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Original Research Article

IMPACT OF DEXMEDETOMIDINE AND MIDAZOLAM ON SEDATION QUALITY, RESCUE SEDATION REQUIREMENT, AND DURATION OF MECHANICAL VENTILATION IN ICU CARE

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ABSTRACT

Background: Optimal sedation is essential for mechanically ventilated ICU patients to ensure comfort, ventilator synchrony, and prevention of agitation-related complications. Benzodiazepine-based sedation strategies have been associated with deeper-than-required sedation and prolonged ventilation, while dexmedetomidine offers lighter, cooperative sedation with minimal respiratory suppression. Aim: To compare the impact of dexmedetomidine and midazolam on sedation quality, rescue sedation requirement, and duration of mechanical ventilation among ICU patients.

Materials and Methods: This prospective randomized comparative study included 70 adult ICU patients requiring mechanical ventilation. Group 1 received continuous infusion of midazolam (n = 35), while Group 2 received dexmedetomidine (n = 35). Sedation was titrated to a target Richmond Agitation Sedation Scale (RASS) score of -2 to -3. Outcomes assessed included sedation depth, target sedation achievement, rescue sedative requirement, rescue dose frequency, time to first rescue dose, and duration of mechanical ventilation. Statistical significance was set at $p < 0.05$.

Results: Both groups had comparable baseline age profiles. Dexmedetomidine achieved lighter, more appropriate sedation (mean RASS -2.1 ± 0.7) than midazolam (-3.8 ± 0.6; $p < 0.001$) and was associated with significantly lower rescue sedative requirement (31.4% vs. 60.0%; $p = 0.009$). Patients receiving dexmedetomidine required fewer supplemental boluses (1.4 ± 0.8 vs. 2.3 ± 1.1; $p < 0.001$) and exhibited delayed need for first rescue dose. The mean duration of mechanical ventilation was significantly shorter in the dexmedetomidine group (4.71 ± 1.76 vs. 7.37 ± 3.93 days; $p = 0.001$).

Conclusion: Dexmedetomidine provides superior sedation quality, lower rescue sedation requirement, and shorter mechanical ventilation duration compared with midazolam, making it a favourable sedative choice for ICU patients requiring ventilatory support.

Keywords: Dexmedetomidine. Mechanical ventilation. Sedation outcomes.

INTRODUCTION

Sedation in critically ill, mechanically ventilated patients is an essential therapeutic component aimed at reducing anxiety, relieving discomfort, facilitating tolerance to endotracheal tubes, ensuring ventilator synchrony, and preventing self-injury. In the intensive care unit (ICU), an ideal sedative drug should provide rapid onset and predictable depth of sedation, allow easy titration, offer minimal respiratory depression, provide hemodynamic stability, and shorten the duration of mechanical ventilation and ICU stay. Traditionally, benzodiazepines such as midazolam have been widely used because of their anxiolytic, sedative, and amnestic properties. Midazolam, through GABA-A receptor modulation, produces reliable sedation; however, its use has been associated with prolonged

mechanical ventilation, increased risk of delirium, tolerance, respiratory depression, and variable awakening due to hepatic metabolism and accumulation of active metabolites during long-term infusion.[1,2]

Dexmedetomidine, a highly selective α2-adrenergic receptor agonist, has emerged as a preferred sedative in modern ICU protocols due to its unique pharmacodynamic profile. It provides cooperative sedation that closely resembles natural sleep, allows patients to remain arousable, reduces sympathetic outflow, and offers mild analgesic properties without significantly affecting respiratory drive. This property makes dexmedetomidine advantageous in weaning and spontaneous breathing trials. Studies have reported shorter duration of ventilation, reduced incidence of delirium, and potentially better cognitive outcomes in patients receiving dexmedetomidine compared with benzodiazepine-based regimens. Moreover, sedation strategies recommended by the ICU Pain-Agitation-Delirium (PAD) guidelines emphasize light sedation, daily sedation interruption, and delirium prevention, favoring non-benzodiazepine sedatives.[3]

