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Comparison of Dexmedetomidine and Midazolam as Premedication for Attenuation of Perioperative Anxiety and Hemodynamic Response

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Abstract **Background:** Preoperative anxiety can produce significant sympathetic activation and undesirable hemodynamic changes during anesthesia and airway manipulation. **Objective:** To compare the effects of dexmedetomidine and midazolam as premedication on perioperative anxiety and hemodynamic response in patients undergoing elective surgery under general anesthesia. **Methods:** This prospective randomized comparative study was conducted at Poonch Medical College Rawalakot from June 2025 to June 2026 included 80 patients undergoing elective surgery under general anesthesia. Patients were randomly allocated into two equal groups of 40 patients each. Group D received dexmedetomidine, while Group M received midazolam as premedication. **Results:** Baseline demographic and hemodynamic characteristics were comparable between the groups. Anxiety scores were significantly lower with dexmedetomidine at 30 minutes (3.8±0.9 vs. 4.7±1.0, p<0.001) and before induction (3.5±0.8 vs. 4.3±1.0, p<0.001). The mean reduction in anxiety was greater with dexmedetomidine (3.6±1.1 vs. 2.7±1.0, p<0.001). Following intubation, heart rate (91.7±12.1 vs. 105.8±14.3 beats/min) and MAP (102.6±10.4 vs. 115.3±12.1 mmHg) were significantly lower with dexmedetomidine (both p<0.001). Sedation scores were higher with dexmedetomidine (2.6±0.5 vs. 2.1±0.4, p<0.001). Bradycardia was observed in 5 patients (12.5%) receiving dexmedetomidine and 1 patient (2.5%) receiving midazolam. The between-group difference was not statistically significant using a two-sided Fisher's exact test (p = 0.201). **Conclusion:** Patients who received dexmedetomidine had lower preoperative anxiety scores, decreased heart rate and blood pressure at intubation, and increased sedation scores compared with patients who received midazolam. There was a numerical increase in bradycardia events in the dexmedetomidine group, but this was not statistically significant. Thus, with appropriate monitoring for cardiovascular effects, dexmedetomidine may be an alternative to traditional premedications for the perioperative period.

Key Words Dexmedetomidine, Midazolam, Preoperative anxiety, Premedication, Hemodynamic response, Sedation

INTRODUCTION

Patients who have to undergo surgery often experience anxiety before the operation, which can happen for many reasons, including: fear of anesthesia; fear of pain; fear of complications from surgery; fear of not being able to control the surgery; and fear of the outcome [1]. Anxiety can be different for different people and may depend on the type of surgery, past surgery experience and psychological factors [2]. Increased anxiety can stimulate the sympathetic and neuroendocrine system, leading to tachycardia, hypertension, and higher oxygen consumption, which can lead to higher perioperative stress [3]. It can also lead to greater need for anaesthetic and analgesic needs and hurt recovery after the surgery. The role of peri-operative management is important because they could improve patient comfort by providing good anxiolysis which can help to make transition to anesthesia smoother [4]. The ideal premedicant will provide anxiolysis and sedation without causing excessive respiratory and cardiovascular depression. Midazolam is a short-acting benzodiazepine that has been used more than any other and is known for its rapid onset, reliable anxiolytic action and amnestic effects [5]. However, postoperative cognitive impairment, psychomotor impairment, excessive sedation, and respiratory depression can be a limitation, especially in vulnerable patients. Dexmedetomidine is a highly selective α2-AR agonist with

sedative and anxiolytic properties, mostly mediated by the reduction of central sympathetic activity. It has a sedative effect that differs from that of benzodiazepines, and typically preserves spontaneous ventilation [6]. Dexmedetomidine has also been shown to have sympatholytic effects that could decrease the cardiovascular response to anxiety, laryngoscopy, tracheal intubation, and surgical stimulation [7]. A comparison of dexmedetomidine and midazolam is relevant as both drugs are used for preoperative anxiolysis. Midazolam cause anxiolysis by activation of gamma-aminobutyric acid type A receptors and results in sedation and amnesia of events in the period after which it was administered. Dexmedetomidine causes sedation by activation of central alpha-2 adrenergic receptors and also anxiolysis. Sympatholytic effects of the drug may reduce the extent of increase in heart rate and blood pressure during airway manipulation. However, the potential for causing bradycardia and hypotension must be taken into account when choosing a premedication.

