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Original Research Article

TO INVESTIGATE THE IMPACT OF INTRATHECAL ADMINISTRATION OF DEXMEDETOMIDINE AND CLONIDINE AS ADJUVANTS AGENTS TO 0.5% HYPERBARIC BUPIVACAINE DURING SPINAL ANAESTHESIA: A COMPARATIVE STUDY

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Abstract

Background: To investigate the impact of intrathecal administration of dexmedetomidine and clonidine as adjuvants agents to 0.5% hyperbaric bupivacaine during spinal anaesthesia: A comparative study. **Materials and Methods:** 50 patients who were scheduled to elective urological procedures under spinal anaesthetic were included in this study. This study aimed to investigate the impact of intrathecal administration of clonidine (30 micrograms) and dexmedetomidine (5 micrograms) on various parameters, including the time of onset, peak effect, and duration of sensory and motor block, as well as the hemodynamic effects. Additionally, the study examined the duration of complete and effective analgesia, sedation levels, and any potential side effects associated with the drugs. The total number of patients, which was 50, was separated into two distinct groups. **Result:** The group administered with Dexmedetomidine exhibited a significantly earlier start of sensory block (mean time of 108.22±11.63 seconds) compared to the group administered with Clonidine (mean time of 137.01±12.58 seconds), with a statistically significant p-value of less than 0.005. The results indicate that the time it takes for motor block to occur after injection, known as onset of motor block, was earlier in the Dexmedetomidine group (122.87±10.14 seconds) compared to the Clonidine group (149.89±11.47 seconds). The Visual Analogue Scale (VAS) scores were seen to be significantly lower in the group receiving dexmedetomidine compared to the group receiving clonidine at the 3rd, 5th, and 6th hours. The differences in VAS scores between the two groups were found to be statistically significant, with p-values of 0.001, 0.01, and 0.006, respectively. The diastolic blood pressures at various time intervals show no statistically significant differences ($p>0.05$) between the two groups, except at 5, 10, 30, and 60 minutes. In these instances, the clonidine group has a significantly lower systolic blood pressure compared to the dexmedetomidine group ($p<0.05$). **Conclusion:** We concluded that the administration of 5 µg dexmedetomidine seems to provide a compelling alternative to the use of 30 µg clonidine as a supplementary agent to spinal bupivacaine during surgical interventions. The intervention offers high-quality intraoperative analgesia, maintains stable hemodynamic circumstances, provides sufficient sedation, minimises adverse effects, and delivers excellent postoperative analgesia.

INTRODUCTION

Spinal anaesthesia is considered to be a very straightforward regional anaesthetic procedure to administer. The safe implementation of spinal anaesthesia encompasses many key steps, including the careful selection and preparation of the patient, the accurate identification and access to the cerebrospinal fluid (CSF), the administration of suitable anaesthetic medications and adjuvants, the effective management of physiological side effects, and the continuous monitoring of the patient both throughout the operation and in the first stages of recovery.[1] The use of hyperbaric bupivacaine 0.5% in spinal anaesthesia is widely advocated. The primary factors contributing to the widespread use of

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spinal blocks are their clearly defined termination locations and the consistent ability of anesthesiologists to administer them effectively with a singular injection. The adaptability of spinal anaesthesia is facilitated by a diverse array of local anaesthetics and additives, which provide the ability to regulate the extent, onset time, and duration of spinal anaesthesia. The degree of the neuronal blockade generated by spinal anaesthesia is determined by the dispersion of local anaesthetic solutions throughout the subarachnoid space.[2]

Bupivacaine, a commonly used local anaesthetic, is often utilised for spinal anaesthesia, however its duration of action is quite limited. Various adjuvants have been administered intrathecally with local anaesthetics in order to enhance the intraoperative analgesia and extend its duration throughout the postoperative phase.[3] Opioids are often used as intrathecal adjuvants, demonstrating little motor or autonomic inhibition. Nevertheless, the occurrence of adverse effects such as pruritus, nausea, vomiting, urine retention, and delayed respiratory depression has inspired more investigation into other nonopioid analgesics that exhibit reduced side effects.[4]