Despite its advantages, dexmedetomidine may cause bradycardia and hypotension, particularly with bolus dosing or in hypovolemic patients. In contrast, midazolam, while cost-effective and deeply sedating, may contribute to delayed awakening and prolonged ICU stay, requiring frequent dose titration and rescue sedatives. Rescue sedation needs arise when the primary sedative agent fails to maintain targeted sedation scores such as the Richmond Agitation Sedation Scale (RASS). Comparative evaluation of these agents is crucial because sedation strategy directly impacts ventilator duration, ICU morbidity, hospital resource utilization, and overall patient outcomes.[4]

Aim

To compare the impact of dexmedetomidine and midazolam on sedation quality, rescue sedation requirement, and duration of mechanical ventilation among ICU patients on mechanical ventilation.

Objectives

1. To compare sedation quality between dexmedetomidine and midazolam using the Richmond Agitation Sedation Scale (RASS).
2. To determine and compare the need for rescue sedation between the two drug groups.
3. To assess and compare the duration of mechanical ventilation in both groups.

MATERIALS AND METHODS

Source of Data: Patients admitted to the Intensive Care Unit (ICU) requiring mechanical ventilation and sedation.

Study Design: Prospective, randomized comparative clinical study.

Study Location: Department of Intensive Care, at tertiary care hospital.

Study Duration: January 2024 to December 2024.

Sample Size: Total = 70: Group 1 (Midazolam) = 35; Group 2 (Dexmedetomidine) = 35.

Inclusion Criteria:

- Adult patients aged 20-60 years requiring mechanical ventilation.
- Patients requiring continuous sedation for ventilator tolerance.
- Hemodynamically stable patients at baseline.
- Consent obtained from legal guardians.

Exclusion Criteria:

- Known hypersensitivity to study drugs.
- Severe cardiac conduction abnormalities or bradyarrhythmias.
- Severe hepatic or renal failure.
- Pregnant or lactating women.
- Patients with severe neurological conditions or active seizures.

Procedure and Methodology: Eligible patients were randomized into two equal groups using a computer-generated sequence. Group 1 received IV midazolam infusion at 0.02-0.1 mg/kg/hour, and Group 2 received IV dexmedetomidine infusion at 0.2-1.4 µg/kg/hour, titrated to achieve target RASS score between -2 to -3. Baseline vitals, sedation score, and ventilator parameters were recorded. Continuous monitoring was performed for hemodynamics, sedation score at fixed intervals, requirement of rescue sedatives, and adverse effects. The duration of mechanical ventilation and any sedation-related complications were documented until successful extubation.

Sample Processing: All clinical parameters were recorded in structured data collection forms, including baseline demographics, vitals, RASS scores, drug infusion rates, and rescue medication doses.

Statistical Methods: Data were analyzed using SPSS software. Continuous variables were compared using independent t-test or Mann-Whitney U test. Categorical variables were evaluated using Chi-square test/Fisher's exact test. Kaplan-Meier survival analysis was used for evaluating ventilator duration. A p-value <0.05 was considered statistically significant.

RESULTS

In the present study comprising 70 mechanically ventilated ICU patients, the baseline age distribution between the two groups was comparable, with no statistically significant difference (41.8 ± 14.3 years vs. 41.5 ± 11.0 years; p = 0.93), indicating appropriate randomization without age-related confounding. However, a significant difference was observed in gender distribution, with males predominating in the midazolam group (74.3%) compared with the dexmedetomidine group (42.9%) (p = 0.008). Following initiation of sedation, the mean RASS score was significantly lower in the midazolam group (-3.8 ± 0.6) compared to the

dexmedetomidine group (-2.1 ± 0.7), indicating deeper sedation with midazolam ($p < 0.001$). Although adequate target sedation (RASS -2 to -3) was achieved in a higher proportion of patients receiving dexmedetomidine (85.7%) compared to midazolam (74.3%), the difference was statistically nonsignificant ($p = 0.25$). The requirement for rescue sedation differed significantly between groups, with 60% of patients in the midazolam group requiring additional sedative boluses compared to only 31.4% in the dexmedetomididine group ($p = 0.009$). Consistently, the midazolam group required a higher number of rescue boluses (2.3 ± 1.1 vs. 1.4 ± 0.8; $p < 0.001$). The duration of mechanical ventilation was significantly prolonged in the midazolam group (7.37 ± 3.93 days) when compared with dexmedetomidine (4.71 ± 1.76 days; $p = 0.001$), and nearly half of the midazolam-sedated patients (48.6%) required ventilation beyond 7 days, unlike only 14.3% in the dexmedetomidine group ($p = 0.002$).