It is the hemodynamic effects of dexmedetomidine that are most significant in the perioperative period. This effect of the drug can diminish the sympathetic overdrive that may occur during stressful periods of anesthesia that might be expected to lead to an increase in heart rate and blood pressure. Previous clinical research has shown that dexmedetomidine will lower the hemodynamic response of tracheal intubation and other airway interventions [8]. However, too much sympatholytic activity can lead to bradycardia and hypotension and proper dosing and monitoring of cardiovascular function are important [9]. There has been increasing evidence that dexmedetomidine is an alternative to the traditional benzodiazepine premedication. Comparative studies have shown favourable anxiolytic and sedative effects of dexmedetomidine, although the extent of benefit depends on the dose, route, timing, type of surgery and patient characteristics [10]. Several studies have shown that dexmedetomidine provides superior peri-operative sedation and hemodynamic stability while other studies have found results similar to those obtained with midazolam [11]. More recent systematic reviews also have indicated that dexmedetomidine might have clinically relevant anxiolytic effects in adult perioperative patients. However, hypotension and bradycardia continue to be a concern with its routine use. The relative merits of dexmedetomidine versus midazolam for both anxiety control and hemodynamic stability are difficult to determine because of differences between the available studies [12,13]. Past studies comparing the anxiolytic premedication agents, midazolam and diazepam, have employed different regimens of premedication, studied different populations of surgical patients, utilized various methods for assessing preoperative anxiety and monitoring hemodynamic effects, and implemented diverse anesthetic/surgical plans. Thus, while comparative studies of the two agents have been conducted, it is difficult to draw conclusions regarding clinical utility based on the differing studies. The present study was therefore undertaken to compare, within the context of a single operating room and employing identical anesthetic/surgical plans, the preoperative anxiety of patients as well as their hemodynamic responses, level of sedation, and incidence of adverse events in those patients undergoing elective surgery under general anesthesia and who had received either midazolam or diazepam as premedication.

Objective

To compare the effects of dexmedetomidine and midazolam as premedication on perioperative anxiety and hemodynamic response in patients undergoing elective surgery under general anesthesia.

METHODS

This prospective randomized comparative study was conducted among 80 patients undergoing elective surgery under general anesthesia at Poonch Medical College Rawalakot from June 2025 to June 2026. Patients were randomly allocated into two equal groups of 40 each: Group D received dexmedetomidine and Group M received midazolam as premedication. Participants were allocated to the two treatment groups using [actual method of sequence generation]. Allocation concealment was performed using (actual method, if applicable). Blinding was implemented for (participants, clinicians, outcome assessors, and/or data analysts, as applicable). The allocation and assessment procedures were followed consistently throughout the study. Patients of either gender scheduled for elective surgery under general anesthesia were included. Patients with allergy to either study drug, significant cardiovascular or respiratory disease, severe hepatic or renal dysfunction, psychiatric or cognitive disorders affecting anxiety assessment, regular use of sedative or anxiolytic medication, or incomplete data were excluded. Eligible participants were identified through and assessed against the predefined inclusion and exclusion criteria before enrollment. The sample size was determined using (actual sample-size calculation or recruitment rationale). A total of 80 participants were included in the reported analysis, with 40 participants in each treatment group. Baseline demographic characteristics, vital signs, and preoperative anxiety were recorded. Heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure, and oxygen saturation were assessed before premedication and at predefined perioperative time points, including before induction, during laryngoscopy and intubation, and after intubation. Anxiety, sedation, hemodynamic response, and adverse effects were compared between the two groups. Anesthetic management included induction with specific agents and doses, maintenance with specific anesthetic agents, and perioperative analgesia with specific medications. In addition to the main anesthetic regimen, premedications and concomitant anesthetics were used in specific cases in both treatment groups. The anesthetic management was either standardized or not between the

two treatment groups. The use of rescue medications was in accordance with specific clinical criteria and was documented for subsequent analysis.

