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**Original Article**

# Synergistic effect of Dexmedetomidine on Subarachnoid Block with Hyperbaric Bupivacaine

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## ABSTRACT

***Objective:*** *to evaluate the impact of Dexmedetomidine when administered intravenously in infusion or bolus on subarachnoid anesthesia in combination with hyperbaric bupivacaine.*

***Methods:*** *A randomized controlled trial was conducted at the department of Anesthesia, Lady Reading Hospital Peshawar, Pakistan, from January 2023 to June 2023. T10 level was pointed for assessment of snsory blockade, and periodically motor blockade was assessed until the achievement of modified Bromage score 3. Sedation levels were assessed using the Ramsay score, while adverse effects such as nausea, bradycardia and hypotension, vomiting, diarrhea, and pruritus were closely monitored and documented throughout the study period.*

***Results:*** *The onset time of sensory blockade was significantly higher in Group Dex+B compared to Group B ($p < 0.001$). Additionally, the duration of sensory blockade was also significantly higher in Group Dex+B than in Group B ($p < 0.001$). Moreover, the rate of recovery of complete sensory block was notably higher in Group Dex+B compared to Group B ($p < 0.001$). Similarly, when considering motor blockade, both the onset and recovery were significantly faster in Group Dex+B than in Group B ($p < 0.001$)*

***Conclusion:*** *The findings from our study indicate that when administered intravenously, Dexmedetomidine, whether given as a bolus or through continuous infusion, leads to a significant extension in sensory and motor blockade duration.*

***Keywords:*** *Synergistic effect, Dexmedetomidine, Bupicaine, Subarachnoid block, Hyperberic*

## 1. INTRODUCTION

Dexmedetomidine is a highly selective alpha-2 adrenergic agonist that is commonly used in anesthesia and sedation[1]. When administered intravenously, Dexmedetomidine can have various effects, including sedation, analgesia, and sympatholysis[2]. In the context of subarachnoid block (also known as spinal anesthesia) with hyperbaric bupivacaine, Dexmedetomidine has been studied for its potential synergistic effects[3]. Hyperbaric bupivacaine is a local anesthetic agent commonly used in spinal anesthesia due to its ability to provide rapid onset and prolonged sensory and motor blockade duration [4].

Several studies have investigated the combination of intravenous Dexmedetomidine with hyperbaric bupivacaine in subarachnoid block and have reported synergistic effects, particularly in terms of prolonging the sensory and motor blockade duration, improving postoperative analgesia[5], reducing intraoperative and postoperative opioid consumption, and enhancing patient satisfaction[6]. The mechanism of this synergistic effect is thought to be related to the central nervous system effects of Dexmedetomidine, including its ability to modulate pain pathways, enhance spinal anesthesia, and provide sedation and anxiolysis[7].

Combination of Dexmedetomidine and hyperbaric bupivacaine has shown promising results in various studies, the optimal dosing, timing, and patient selection criteria are still areas of ongoing research and debate[8,9]. As with any anesthesia technique, individual patient factors, such as age, comorbidities, and concurrent medications, should be considered when determining the most appropriate approach[10].

Study conducted to evaluate the effectiveness of Dexmedetomidine intravenous administration when given at 1 µg/kg dose in bolus form or 0.51 µg/kg infusion in managing sensorimotor effects post subarachnoid anesthesia induced by 12.5mg hyperbaric bupivacaine. Additionally, the profile of adverse-effect of both regimens of Dexmedetomidine at the same dose was assessed, highlighting the scarcity of local data on the sensorimotor impact of different Dexmedetomidine administration protocols despite its extensive study in international contexts.

## 2. METHODOLOGY

A randomized controlled trial was conducted at the department of Anesthesia, Lady Reading hospital Peshawar, Pakistan, from January 2023 to June 2023. This trial enrolled a total of 50 patients aged between 18-65 years who were scheduled for surgery of lower limb in supine position and under subarachnoid anesthesia, including individuals belonging to various ASA Physical Status classes. The sample size was determined based on a previous study conducted by Furqan et al[11].