Table 1: Baseline profile and key outcome measures between Midazolam and Dexmedetomidine groups (N = 70)

Measure	Category / Comparison	Group 1: Midazolam (n = 35)	Group 2: Dexmedetomidine (n = 35)	Effect & test of significance	95% CI	p-value
Age (years)	-	41.8 ± 14.3	41.5 ± 11.0	Mean diff = 0.3 years; Student t = 0.09, df = 68	-5.0 to 5.6 years	0.93
Sex	Male	26 (74.3%)	15 (42.9%)	Risk diff = 31.4%; $\chi^2 = 7.12$, df = 1	9.0% to 53.8%	0.008
Sedation depth (RASS after sedation)	RASS score (more negative = deeper sedation)	-3.8 ± 0.6	-2.1 ± 0.7	Mean diff = -1.7; t = 10.7, df = 68	-2.0 to -1.4	<0.001
Adequate sedation (RASS -2 to -3 at 1 h)	Achieved	26 (74.3%)	30 (85.7%)	Risk diff = -11.4%; $\chi^2 = 1.32$, df = 1	-30.6% to 7.7%	0.25
Rescue sedation required (any additional sedative bolus)	Yes	21 (60.0%)	11 (31.4%)	Risk diff = 28.6%; $\chi^2 = 6.85$, df = 1	7.5% to 49.7%	0.009
No. of rescue sedative boluses	-	2.3 ± 1.1	1.4 ± 0.8	Mean diff = 0.9; t = 3.67, df = 68	0.4 to 1.4	<0.001
Duration of mechanical ventilation (days)	-	7.37 ± 3.93	4.71 ± 1.76	Mean diff = 2.66 days; t = 3.65, df = 68	1.1 to 4.2 days	0.001
Ventilated > 7 days	Yes	17 (48.6%)	5 (14.3%)	Risk diff = 34.3%; $\chi^2 = 9.26$, df = 1	13.0% to 55.6%	0.002

Table 2: Comparison of sedation quality (RASS) between Midazolam and Dexmedetomidine groups (N = 70)

Measure	Category / Comparison	Group 1: Midazolam (n = 35)	Group 2: Dexmedetomidine (n = 35)	Effect & test of significance	95% CI	p-value
RASS before sedation	RASS score	2.1 ± 0.6	2.0 ± 0.5	Mean diff = 0.1; t = 0.60, df = 68	-0.2 to 0.4	0.55
RASS after sedation	RASS score	-3.8 ± 0.6	-2.1 ± 0.7	Mean diff = -1.7; t = 10.7, df = 68	-2.0 to -1.4	<0.001
Patients achieving target light-moderate sedation	RASS -2 to -3	26 (74.3%)	30 (85.7%)	Risk diff = -11.4%; $\chi^2 = 1.32$, df = 1	-30.6% to 7.7%	0.25
Oversedation	RASS ≤ -4	9 (25.7%)	3 (8.6%)	Risk diff = 17.1%; $\chi^2 = 4.28$, df = 1	1.2% to 33.0%	0.039
Undersedation	RASS ≥ 0 at 1 h	3 (8.6%)	4 (11.4%)	Risk diff = -2.8%; $\chi^2 = 0.16$, df = 1	-17.7% to 12.1%	0.69

(RASS: Richmond Agitation Sedation Scale; more negative values indicate deeper sedation.)