Data Collection

Data were collected using a structured proforma. Demographic characteristics, type of surgery, baseline anxiety score,heart rate, systolic and diastolic blood pressure, mean arterial pressure, and oxygen saturation were recorded. Anxiety and hemodynamic parameters were assessed at predefined time points. Hemodynamic measurements were recorded at baseline, after premedication, immediately before induction, immediately after tracheal intubation, and five minutes after intubation. Heart rate and mean arterial pressure were the principal hemodynamic variables reported at these time points. Sedation level and adverse effects, including bradycardia, hypotension, excessive sedation, and respiratory depression, were also documented. All data were entered into a structured database while maintaining patient confidentiality.

Statistical Analysis

Data were analyzed using SPSS version 26.0. Continuous variables were presented as mean±standard deviation or median with interquartile range according to distribution, while categorical variables were presented as frequencies and percentages. Normality was assessed using the Shapiro–Wilk test. Independent samples t-test or Mann–Whitney U test was used for continuous variables, while Chi-square or Fisher's exact test was used for categorical variables. A p-value <0.05 was considered statistically significant.

RESULTS

The two groups were comparable at baseline. 80 patients from the two groups were analyzed in this report. There were 40 patients in each of the dexmedetomidine group and the midazolam group. The patients' demographic data and clinical data on admission, such as age, sex, BMI, ASA physical status and heart rate and blood pressure, had no significant difference between the two groups (p>0.05). Mean age was 42.6±11.4 years in the dexmedetomidine group and 43.1±10.8 years in the midazolam group (p = 0.842), with similar BMI (25.8±3.4 vs. 26.1±3.7 kg/m², p = 0.708). Males comprised 57.5% vs. 55.0% and females 42.5% vs. 45.0% (p = 0.823). ASA I patients were 60.0% vs. 57.5% and ASA II 40.0% vs. 42.5% (p = 0.821). Baseline HR (86.4±11.2 vs. 87.1±10.8 beats/min, p = 0.778), SBP (128.6±13.5 vs. 129.4±14.1 mmHg, p = 0.795), DBP (78.4±8.7 vs. 79.1±9.2 mmHg, p = 0.729), and MAP (95.1±9.1 vs. 95.9±9.5 mmHg, p = 0.701) were also similar (Table 1).

Baseline anxiety scores were similar (7.1±1.1 vs. 7.0±1.2, p=0.689). Adequate reduction in preoperative anxiety in both groups of patients was noted following premedication. Patients who received dexmedetomidine had significantly lower scores of anxiety at 30 minutes following premedication and on the morning of the surgery immediately before induction of anesthesia when compared with the haloperidol-treated patients (both p < 0.001). The mean decrease from the baseline scores also was significantly greater in patients who received dexmedetomidine than in those who received haloperidol (3.6±1.1 vs 2.7±1.0, p < 0.001). At 30 minutes, anxiety decreased to 3.8±0.9 with dexmedetomidine compared with

Table 1: Baseline Demographic and Clinical Characteristics of Study Participants (N = 80)

Variable	Dexmedetomidine Group (N = 40)	Midazolam Group (N = 40)	p-value
Age, years	42.6±11.4	43.1±10.8	0.842
Male	23 (57.5%)	22 (55.0%)	0.823
Female	17 (42.5%)	18 (45.0%)	-
BMI, kg/m²	25.8±3.4	26.1±3.7	0.708
ASA I	24 (60.0%)	23 (57.5%)	0.821
ASA II	16 (40.0%)	17 (42.5%)	-
Baseline HR, beats/min	86.4±11.2	87.1±10.8	0.778
Baseline SBP, mmHg	128.6±13.5	129.4±14.1	0.795
Baseline DBP, mmHg	78.4±8.7	79.1±9.2	0.729
Baseline MAP, mmHg	95.1±9.1	95.9±9.5	0.701

Table 2: Comparison of Preoperative Anxiety Scores Between the Two Groups

Time point	Dexmedetomidine Group (N = 40)	Midazolam Group (N = 40)	p-value
Before premedication	7.1±1.1	7.0±1.2	0.689
30 min after premedication	3.8±0.9	4.7±1.0	<0.001
Before induction	3.5±0.8	4.3±1.0	<0.001
Mean reduction from baseline	3.6±1.1	2.7±1.0	<0.001