The investigation of α2-adrenergic agonists as novel neuraxial adjuvants aims to enhance the efficacy of subarachnoid blockade in terms of sensory and motor blockades. Numerous studies have been conducted to substantiate the effectiveness of these adjuvants when used separately.[5] In addition to this, dexmedetomidine and clonidine have been identified as being beneficial. The principal mechanism of action is hypothesised to occur at the spinal cord level. This encompasses both pre and postsynaptic locations where the activity takes place. The activation of α2-receptors presynaptically leads to the inhibition of substance P release from afferent "c" fibres located inside the dorsal horn. In the postsynaptic context, it has an inhibitory effect on the progression and subsequent propagation of consolidated pain signals inside second-order neurons located in the substantia gelatinosa. The potential use of clonidine, a specific partial agonist of α2-adrenergic receptors, is now being assessed as a supplementary treatment to intrathecal local anaesthetics, with no observed clinically relevant adverse effects.[6,7] Dexmedetomidine, an emerging α2-adrenergic agonist, is now being assessed because to its notable attributes, including high specificity, potency, and selectivity. This compound exhibits the ability to maintain stable hemodynamic circumstances and provide satisfactory intraoperative and extended postoperative analgesia, while minimising adverse effects.[5] Although clonidine has been used as an adjunct to bupivacaine in subarachnoid blocks, there is a limited body of research about the intrathecal administration of dexmedetomidine.

MATERIALS AND METHODS

Following the acquisition of approval from the institutional ethics committee and the collection of written informed permission from the participants, a hospital-based double-blind, prospective randomised controlled trial was carried out on a cohort of 50 patients who were scheduled to have elective urological procedures under spinal anaesthetic. This research covered male patients, namely those with ASA grades I and II, within the age range of 18 to 58 years. Patients classified as ASA grades 3 and 4, exhibit physical dependence on opioids and benzodiazepines. The research excluded patients with severe systemic illness, metabolic disorders, cardiac disorders, congenital diseases, or neurologic abnormalities due to contraindications to regional anaesthesia. This study aimed to investigate the impact of intrathecal administration of clonidine (30 micrograms) and dexmedetomidine (5 micrograms) on various parameters, including the time of onset, peak effect, and duration of sensory and motor block, as well as the hemodynamic effects. Additionally, the study examined the duration of complete and effective analgesia, sedation levels, and any potential side effects associated with the drugs. The total number of patients, which was 50, was separated into two distinct groups. The study had two groups, namely Group A, which received clonidine, and Group B, which received dexmedetomidine. There were 25 participants assigned to each group.

Statistical Methods

The use of statistical methods for the purpose of analysing data.The data collected was organised and analysed using the Microsoft Office spreadsheet programme, Excel. The data were presented in the form of means, standard deviations, ranges, numerical values, and proportions. The Z test was used for categorical factors such as sex, ASA class, hypotension, bradycardia, etc., with p-values given at a 95% confidence range.A p-value was deemed statistically significant if it was less than 0.05.

RESULTS

The group administered with Dexmedetomidine exhibited a significantly earlier start of sensory block (mean time of 108.22±11.63 seconds) compared to the group administered with Clonidine (mean time of 137.01±12.58 seconds), with a statistically significant p-value of less than 0.005. The length of time it took for the sensory block to regress to S1 was found to be substantially longer in the Dexmedetomidine group (405.74±13.63 minutes) compared to the Clonidine group (294.52±11.74 minutes), with a statistically significant p-value of less than 0.001. The group administered with Dexmedetomidine exhibited a substantially longer duration of effective analgesia (471.59±15.63 minutes) compared to the group administered with Clonidine (381.55±14.74 minutes), with a

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statistically significant p-value of less than 0.001. The attainment of peak sensory level occurred at an earlier time point in the group administered Dexmedetomidine (20.21±3.14 minutes), compared to the group administered Clonidine (28.11±3.63 minutes), with a statistically significant p-value of less than 0.001.

The results indicate that the time it takes for motor block to occur after injection, known as onset of motor block, was earlier in the Dexmedetomidine group (122.87±10.14 seconds) compared to the Clonidine group (149.89±11.47 seconds). Similarly, the time it took to achieve Bromage-3, indicating peak motor block, was also earlier in the Dexmedetomidine group (5.72±1.69 minutes) compared to the Clonidine group (6.88±1.77 minutes). These differences were statistically significant, with p-values of 0.04 and 0.01, respectively. The inclusion of dexmedetomidine resulted in a considerably longer period of motor block (351.22±9.61 min) compared to clonidine (239.79±8.98 min), as shown by a statistically significant p-value of 0.001. The Visual Analogue Scale (VAS) scores were seen to be significantly lower in the group receiving dexmedetomidine compared to the group receiving clonidine at the 3rd, 5th, and 6th hours. The differences in VAS scores between the two groups were found to be statistically significant, with p-values of 0.001, 0.01, and 0.006, respectively.