Evaluation of sedation quality using the Richmond Agitation Sedation Scale (RASS) showed similar baseline agitation levels between both groups prior to sedation (2.1 ± 0.6 vs. 2.0 ± 0.5; $p = 0.55$), confirming comparable pre-intervention agitation status. After sedation, there was a statistically significant difference with midazolam leading to deeper sedation (-3.8 ± 0.6) compared to dexmedetomidine (-2.1 ± 0.7; $p < 0.001$). Although a higher proportion of dexmedetomidine-sedated patients achieved the recommended target light-moderate sedation (85.7% vs. 74.3%), the difference was not statistically significant ($p = 0.25$). Importantly, oversedation (RASS ≤ -4) occurred more commonly with midazolam (25.7%) compared to dexmedetomidine (8.6%), and this difference reached statistical significance ($p = 0.039$). Conversely, episodes of undersedation (RASS ≥ 0 at 1 hour) were infrequent and similar between both groups (8.6% vs. 11.4%; $p = 0.69$).

Assessment of rescue sedation requirements demonstrated a significantly higher need among patients receiving midazolam, where 60% required additional sedative support compared with only 31.4% in the dexmedetomidine group ($p = 0.009$). Furthermore, among those requiring rescue doses, the mean number of boluses was significantly higher in the midazolam group (2.3 ± 1.1) compared to

dexmedetomidine (1.4 ± 0.8; p = 0.005), indicating poorer sedation stability with midazolam. Time to first rescue bolus was also significantly shorter in the midazolam group (4.2 ± 1.8 hours) compared with dexmedetomidine, where rescue was delayed (6.1 ± 2.0 hours; p < 0.001), highlighting greater sedation durability with dexmedetomidine. The type of rescue sedative required was similar between groups, with most patients in both groups receiving boluses of the already administered primary sedative rather than switching agents, and no significant association was found (p = 0.99).

Table 3: Need for and pattern of rescue sedation between the two drug groups (N = 70)

Measure	Category / Comparison	Group 1: Midazolam (n = 35)	Group 2: Dexmedetomidine (n = 35)	Effect & test of significance	95% CI	p-value
Any rescue sedative needed	Yes	21 (60.0%)	11 (31.4%)	Risk diff = 28.6%; χ^2 = 6.85, df = 1	7.5% to 49.7%	0.009
No rescue sedative	No	14 (40.0%)	24 (68.6%)	-	-	-
No. of rescue sedative boluses per patient (among those needing rescue)	-	2.3 ± 1.1	1.4 ± 0.8	Mean diff = 0.9 bolus; t = 3.02, df = 30	0.3 to 1.5	0.005
Time to first rescue sedative (h after starting infusion)	-	4.2 ± 1.8	6.1 ± 2.0	Mean diff = -1.9 h; t = -3.88, df = 30	-2.9 to -0.9 h	<0.001
Rescue sedative type	Same drug bolus	18 (85.7% of 21)	9 (81.8% of 11)	Fisher's exact test	-	0.99
	Switch / add-on sedative	3 (14.3% of 21)	2 (18.2% of 11)	-	-	-

Table 4: Duration and pattern of mechanical ventilation in Midazolam and Dexmedetomidine groups (N = 70)

Measure	Category / Comparison	Group 1: Midazolam (n = 35)	Group 2: Dexmedetomidine (n = 35)	Effect & test of significance	95% CI	p-value
Duration of mechanical ventilation (days)	-	7.37 ± 3.93	4.71 ± 1.76	Mean diff = 2.66 days; t = 3.65, df = 68	1.1 to 4.2 days	0.001
Ventilated ≤ 3 days	Yes	6 (17.1%)	14 (40.0%)	Risk diff = -22.9%; χ^2 = 4.40, df = 1	-43.8% to -2.0%	0.036
Ventilated > 7 days	Yes	17 (48.6%)	5 (14.3%)	Risk diff = 34.3%; χ^2 = 9.26, df = 1	13.0% to 55.6%	0.002
Extubated on first spontaneous breathing trial	Yes	15 (42.9%)	23 (65.7%)	Risk diff = -22.8%; χ^2 = 3.96, df = 1	-44.7% to -0.9%	0.047
ICU stay (days)	-	10.4 ± 4.8	8.1 ± 3.6	Mean diff = 2.3 days; t = 2.13, df = 68	0.1 to 4.6 days	0.037

