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ASSESSMENT OF THE EFFECT OF DEXMEDETOMIDINE WHEN ADDED TO LOCAL ANAESTHETIC IN THE DURATION OF SUBARACHNOID BLOCKADE / SPINAL ANESTHESIA- AN EXPERIMENTAL DOUBLE BLIND RANDOMIZED CONTROL TRIAL

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ABSTRACT

Background: Spinal Anaesthesia is a commonly used technique in many surgeries as it provides sufficient surgical Anaesthesia but if only local anaesthetic is used, it has a shorter duration of action and is inadequate for visceral analgesia. Different additives have been used to increase duration and quality of anaesthesia like clonidine, fentanyl etc. Injection Dexmedetomidine is selective alpha- 2 adrenergic agonist and can be used as an additive in subarachnoid block. The study aims to assess the effect of dexmedetomidine when added to local anesthetic in subarachnoid block / spinal anaesthesia. **Methods and Materials:** 50 willing patients who were scheduled for a subarachnoid block were divided into Group D and Group B. Patients in Group D got 20 mg of 0.5% bupivacaine Heavy and 10 micrograms of dexmedetomidine. Group B received 20 mg of 0.5% hyperbaric bupivacaine combined with saline (to make the same volume). It was noted how long it took to establish sensory block to the T10 dermatome, how long it took to get the motor block to the Bromage scale 3, how long the sensory and motor blocks lasted, and how long the postoperative analgesia lasted. Blood pressure, respiration rate, and other vital signs were observed. **Results:** In group B patients, the time required for sensory blockade to reach the T10 dermatome was 6.72 ± 0.73 min. The time required to reach the motor block to Bromage 3 was 17.32 ± 1.34 min. The duration needed for motor block to regress to Bromage 0 was 140.76 ± 4.12 min, the duration of the sensory block was 158.24 ± 5.31 min, and the duration of analgesia in the postoperative period was 169.76 ± 6.46 min. In Group D we found that the time needed for the sensory block to reach T10 dermatome was 4.6 ± 0.76 min. The time required to achieve motor block to Bromage 3 was 10.52 ± 1.08 min. The time required for the motor block to regress to Bromage 0 was 258 ± 11.1 min, the period of sensory block was 293.32 ± 12.13 min and the duration of postoperative analgesia was 323± 12.82 min. **Conclusion:** Dexmedetomidine enhances the

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anaesthetic conditions and offers superior postoperative analgesia when combined with local anaesthesia in spinal anaesthesia.
Keywords: Dexmedetomidine, spinal anaesthesia, postoperative analgesia.

INTRODUCTION

The spinal cord contains alpha-2 adrenoreceptors. The partial alpha-2 agonist clonidine has been effectively used in spinal and epidural anaesthesia. Intrathecal clonidine not only prolongs postoperative analgesia and reduces the requirement for morphine postoperatively, but it also enhances the quality of analgesia. (1-5). Dexmedetomidine is a 7-10 times more selective alpha-2 adrenergic agonist than clonidine. When dexmedetomidine was tried as an intrathecal injection in rats, it was found to be a very powerful anti-nociceptive medication (6, 7). Low-dose dexmedetomidine has been used safely for spinal anaesthesia in humans. (8,9).

Aim: To assess the effectiveness of dexmedetomidine as an adjuvant along with hyperbaric bupivacaine for spinal anaesthetic block.

Objectives: To study how dexmedetomidine affected the beginning of motor and sensory block, the type of block, the duration of postoperative analgesia, and any associated side effects.

MATERIALS AND METHODS

50 ASA grades I and II patients who consented and were posted for lower limb orthopaedic surgery were included in the study after receiving ethics committee approval. Individuals with respiratory illnesses as well as those with heart diseases, hypertension, or who were using ACE inhibitors, calcium channel blockers(CCB), alpha-2 adrenergic receptor antagonists, were excluded from the study. Standard non-invasive monitoring was employed. Subarachnoid block was performed at the level of L3-L4 using a 25 gauge Quincke spinal needle in the sitting position. After obtaining approval from the Institutional Ethics Committee, the patients were chosen randomly and divided into two groups. Each group received the same dose and volume. The patients in Group D received 20 mg of 0.5% hyperbaric bupivacaine and 10 mcg of dexmedetomidine while the Group B patients were given 20 mg of 0.5% hyperbaric bupivacaine + normal saline (to achieve the same volume).