Sampling technique was Non-probability consecutive sampling employed to randomly assign patients into three groups: Group B received 2 ml of intrathecal bupivacaine; Group Dex+B received a combination of bupivacaine and Dexmedetomidine infusion; and Group BDexI received bupivacaine followed by Dexmedetomidine infusion. The researcher was responsible for all aspects of preparing drug mixtures, administering the drugs for subarachnoid block induction, and recording soinal anesthesia effect on sensorimotor.

Once the free flow of CSF (cerebrospinal fluid) was confirmed, hyperbaric bupivacaine was administered intrathecal either through the interspaces of L3-L4 or L4-L5, zero point time was recorded from the time of administration. T10 level was pointed for assessment of

snsory blockade, and periodically motor blockade was assessed until the achievement of modified Bromage score 3. Sedation levels were assessed using the Ramsay score, while adverse effects such as nausea, bradycardia and hypotension, vomiting, diarrhea, and pruritus were closely monitored and documented throughout the study period. SPSS Statistics Version 27 was used,. Tests of significance were chi-square and ANOVA tests were utilized to compare variables between the different groups, with a predefined significance level set at $p \leq .05$ to determine statistical significance.

## 3. RESULTS

The comparison between Group B and Group Dex+B was conducted and the results were presented in Tables I and II. Firstly, it was found that there were statistically differences was insignificant ($p > 0.050$) in terms of certain parameters between the two groups, as indicated in Table I. However, notable differences were observed in several aspects between the two groups. The onset time of sensory blockade was significantly higher in Group Dex+B compared to Group B ($p < 0.001$). Additionally, the duration of sensory blockade was also significantly higher in Group Dex+B than in Group B ($p < 0.001$). Moreover, complete sensory recovery rate was notably higher in Group Dex+B compared to Group B ($p < 0.001$).

Similarly, when considering motor blockade, both the onset and recovery were significantly faster in Group Dex+B than in Group B ($p < 0.001$). Furthermore, Ramsey sedation score2 was found to be higher in Group B than in Group Dex+B, with a statistically significant difference ($p < 0.001$), as detailed in Table II. Regarding adverse effects, the distribution was nearly equal between Group B and Group Dex+B, ($p > 0.001$), as illustrated in Figure I.

**Table. I**

**Demographic and baseline variables of the groups**

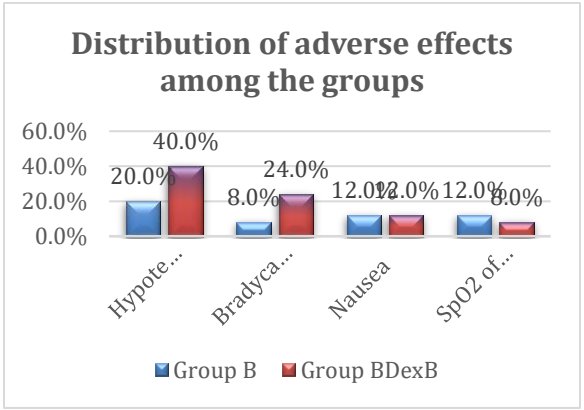
| Variable | Group B 25 (50.0%) | Group Dex+B 25 (50.0%) | p-value |
|---|---|---|---|
| Age (years) | 36.52±4.68 | 35.04±3.59 | 0.686 |
| Gender | | | |
| Male | 13 (52.0) | 12 (48.0) | 0.777 |
| Female | 12 (48.0) | 13 (52.0) | |
| Weight (kg) | 60.40±6.21 | 59.88±5.90 | 0.781 |
| ASA status | | | |
| I | 17 (68.0) | 15 (60.0) | 0.556 |
| II | 8 (32.0) | 10 (40.0) | |
| Mean±S.D, N (%) | | | |