Table 3: Comparison of Heart Rate and Mean Arterial Pressure at Different Perioperative Time Points Between the Study Groups

Time point	Heart Rate (beats/min) Dexmedetomidine	Heart Rate (beats/min) Midazolam	p-value	MAP (mmHg) Dexmedetomidine	MAP (mmHg) Midazolam	p-value
Baseline	86.4±11.2	87.1±10.8	0.778	95.1±9.1	95.9±9.5	0.701
After premedication	75.8±9.6	81.9±10.2	0.008	89.2±8.3	93.1±8.9	0.048
Before induction	74.6±8.8	80.7±9.7	0.004	88.4±7.9	92.4±8.6	0.034
Immediately after intubation	91.7±12.1	105.8±14.3	<0.001	102.6±10.4	115.3±12.1	<0.001
5 min after intubation	82.3±10.2	92.6±11.7	<0.001	94.8±8.6	103.7±10.2	<0.001

Table 4: Sedation and Adverse Effects Following Premedication

Variable	Dexmedetomidine Group (N = 40)	Midazolam Group (N = 40)	p-value
Ramsay Sedation Score	2.6±0.5	2.1±0.4	<0.001
Adequate sedation	35 (87.5%)	29 (72.5%)	0.098
Bradycardia	5 (12.5%)	1 (2.5%)	0.042
Hypotension	4 (10.0%)	1 (2.5%)	0.164
Nausea/vomiting	3 (7.5%)	4 (10.0%)	0.692
Respiratory depression	1 (2.5%)	2 (5.0%)	0.556
Excessive sedation	2 (5.0%)	1 (2.5%)	0.556
Need for rescue sedative	2 (5.0%)	6 (15.0%)	0.132
Any adverse event	9 (22.5%)	8 (20.0%)	0.789

4.7±1.0 with midazolam (p<0.001), and before induction to 3.5±0.8 vs. 4.3±1.0 (p<0.001). The mean reduction from baseline was greater with dexmedetomidine (3.6±1.1 vs. 2.7±1.0, p<0.001).

After premedication, HR was 75.8±9.6 vs. 81.9±10.2 beats/min (p = 0.008) and MAP was 89.2±8.3 vs. 93.1±8.9 mmHg (p = 0.048) in the dexmedetomidine and midazolam groups, respectively. Before induction, HR was 74.6±8.8 vs. 80.7±9.7 (p = 0.004) and MAP was 88.4±7.9 vs. 92.4±8.6 mmHg (p = 0.034). Immediately after intubation, HR (91.7±12.1 vs. 105.8±14.3, p<0.001) and MAP (102.6±10.4 vs. 115.3±12.1 mmHg, p<0.001) were significantly lower with dexmedetomidine. At 5 minutes, HR remained lower (82.3±10.2 vs. 92.6±11.7, p<0.001), as did MAP (94.8±8.6 vs. 103.7±10.2 mmHg, p<0.001) (Table 3).

Ramsay Sedation Score was higher with dexmedetomidine (2.6±0.5 vs. 2.1±0.4, p<0.001), while adequate sedation occurred in 87.5% vs. 72.5% (p = 0.098). Bradycardia occurred in 5 patients (12.5%) in the dexmedetomidine group compared with 1 patient (2.5%) in the midazolam group. The difference was not statistically significant using a two-sided Fisher's exact test (p = 0.201). Hypotension occurred in 4 patients (10.0%) and 1 patient (2.5%), respectively, with no statistically significant between-group difference (reported p = 0.164). Rescue sedation was required in 5.0% vs. 15.0% (p = 0 .132), while overall adverse events occurred in 22.5% vs. 20.0% (p=0.789) (Table 4).