Table 1: Comparison of Intra and Post-Operative Sensory Block, Motor Block and VAS Score

Sensory Block	**Clonidine=25**		**Dexmedetomidine=25**		**P-Value**
	Mean	**Sd**	**Mean**	**Sd**	
Onset of sensory block (secs)	137.01	12.58	108.22	11.63	0.005
Duration of sensory block (mins)	294.52	11.74	405.74	13.63	0.001
Duration of effective analgesia (mins)	381.55	14.74	471.59	15.63	0.001
Peak sensory level in mins	28.11	3.63	20.21	3.14	0.001
Motor Block					
Onset of motor block (secs)	149.89	11.47	122.87	10.14	0.04
Peak motor block (mins)	6.88	1.77	5.72	1.69	0.01
Duration of motor block (mins)	239.79	8.98	351.22	9.61	0.001
VAS Score					
2 hours	0.0	0.0	0. 0	0. 0	-
3 hours	2.12	0.21	1.37	0.36	0.001
4 hours	2.71	0.66	2.84	0.39	0.26
5 hours	3.91	0.88	3.51	0.44	0.01
6 hours	4.85	0.39	4.39	0.49	0.006

Table 2: Sedation Score

Sedation Score	**Clonidine=25**		**Dexmedetomidine=25**	
	Number	**Percentage**	**Number**	**Percentage**
Grade 0	21	84	0	0
Grade 1	3	12	22	88
Grade 2	1	4	3	12
Grade 3	0	0	0	0
Grade 4	0	0	0	0
Grade 5	0	0	0	0

A total of 84% of the patients in the Clonidine group exhibited grade 0 sedation, whereas none of the patients in the Dexmedetomidine group shown grade 0 sedation. In the Dexmedetomidine group, a majority of patients (88%) exhibited a sedation score of grade 1, but in the Clonidine group, this proportion was much lower at 12%. The remaining patients, including 12% in the Dexmedetomidine group and 4% in the Clonidine group, had a sedation score classified as grade 2. The group of patients administered with Dexmedetomidine had a substantially greater sedation score, with a p-value of less than 0.001.

Table 3. Heart Rate/Min and SBP mm hg

Heart Rate/Min	**Clonidine=25**		**Dexmedetomidine=25**		**P-Value**
	Mean	**Sd**	**Mean**	**Sd**	
0 minute	82.01	5.25	81.11	3.36	0.24
5 minutes	77.02	4.63	78.85	4.06	0.19
10 minutes	73.31	4.21	75.11	4.39	0.23
15 minutes	69.06	4.74	71.14	2.98	0.41
30 minutes	66.21	4.63	70.01	3.31	0.001
60 minutes	68.25	±4.66	71.07	3.33	0.11
120 minutes	71.99	3.14	71.89	3.14	0.24
180 minutes	74.22	2.87	75.12	3.79	0.26
SBP mm hg					
0 minute	120.05	4.41	121.77	4.47	0.15
5 minutes	114.10	4.48	117.41	3.63	0.17
10 minutes	110.09	4.74	112.21	3.85	0.36
15 minutes	104.09	3.79	107.91	3.79	0.29

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30 minutes	98.99	4.29	103.37	3.87	0.25
60 minutes	101.89	3.69	106.27	3.52	0.03
120 minutes	106.99	3.25	109.11	2.88	0.21
180 minutes	110.11	2.87	111.01	2.91	0.18

The systolic blood pressure at 60 minutes is significantly lower in the clonidine group than the dexmedetomidine group (p-value of 0.03). There was no change in systolic blood pressure between the two groups during the rest of the period.