The assigned groups or the solution used weren't known to the anesthesiologist who carried out the procedure. An anesthesiologist delivered the spinal block, vital signs were recorded every two minutes for the 15 minutes, then every five minutes till end of the procedure, and once every fifteen minutes in the recovery until the patient was shifted out. The midline pinprick sensation was used to gauge the dermatome's sensory level. The degree of the motor dermatome was determined by a modified Bromage scale: Grade 0: No motor block; Grade 1: Impossibility to raise extended leg; Grade 2: Impossibility to raise leg; Grade 3: At grade 3, the motor limb has a complete block. After 15 minutes of spinal anaesthesia, sensory and motor blockades were measured using the Bromage scale every minute. After that, they were measured every two minutes until the highest level of the dermatome was reached.

The Bromage scale and sensory dermatome were recorded every 15 minutes in the recovery area until the patient was shifted out. The time to achieve the T10 dermatome and motor block to Bromage 3 was noted in the operating room. In the recovery room, the time required for the sensory block to regress to S1 dermatome and the time to achieve Bromage 0 was recorded. Hypotension was defined for the study as a mean arterial pressure of less than 60 mmHg, while bradycardia was defined as a heart rate of less than 55 beats per minute. The patients received injections of Ondansetron 4 mg as pre-medication. Within 24 hours and 4

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weeks in the outpatient clinic following the surgeon's discharge, all patients were contacted. The anesthesiologist examined the patients to determine if there was any neurological damage brought on by spinal anaesthetic, as well as any new deficits or complications.

OBSERVATIONS AND RESULTS

Following a positive CSF aspiration, subarachnoid blocks were administered to all patients. The ANOVA test was used to compare and interpret the data. All 50 patients adhered to the study procedure and all patient's data were analyzed. Demographic data shows that there were 10 male and 15 female patients in group D and 13 male and 12 female patients in group B. The mean age in group D was 39.52 ± 57 years and in group B mean age was 41.48 ± 13.01 years (Table 1)

Table 1: Demographic Data distribution in Group D & Group B

Group	**Age(in years)***	**Male**
Group D	39.52 ± 9.57	10
Group B	41.48 ± 13.01	13

*Mean age ± SD

After the addition of dexmedetomidine, the time required to achieve the T10 dermatome was 4.6± 0.76 minutes. Compared to plain bupivacaine (6.72 ± 0.73 min), it was statistically significant. The time required to reach the motor block to Bromage scale 3 was 10.52 ± 1.08 minutes in group D & that required in group B was 17.32 ± 1.34 min. This difference was also statistically significant. In group D, the duration required to regress the motor block to Bromage scale '0' was 258 ± 11.1 min. In group B it took 140.76 ± 4.12 min. The mean duration of sensory block was 293.32 ± 12.13 min and 158.24 ± 5.31 minutes in group D and in group B, respectively. The mean duration of postoperative analgesia in group D and group B was 323 ± 12.82 minutes and 169.76 ± 6.46 minutes respectively. A statistically significant difference was seen between the two groups in both of the above counts **(Table 2 & Fig. 1)**.

In group D, 4 patients experienced bradycardia (HR < 60 beats/min), for which Inj. Glycopyrolate 0.2 mg IV was administered. One patient experienced nausea and vomiting with hypotension. The duration of surgery was 85 ± 13.9 min in group D and 89.84 ± 12.88 min in group B (Table 2).

Table 2: Time required to achieve specific Dermatome and Bromage Scale

	Group D *	Group B *	p value
Sensory block to reach T10 dermatome (in min)	4.6 ± 0.76	6.72 ± 0.73	< 0.05
Motor block to reach Bromage scale 3 (in min)	10.52 ± 1.08	17.32 ± 1.34	< 0.05
Motor block to reach the Bromage scale 0 (in min)	258 ± 11.1	140.76 ± 4.12	< 0.05
Duration of sensory block (in min)	293.32 ± 12.13	158.24 ± 5.31	< 0.05

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Duration of post operative analgesia (in min)	323 ± 12.82	169.76 ± 6.46	< 0.05
Duration of surgery (in min)	85 ± 13.9	89.84 ± 12.88	> 0.05

