigmine, verapamil, and butorphenol. Adjuvants not only increase the effectiveness of the block but also lengthen its duration, reducing the need for continuous perineural catheters and post-operative analgesics.

The mean duration of the sensory and motor blocks in groups 2 and 3 of this study both significantly increased. Dexmedetomidine was added, and when compared to the clonidine and control group, the increase in duration was statistically found to be highly significant. As previously reported by Kenan Kaygusuz MD et al.^[9] and Singelyn FJ et al.^[10], this prolonged duration of sensory and motor blockade following the addition of clonidine and dexmedetomidine to local anesthetics in peripheral nerve blocks has also been observed in other studies. Even though some studies found no benefit to adding clonidine to local anesthetics in terms of lengthening the duration of the block, Erlacher W et al.^[11] discovered that adding clonidine to ropivacaine 0.75% did not result in any benefit in terms of blocking the brachial plexus when compared with pure ropivacaine 0.75%. However, the majority of studies note benefits with regard to block quality and duration. Adding a small dose (30mcg) of clonidine to 0.5% bupivacaine significantly extended the duration of analgesia, according to Chakraborty S et al.^[12] without causing any clinically significant adverse reactions other than sedation. Dexmedetomidine use in group III led to a quicker onset of sensory and motor block. The hyperpolarization activated cation current, which is necessary to return a peripheral nerve to its resting potential, is blocked, which causes the block to manifest more quickly.^[13] The use of dexmedetomidine with local anesthetics has sped up the onset time of sensory and motor block, but the role of clonidine as an adjuvant to ropivacaine in faster onset of block is debatable. The majority of prior studies showed no effect on block onset.

Adjuvants dexmedetomidine and clonidine significantly reduced the need for analgesics during the postoperative period. For this, there have been four suggested mechanisms: - analgesia mediated centrally, vasoconstrictive effects mediated by 2 adrenoceptors, attenuation of inflammatory response, and direct action on peripheral nerve.^[15,16] By using alfa agonists to block the conduction of C and A fibers and increase potassium conductance, analgesia was prolonged after neural blockade.^[17] Additionally, clonidine increases the inhibition of the C-fiber compound action potential caused by

lignocaine. Clonidine's lipophilic nature allows for quick absorption into the cerebrospinal fluid and binding to the spinal cord's adrenoceptors, which blocks both spinal and peripheral nerve endings' primary afferent terminals.^[18] Alpha 2 agonists act centrally to produce analgesia and sedation by activating alpha 2 adrenoceptors in the locus coeruleus and inhibiting substance P release in the nociceptive pathway at the level of the dorsal root neuron.^[19]

At any of the measured intervals in any of the three groups, there was no clinical or statistical change in the arterial saturation (SPO2) or respiratory rate. Kenan Kaygusuz MD et al. and Singh S et al.^[20] are two examples. In addition, clonidine and dexmedetomidine were used as adjuvants to local anesthetics at a dose of 1mcg/kg body weight, with no appreciable differences in the ventilatory frequency or oxygen saturation. The recorded haemodynamic parameters (heart rate, mean blood pressure) did show a statistically insignificant difference between the three groups, with clonidine and dexmedetomidine resulting in a lower heart rate and blood pressure, but since the difference was insignificant, no pharmacological intervention was required.

Demographic Data

The demographic characteristics of the three groups in the current study, 1, 2, and 3, were comparable, and they did not statistically differ from one another. Our patients' demographics between these groups were statistically unremarkable ($P > 0.05$), and they matched those of Kanvee V. and Patel K.'s study.^[21] quite closely.

Mean Onset of Sensory Block

In our investigation Patients in groups 1, 2, and 3 experienced sensory block on average within 13.5, 12.9, and 10.3 minutes, respectively.

When compared to patients in groups 1 and 2, the onset of sensory block occurred significantly more quickly in group 3 patients. ($p < 0.05$; Significant).

Between group R and group 2, there was no statistically significant difference in the mean time for the onset of sensory block. Not significantly ($p=0.136$) However, the difference between groups 1 and 3 as well as groups 2 and 3 was statistically significant ($p < 0.001$; Significant).

Similar findings were found in a study by Dubey S., Najeeb R., and Sofi A.A.^[22] Esmoglu et al.^[23] conducted a comparison of the effectiveness of clonidine and dexmedetomidine as an adjuvant to ropivacaine in an ultrasound-guided supraclavicular brachial plexus block and the timing of the onset of surgical anesthesia and the duration of the effect. Dexmedetomidine was added to levobupivacaine for axillary brachial plexus block, and it was demonstrated that this shortened the time it took for both sensory and motor block to begin, increased block duration, and prolonged post-operative analgesia.

Dexmedetomidine is an adjuvant that can be used with levobupivacaine to increase the duration and

shorten the onset of sensory and motor block, according to research by Biswas et al.^[24]

Using a nerve stimulator, H. D. Rashmi and H. K. Komala.^[25], investigated the effects of dexmedetomidine as an adjuvant to 0.75% ropivacaine in interscalene brachial plexus block. They came to the conclusion that this combination significantly sped up the onset of the block and prolonged the duration of the sensory and motor blockade.

In their study of ultrasound-guided single injection infraclavicular brachial plexus blocks using bupivacaine alone or in combination with dexmedetomidine for pain management in upper limb surgery, Amany S. Ammar and Khaled M. Mahmoud.^[26] discovered that the addition of dexmedetomidine to bupivacaine during the placement of an ICB increases the onset of sensory and motor blockade.