Table 4. Comparison of DBP mm hg, MBP mm hg and Respiratory Rate

DBP mm hg	**Clonidine=25**		**Dexmedetomidine=25**		**P-Value**
	Mean	**Sd**	**Mean**	**Sd**	
0 minute	79.45	3.48	80.36	3.63	0.36
5 minutes	74.05	3.69	77.03	3.85	0.07
10 minutes	70.11	3.74	72.33	4.85	0.07
15 minutes	65.34	3.66	66.98	3.33	0.41
30 minutes	61.47	3.73	64.52	3.89	0.001
60 minutes	60.87	3.96	67.71	3.66	0.001
120 minutes	68.35	2.87	69.24	3.41	0.31
180 minutes	69.98	2.87	69.55	2.24	0.21
MBP mm hg					
0 minute	93.02	3.89	94.12	3.69	0.41
5 minutes	87.31	3.58	89.04	3.61	0.11
10 minutes	84.06	3.85	85.24	3.71	0.36
15 minutes	78.11	3.70	80.02	3.10	0.37
30 minutes	75.12	3.41	78.05	3.47	0.05
60 minutes	76.03	3.02	80.02	3.33	0.001
120 minutes	80.06	3.06	82.09	3.19	0.23
180 minutes	83.77	2.87	83.69	2.02	0.36
Respiratory Rate					
Pre op	17.88	1.58	18.10	1.96	0.58
5 minute	17.77	1.63	18.79	1.36	0.26
10 minutes	17.01	1.02	16.99	1.36	0.71
15 minutes	17.22	1.06	17.59	1.31	0.81
30 minutes	18.27	1.11	17.88	1.74	0.52
60 minutes	17.99	1.98	20.11	1.39	0.63
90 minutes	17.88	1.07	16.63	1.11	0.07
120 minutes	16.03	1.10	18.58	1.62	0.04
180 minutes	16.03	1.98	18.55	1.33	0.001

The diastolic blood pressures at various time intervals show no statistically significant differences ($p>0.05$) between the two groups, except at 5, 10, 30, and 60 minutes. In these instances, the clonidine group has a significantly lower systolic blood pressure compared to the dexmedetomidine group ($p<0.05$). The clonidine group had substantially lower mean blood pressure levels at 30 and 60 minutes compared to the dexmedetomidine group, as shown by a p-value of less than 0.05. Based on the aforementioned findings, it can be deduced that there were no substantial changes in hemodynamic parameters, including heart rate, systolic blood pressure, diastolic blood pressure, or mean arterial pressure, seen over the majority of the duration between the two examined groups. The respiratory rates observed at various time intervals in both groups exhibit a high degree of similarity over the majority of the duration. The occurrence of adverse effects, specifically hypotension and bradycardia, was notably more frequent among patients who were administered intrathecal clonidine. Hypotension was observed in four patients in the clonidine group, whereas none experienced this side effect in the dexmedetomidine group ($p<0.001$). Similarly, bradycardia was observed in four patients in the clonidine group, while none experienced it in the dexmedetomidine group ($p<0.001$). The clonidine group effectively addressed hypotension by administering intravenous fluids consisting of 500 cc of Ringer Lactate solution. None of the aforementioned interventions necessitated the administration of ephedrine for the purpose of correcting hypotension. None of the patients in either group need the administration of atropine for the treatment of bradycardia, despite the observation of bradycardia in four patients within the clonidine group. It is worth noting that the observed instances of bradycardia were of little clinical importance. There was no occurrence of respiratory depression seen in any of the patients belonging to either group.

DISCUSSION

The provision of postoperative analgesia necessitates the use of interventions that exhibit prolonged duration, optimal efficacy, and minimal adverse effects. The most often used local anaesthetic for spinal anaesthesia is hyperbaric bupivacaine 0.5%. Nevertheless, the duration of its analgesic effects after surgery is restricted. Therefore, the use of an adjunct to these regional anaesthetics represents a dependable approach for extending the duration of