* Time (mins) ± SD

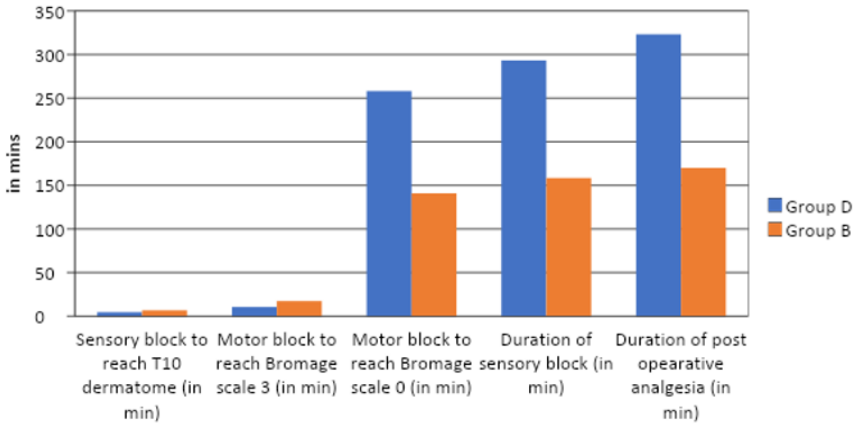

Table 3: Complications

Complication	Group D	Group B	P value
Nausea and vomiting	1	0	> 0.05
Hypotension	1	0	> 0.05
Bradycardia	4	0	< 0.05
Respiratory depression	0	0	> 0.05

DISCUSSION

In spinal anaesthesia, various substances have been employed as adjuvants to local anaesthetics, including clonidine(1–5), opioids, magnesium sulphate, and phenylnephrine. Small dosages of dexmedetomidine have been used for spinal anaesthesia without neurological damage or hemodynamic instability (8,9). The maximum dose of dexmedetomidine (100 ug) was given to animals like sheep, none of which experienced neurological damage (10). In our trial, we utilised 10 ug of dexmedetomidine, and a one-week checkup showed no problems or neurological damage. Dexmedetomidine and clonidine have comparable effects when given intrathecally in a ratio of 1:10. It was demonstrated by Kanazi in 2006 (8). Asano demonstrated that when given via the epidural route, the binding affinity of the alpha 2 agonist to the spinal alpha 2 adrenergic receptor correlated well (6). Dexmedetomidine is the superior adjuvant in terms of hemodynamic stability and postoperative analgesia, according to Bajwa, who administered dexmedetomidine and clonidine epidurally (11).

Little dosages of clonidine in spinal anaesthesia have been demonstrated by Strebel to significantly increase the duration of anaesthesia and post- operative analgesia along with bupivacaine in a dose-dependent manner, with 150 ug of clonidine appearing to be the preferred dose for neuraxial anaesthesia prolongation (12). As compared to clonidine, dexmedetomidine has a 1:10 binding affinity. Hence, based on our research, we predicted that 10 mcg of dexmedetomidine would have the same effects as 100 mcg of clonidine.

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Clonidine, an alpha 2 adrenergic agonist, was administered intrathecally without any issues or neurological deficits prior to the development of dexmedetomidine. It is not known how an alpha-2 agonist lengthens the motor and sensory effects of topical anaesthetics. The alpha-2 adrenergic agonists act on presynaptic C fibres and post-synaptic dorsal horn neurons. The intrathecal alpha 2 adrenergic agonist They act by depressing the release of C-fiber transmitters and by hyperpolarization of post-synaptic dorsal horn neurons(13). This explains why the alpha 2 adrenergic agonist, when combined with local anaesthetics used in neuroaxial anaesthesia, has an antinociceptive effect. The motor activity of local anaesthetic is prolonged by binding of alpha 2 adrenergic agonist to motor neurons in the dorsal horn.(8)

In this study, 10 µg of dexmedetomidine was used with 20 mg bupivacaine under spinal anesthesia. We observe that dexmedetomidine causes significant bradycardia (Table 3). There was no respiratory depression or decrease in blood pressure. In his study, Kanazi found that motor blockade had a significantly early onset and the duration of motor and sensory blockade was increased after adding 3 µg dexmedetomidine in spinal anaesthesia (8). This conclusion is supported by our findings. Also, we found that the time required for the sensory block to reach to the T10 dermatome was less. This may be attributed to the larger dose of dexmedetomidine and the more volume of total drug that we used in our study as compared to the study done by Kanazi.

CONCLUSION

Finally, we can conclude that the use of dexmedetomidine along with bupivacaine causes the early onset of motor and sensory blockade during spinal anaesthesia. Dexmedetomidine also helps in prolonging local anaesthetic blockade and provides good analgesia post-operatively. However, dexmedetomidine causes some bradycardia, which is treatable with anticholinergic drugs. Dexmedetomidine does not have any other notable side effects besides bradycardia. For superior intraoperative and postoperative analgesia with few side effects, we therefore recommend using dexmedetomidine as an adjuvant to local anaesthetic in spinal anaesthesia.

Conflict of Interest: The authors declare no conflict of interest.

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