Mean Onset of Motor Blockade

In our study, the mean motor block onset time for patients in groups 1 and 2 was 16.9 ± 1.27 minutes, 16.4 ± 1.22 minutes, and 14.9 ± 1.47 minutes, respectively. When compared to patients in groups 1 and 2, the onset of motor block was noticeably quicker in group 3 patients. (F=18.528; p<0.05; Significant; ANOVA value).

The statistical difference between group 1 and group 2's mean times for the onset of motor block was insignificant. Not significantly (p=0.125) However, there was a statistically significant difference between groups 1 and 3, as well as between groups 2 and 3 (p < 0.001; Significant).

Eighty patients scheduled for elective forearm and hand surgeries were evaluated by Feroz Ahmad Dar, Mohd Rafiq Najar, and Neelofar Jan.^[27] who looked at the effects of adding dexmedetomidine to ropivacaine for axillary brachial plexus blockade. The addition of dexmedetomidine resulted in shorter sensory and motor block onset times, longer sensory and motor blockade durations, and longer analgesic durations.

Kavitha Jinjal et al. (2015) made comparable observations.^[28] They found that, compared to clonidine, the addition of dexmedetomidine to the local anesthetic solution accelerated the onset of motor block. This difference was statistically significant.

MEAN DURATION OF SENSORY BLOCK (DURATION OF ANALGESIA)

In our study, the median time for a patient to experience a sensory block after receiving ropivacaine was 388.5 ± 32.46 minutes, compared to 515.3 ± 35.89 minutes for group 2 and 685.3 ± 36.27 minutes for group 3. The group 3 patients had the longest sensory block duration, followed by group 2 patients, and group 1 patients had the shortest.

Similar findings were found in a study conducted by Kanvee V. and Patel K.^[21] that compared clonidine and dexmedetomidine as an adjuvant to ropivacaine in supraclavicular brachial plexus block and came to the conclusion that dexmedetomidine, when added

to ropivacaine in brachial plexus block, has significantly longer duration of sensory and motor Blockade and duration of post operative analgesia.

After comparing dexmedetomidine and clonidine (both 2 agonist drugs) as an adjuvant to local anesthesia in supraclavicular brachial plexus block, Sarita S. Swami et al.^[29] came to the conclusion that dexmedetomidine, when added to local anesthesia in supraclavicular brachial plexus block, increased the duration of sensory and motor block as well as the duration of analgesia. Patients receiving dexmedetomidine experienced a longer duration of rescue analgesia. Compared to clonidine, it improved the block's quality as well.

In a comparison study of the effects of additional Alpha 2 agonists and local anesthetic in infraclavicular brachial plexus block, R Sreeja et al.^[30] discovered that the dexmedetomidine group offers a more rapid and prolonged analgesic action without significantly negative side effects.

Dexmedetomidine prolongs the effect of bupivacaine in supraclavicular brachial plexus block, according to the findings of Agarwal et al.^[31]

Mean Duration of Motor Blockade

Archana Tripathi et al.^[32], conducted a comparison study of dexmedetomidine and clonidine as an adjunct to bupivacaine in supraclavicular brachial plexus block and came to the conclusion that dexmedetomidine, when added to clonidine, improves the quality of anesthesia and prolongs the durations of sensory and motor block and duration of analgesia.

In a study by D. Marhofer et al.^[33], dexmedetomidine was used as an adjuvant to ropivacaine in the treatment of ulnar nerve block, and the researchers discovered that this significantly increased the duration of the motor block and sped up its onset.

Dexmedetomidine is a superior alternative to clonidine as an adjuvant for 0.5% ropivacaine in order to achieve early onset and prolong the duration of sensory and motor block as well as postoperative analgesia, according to research by Ovais Nazir, Asif Hussain Bhat, Tarun Sharma, Amit Khatuja, and Rajesh Misra.^[34]

VAS and RESCUE ANALGESIA

Around 21 (70%) of the patients in group 1 received 3 doses of rescue analgesia, while the remaining 9 (30%) received 2 doses. In group 2, only 2 (6.7%) patients received 3 doses of rescue analgesia, compared to 22 (73.3%) patients who received 2 doses, 6 (20%) patients, and 22 (73.3%) patients who received 1 dose. In group 3, approximately 26 (86.7%) patients received one dose of rescue analgesia, while the remaining 4 (13.3%) did not receive any dose. Three doses of rescue analgesia were taken by a significantly greater percentage of patients in group 1 (p < 0.05; significant).

HEMODYNAMIC CHANGES

In our study, the three groups' mean oxygen saturation, pulse rate, and systolic and diastolic blood pressure all changed equally over time. The

statistical differences between the groups were not present at any point in time. ($p>0.05$).

The Sebastian D., Ravi M., Dinesh K., et al. study.^[35], which compared the use of clonidine versus dexmedetomidine as an adjuvant to ropivacaine in supraclavicular brachial plexus block, was consistent with this study.

CONCLUSION

As a result, it was discovered in the current study that adding clonidine and dexmedetomidine to 0.75% ropivacaine caused an early onset of sensory and motor block in the interscalene brachial plexus block. Compared to clonidine, dexmedetomidine had an earlier onset and a longer duration of action. In order to achieve early onset, prolonged duration of sensory and motor block, and postoperative analgesia, dexmedetomidine is therefore thought to be a superior alternative to clonidine.

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