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anaesthesia. The use of a more straightforward methodology has gained significant acceptance in the field. Opioids are the most prevalent class of analgesics, widely used as the primary treatment modality for postoperative pain management.[5] Intrathecal administration of opioids has been shown to extend the duration of analgesia. However, it is important to note that this method may also lead to respiratory depression, pruritus, nausea, vomiting, and urine retention, which may occur at a later stage and in an unexpected manner.[8] Therefore, there emerged a need for improved adjuvants that might extend the duration of analgesia while minimising the aforementioned adverse effects associated with opioids. The antinociceptive activity of intrathecal α2-agonists has been seen in the context of both somatic and visceral pain.[9] Therefore, these substances are used as adjuncts to bupivacaine for the purpose of spinal anaesthesia. The partial α2-adrenergic agonist, clonidine, enhances the sensory and motor block effects of topical anaesthetics. The analgesic action of this substance is achieved by activating postsynaptic α2-receptors located in the substantia gelatinosa of the spinal cord. The activation of the descending inhibitory medullospinal pathways leads to a reduction in the release of nociceptive chemicals from the substantia gelatinosa. Numerous research have been conducted on the intrathecal administration of clonidine. It has been determined to be a conclusive adjunct in extending the duration of analgesic effects. Dexmedetomidine is an α2-receptor agonist that has greater specificity compared to clonidine. It is often used as a premedication agent in the context of general anaesthesia. The administration of this substance has been shown to decrease the amount of opioids and inhalational anaesthetics needed during medical procedures.[10] The demographic characteristics, including age, height, and weight, of the patients examined within the cohort exhibited little variation. All patients included in the study were of the male gender, and the surgical procedures conducted had a high degree of similarity between both groups. To ensure consistency in the intra-operative and post-operative outcomes of the patients, the parameters were maintained at equal levels in both groups. The current research observed that the average time at which sensory block began was found to be significantly shorter in the Dexmedetomidine group (108.22±11.63 seconds) compared to the Clonidine group (137.01±12.58 seconds), with a p-value of less than 0.005. The investigations done by B. S. Sethi et al,[6] yielded comparable findings about the initiation of sensory block while using low dosage clonidine. The length of time it took for the sensory block to regress to S1 was found to be substantially longer in the Dexmedetomidine group (405.74±13.63 minutes) compared to the Clonidine group (294.52±11.74 minutes), with a statistically significant p-value of 0.001. In their study, Popping et al. discovered that the clonidine group exhibited a longer period of sensory block (165.5 ± 30.6 minutes) compared to the control group (139.7 ± 40.4 mins), with a statistically significant difference ($p<0.05$).[11] In a research conducted by Gupta R et al., the administration of D5 (dexmedetomidine 5 μg) was observed to result in a mean sensory regression time to S1 of 476±23 min in the group receiving dexmedetomidine.[9] In a study conducted by Mahmoude M. Al-Mustafa et al, it was shown that the regression time required to reach the S1 dermatome was 338.9±44.8 minutes in group D10, and 277.1±23.2 minutes in group D5.[12] The group administered with Dexmedetomidine exhibited a substantially longer period of effective analgesia (471.59±15.63 minutes) compared to the group administered with Clonidine (381.55±14.74 minutes), with a statistically significant p-value of less than 0.001. In a study conducted by Sethi B S et al. the addition of low dose intrathecal clonidine as an adjuvant to bupivacaine resulted in a statistically significant difference in the average duration of effective analgesia between the two groups. The clonidine group had a mean duration of 614 minutes (with a range of 480 to 1140 minutes), while the control group had a mean duration of 223 minutes (with a range of 150 to 300 minutes).[6] In our study, it is apparent that the initiation of motor block, specifically the time from injection to Bromage 0, was earlier in the Dexmedetomidine group (122.87±10.14 seconds) compared to the Clonidine group (149.89±11.47 seconds). Additionally, the peak motor block, indicated by the time required to achieve Bromage-3, was also earlier in the Dexmedetomidine group (5.72±1.69 minutes) compared to the Clonidine group (6.88±1.77 minutes). These differences between the two groups were found to be statistically significant, with p-values of 0.04 and 0.01, respectively. The inclusion of dexmedetomidine resulted in a considerably longer period of motor block (351.22±9.61 min) compared to clonidine (239.79±8.98 min), as shown by a statistically significant p-value of 0.001. In a study conducted by Sethi et al., it was shown that the clonidine group exhibited a significantly longer duration of motor blockage compared to the control group ($p<0.05$).[6] In a study conducted by Kim JE et al, it was shown that the length of motor block in teenagers with clonidine was 251±79 minutes, while in the control group it was 181±59 minutes. These findings suggest that the addition of clonidine leads to a significant prolongation of the motor block duration.[13] In a study conducted by Al-Mustafa et al., it was shown that the duration for regression to Bromage 0 was found to be 246.4±25.7 minutes while using dexmedetomidine at a dosage of 5 mcg (D5).[12] According to a research conducted by Gupta R, the duration of regression time for motor block to achieve modified Bromage 0 was recorded as 421±21 minutes while using dexmedetomidine at a dosage of 5 mcg(D5).[9] The dexmedetomidine group had an earlier start of sensory and motor block compared to the clonidine group. Additionally, the duration of motor and sensory block was considerably prolonged in the dexmedetomidine group when compared to the