DISCUSSION

This study compared the effectiveness of dexmedetomidine and midazolam as premedication in 80 patients undergoing elective surgery. Both groups were well-balanced at baseline, and there was no difference in age, gender, BMI, ASA classification, or baseline heart rate and blood pressure. This similar baseline profile allows the validity of the following between-group comparisons. Previous studies to compare dexmedetomidine versus midazolam in adult surgical patients have also reported similar baseline comparability [14]. Both agents were used to achieve a notable decrease in anxiety, but dexmedetomidine was more effective. Anxiety scores decreased from 7.1±1.1 to 3.8±0.9 after 30 minutes and to 3.5±0.8 before induction in the dexmedetomidine group, compared with 7.0±1.2 to 4.7±1.0 and 4.3±1.0 with midazolam, respectively. The mean reduction was also greater with dexmedetomidine (3.6±1.1 vs. 2.7±1.0, p<0.001). The results of this study are corroborated by previous studies that have demonstrated a significant anxiolytic effect of dexmedetomidine over benzodiazepines with pooled evidence indicating that patients receiving dexmedetomidine had lower anxiety scores. Other studies have also documented similar rates of anxiety for dexmedetomidine compared with midazolam, however, which may be dependent on the dose of medication, route and timing of administration [15]. The present study also showed that dexmedetomidine caused significantly less attenuation of the hemodynamic response to surgery. Mean arterial pressure was also significantly lower in the dexmedetomidine group at the reported perioperative measurement points. These findings suggest that dexmedetomidine was associated with a reduced cardiovascular response during airway manipulation, although other perioperative factors may also have contributed to the observed differences. After 5 minutes, these parameters were still significantly lower with dexmedetomidine. Attenuation of the laryngoscopy and intubation-induced increases of HR and BP have also been shown after dexmedetomidine administration. They are pharmacologically significant since dexmedetomidine is a drug that has an inhibitory effect on sympathetic activity, and thus on the cardiovascular response to airway stimulation [16].

Dexmedetomidine also produced significantly higher Sedation: Ramsay Sedation Score (SS) (2.6±0.5 vs. 2.1±0.4 with midazolam, p<0.001). In 87.5% of the patients, adequate sedation was achieved compared with 72.5% of the patients, which was not statistically significant. Dexmedetomidine has been shown to be a good sedative in previous studies, but comparative studies have not always been able to show significant differences between the two agents. Therefore, the increase in sedation in the present study could be due to the dose of the drugs, the time of administration or the scale used to assess sedation [17,18]. Regarding safety outcomes, bradycardia was numerically more frequent in the dexmedetomidine group than in the midazolam group (12.5% versus 2.5%). However, this difference was not statistically significant when evaluated using a two-sided Fisher's exact test (p = 0.201). Hypotension was also numerically more frequent in the dexmedetomidine group (10.0% versus 2.5%), although the reported difference was not statistically significant. These findings should be interpreted cautiously because the

relatively small sample size limits the precision of adverse-event comparisons. Bradycardia is a consistent finding in previous studies, and is usually not a serious cardiovascular adverse effect with proper dosing and monitoring. It has also been recently compared directly with midazolam and another study again found hypotension and bradycardia occurred more commonly with dexmedetomidine, though this was not statistically significant. There were no differences between the groups in terms of respiratory depression, nausea/vomiting, excessive sedation or overall adverse events, indicating an overall acceptable safety profile in the present study [19]. The number of patients studied was relatively small and the study was carried out in a single clinical setting, so the results of this study may not be broadly applicable to the wider population. The results could have been affected by variations in surgical technique, personal anxiety and reactions to premedication. Longer-term outcomes like postoperative cognitive dysfunction, outcome of recovery or patient satisfaction were not assessed due to the short perioperative follow-up period. Furthermore, the effects of dexmedetomidine and midazolam may differ with different doses and timings.

CONCLUSION

In this study, we found that preoperative anxiety scores were decreased and heart rate and blood pressure at the time of intubation were decreased in patients receiving dexmedetomidine for premedication compared with patients receiving midazolam. Patients who received dexmedetomidine had higher sedation scores than patients who received midazolam. There was a numerical increase in the incidence of bradycardia and hypotension in the dexmedetomidine group, but these were not statistically significant. Thus, in selected patients, dexmedetomidine may provide an alternative to midazolam for premedication. However, patients receiving this agent for premedication should have their cardiovascular system closely monitored and further studies are necessary to determine the safety and clinical significance of the findings of this study.

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