**Table. II**

**Distribution of parameters among the groups**

| Parameter | Group B 25 (50.0%) | Group Dex+B 25 (50.0%) | p-value |
|---|---|---|---|
| Sensory blockade onset in min | 1.94±0.37 | 2.11±0.11 | <0.001 |
| Duration of sensory blockade (min) | 139.41±9.13 | 172.00±12.96 | <0.001 |
| Complete sensory recovery (min) | 216.80±13.25 | 309.64±20.84 | <0.001 |
| Motor blockade onset in min | 2.07±0.19 | 2.33±0.10 | <0.001 |
| Motor recovery (min) | 228.44±17.68 | 333.04±9.85 | <0.001 |
| Ramsey sedation score | | | |
| 2 | 13 (52.0) | 5 (20.0) | <0.001 |
| 3 | 12 (48.0) | 20 (80.0) | |
| Mean±S.D, N (%) | | | |

**Figure. I**



## 4. DICSCUSSION

In this study, we evaluated the effectiveness of Dexmedetomidine with hyperbaric bupivacaine intrathecaly on subarachnoid anesthesia. We compared the outcomes when Dexmedetomidine was administered either as a single bolus or as a continuous infusion. Onset time of sensory blockade in this study was 2.11±0.11 minutes which are longer than control group.

In a study conducted by Furqan et al[11], it was found shortest sensory blockade in a group which patients receiving only bupivacaine, while it was longest in the group that received single bolus of combination of bupivacaine plus Dexmedetomidine. Additionally, the time required for complete sensory and motor recovery was longest in the group receiving both bupivacaine and Dexmedetomidine (Group Dex+B) and shortest in the group receiving only bupivacaine (Group B).

In a study conducted by Whizar-Lugo et al[12], contrast observations were reported that the onset time of sensory block was significantly shorter when Dexmedetomidine was used compared to using bupivacaine alone. This suggests that Dexmedetomidine has a rapid onset of action in inducing sensory block anesthesia.

However, a contrasting perspective was presented by Harsoor et al[13], who suggested different results. According to their findings, on comparison of Dexmedetomidine and control group there was a shorter time of sensory blockade onset in Dexmedetomidine group. This discrepancy in findings may be attributed to variations in study protocols, patient populations, or other factors influencing the pharmacokinetics and pharmacodynamics of these medications in regional anesthesia.

In this study, it was observed that the mean time for recovery of sensory block was prolonged, consistent with previous research by Hong et al[14], who found longer mean times to two-segment regression (39 minutes vs. 78 minutes for cold, 41 minutes vs. 61 minutes for pinprick) and motor regression (23 minutes vs. 46 minutes) in the DMT group compared to the control group. Similar findings were reported by Lee et al[15] that pinprick sensory blockade time and motor blockade time was prolonged in Dexmedetomidine administration.

Studies conducted by Elcıcek et al[16] and Dinesh et al[17] reported similar results to our study regarding the time for onset of motor-block, demonstrating a comparable timeframe. However, in contrast to our findings, Dexmedetomidine administration was associated with a reduction in the time required for motor-block onset by approximately one minute. Conclusion of findings is that Dexmedetomidine may have a beneficial effect in hastening the onset of motor block compared to other agents studied in these research investigations.

Niu et al[18] conducted a meta-analysis showed that Dexmedetomidine prolongs subarachnoid anesthesia duration, enhances postoperative analgesia, and doesn't raise hypotension or other adverse event rates, while Kavya et al[19] demonstrated that intravenous Dexmedetomidine bolus or combination of bolus and infusion extends both sensory and motor blockade of intrathecal hyperbaric bupivacaine without adverse effects.

Limitations: The study has focused on a specific demographic or patient population, such as adults of a certain age range or individuals without specific comorbidities. This could limit the applicability of the findings to a broader population.

## 5. CONCLUSION

The findings from our study indicate that when administered intravenously, Dexmedetomidine, whether given as a bolus or through continuous infusion leads to a

significant extension in sensory and motor blockade duration.

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