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clonidine group. In their investigation, Kanazi et al. discovered that the addition of a modest dosage of dexmedetomidine (3 µg) to a spinal block of bupivacaine (12 mg) resulted in a much faster start of motor block, as well as significantly prolonged sensory and motor block compared to the use of bupivacaine alone.[4] The findings of our study, in which a higher dose (5 µg) of dexmedetomidine was administered, provide further evidence to support the conclusion drawn by Mahmoude M. Al-Mustafa et al,[12] that the effectiveness of dexmedetomidine in facilitating the onset and regression of sensory and motor block in spinal anaesthesia is dependent on the dosage. Furthermore, it was shown that the initiation of sensory block was accelerated when dexmedetomidine was administered. There was no statistically significant disparity seen in the average heart rates throughout various time intervals in the clonidine and dexmedetomidine groups during the intraoperative period, with the exception of the 15 and 30-minute intervals. The clonidine group had a statistically significant reduced mean heart rate compared to the dexmedetomidine group at both the 15-minute and 30-minute time points. Subsequently, the heart rates observed in both cohorts within our research exhibited no statistically significant variation. This demonstrates that our study findings align with the research conducted by Sethi et al, which also saw a higher drop in mean heart rate from 45 minutes to the end of 6 hours in the clonidine group compared to the control group ($p<0.05$).[6] At the 60-minute mark, the clonidine group exhibited a statistically significant decrease in mean systolic blood pressures compared to the dexmedetomidine group ($p<0.05$). The clonidine group exhibited a statistically significant decrease in mean diastolic blood pressure alone at the 30 and 60-minute time points ($p<0.05$). This suggests that the reduction in systolic blood pressure and diastolic blood pressure after the intrathecal injection of clonidine is more significant compared to dexmedetomidine. Although a decrease in blood pressure was seen in the clonidine group, it was of a very minor magnitude. This decrease was effectively managed by administering 500 ml of intravenous Ringers lactate solution, without the need for ephedrine intervention. Sethi et alconducted a study in which low-dose clonidine was administered to a group of participants. The results revealed a statistically significant reduction in blood pressure among the individuals who received clonidine, as compared to the control group. Notably, none of the patients in either group need any therapeutic intervention.[6] Al-Mustafa et al. observed consistent blood pressure levels throughout the intraoperative phase while administering dexmedetomidine at doses of 5 and 10 mcg (referred to as D5 and D10).[12] There was no statistically significant disparity seen in the average respiration rates over various time intervals during the intraoperative period of up to 90 minutes, between the clonidine and dexmedetomidine groups. In a study conducted by Sethi et al. in 2007, it was shown that the clonidine group did not exhibit any statistically significant alterations in respiratory rate or Sp02 compared to the baseline measurements ($p>0.05$). Furthermore, the administration of supplemental oxygen or any other kind of airway management was not deemed necessary.[6] In their study, Kanzi et al. observed that the administration of dexmedetomidine at a dosage of 3 mcg (referred to as D3) did not result in any respiratory depression. The results of our research indicate that the sedation ratings were considerably greater in the group receiving dexmedetomidine compared to the group receiving clonidine ($p<0.05$). This suggests that intrathecal administration of dexmedetomidine is associated with superior sedation compared to clonidine.[4] The Visual Analogue Scale (VAS) score exhibited a statistically significant decrease in the dexmedetomidine group at the 3rd, 5th, and 6th hours when compared to the clonidine group. The p-values for these comparisons were 0.001, 0.01, and 0.006, respectively. The study conducted by Chandra GP et al. shown that the concurrent administration of intrathecal clonidine resulted in a reduction in both the 24-hour intravenous morphine consumption and the 24-hour Visual Analogue Scale (VAS) scores, as compared to the administration of intrathecal morphine alone. Therefore, it can be deduced that both intrathecal dexmedetomidine and clonidine have the effect of extending the duration of post-operative analgesia, resulting in a reduction of visual analogue scores during the post-operative period. However, when combined with bupivacaine, dexmedetomidine demonstrates superior post-operative analgesic properties compared to clonidine.[3] There were no instances of postoperative problems, such as nausea, vomiting, hypotension, or bradycardia, seen in any of the patients within either experimental group. The findings of this study were similar to those reported by Kanzi et al.[4] The administration of dexmedetomidine through intrathecal route is considered off-label. In animal experiments, the maximum dosage of intrathecal dexmedetomidine administered was 100 µg.

CONCLUSION

We concluded that the administration of 5 µg dexmedetomidine seems to provide a compelling alternative to the use of 30 µg clonidine as a supplementary agent to spinal bupivacaine during surgical interventions. The intervention offers high-quality intraoperative analgesia, maintains stable hemodynamic circumstances, provides sufficient sedation, minimises adverse effects, and delivers excellent postoperative analgesia.

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