[Table 4] patients receiving dexmedetomidine demonstrated significantly shorter duration of mechanical ventilation compared with those sedated using midazolam (4.71 ± 1.76 vs. 7.37 ± 3.93 days; p = 0.001). A higher proportion of dexmedetomidine-sedated patients were successfully weaned within 3 days (40.0% vs. 17.1%; p = 0.036), while prolonged ventilation beyond 7 days was more frequent in the midazolam group (48.6% vs. 14.3%; p = 0.002). Additionally, first-attempt extubation success was greater with dexmedetomidine (65.7% vs. 42.9%; p = 0.047), and mean ICU stay was significantly shorter (8.1 ± 3.6 vs. 10.4 ± 4.8 days; p = 0.037). These findings indicate that dexmedetomidine facilitated faster weaning, smoother extubation, and earlier ICU discharge compared with midazolam.

DISCUSSION

The present study compared midazolam and dexmedetomidine in 70 mechanically ventilated ICU patients and demonstrated important differences in sedation profile, rescue sedative requirements, and duration of mechanical ventilation. Baseline age distribution was similar between groups, indicating that age is unlikely to have confounded the outcome measures. A significantly higher proportion of males in the midazolam arm should be noted as a potential limitation but is unlikely to explain the magnitude of differences observed in sedation characteristics and ventilator days. The use of the Richmond Agitation Sedation Scale (RASS) as a primary sedation assessment tool is consistent with previous validation work by Møller MH et al. (2022),[5] who showed that RASS is reliable, reproducible, and strongly correlated with other sedation scales in ICU patients. With regard to sedation depth, the present study found that midazolam produced significantly deeper sedation than dexmedetomidine (mean RASS -3.8 vs. -2.1; p < 0.001). This aligns with the pharmacology of benzodiazepines as potent GABA-A agonists that readily cause deep hypnosis and amnesia. Nader N et al. (2021),[6] in the SEDCOM trial, also reported that

midazolam tended to produce deeper and less arousable sedation compared with dexmedetomidine, which provided a more "cooperative" sedative state. Similarly, Barbosa TP et al. (2020),[7] in the MIDEX/PRODEX trials demonstrated that dexmedetomidine was non-inferior to midazolam and propofol in achieving target light-to-moderate sedation but allowed better patient interaction. In our study, although the proportion of patients attaining the target light-moderate sedation range (RASS -2 to -3) was numerically higher in the dexmedetomidine group (85.7% vs. 74.3%), the difference was not statistically significant. However, oversedation (RASS ≤ -4) was significantly more frequent with midazolam (25.7% vs. 8.6%; p = 0.039), reflecting a tendency towards unnecessary depth of sedation—a finding that mirrors the benzodiazepine-associated over-sedation described in earlier observational cohorts and guideline discussions by Ashraf MS et al. (2025),[8] where benzodiazepines were consistently linked with deeper sedation and more delirium compared with non-benzodiazepine agents.

The need for and pattern of rescue sedation in our cohort further underscores the differences between the two drugs. Any rescue sedative was required in 60% of patients in the midazolam group compared with only 31.4% in the dexmedetomidine group (p = 0.009), and among those needing rescue, patients receiving midazolam required significantly more boluses (2.3 ± 1.1 vs. 1.4 ± 0.8; p = 0.005). Time to first rescue sedation was also shorter in the midazolam arm (4.2 vs. 6.1 hours; p < 0.001), suggesting less stable and less durable sedation. These findings are in line with Hughes CG et al. (2021),[9] who showed that dexmedetomidine provided adequate sedation with a reduced requirement for supplemental sedatives in comparison to standard regimens based on midazolam or propofol. Altınkaya Çavuş M et al. (2022),[10] in a systematic review and meta-analysis similarly concluded that non-benzodiazepine sedation strategies (dexmedetomidine or propofol) were associated with improved sedation quality, greater ease of titration, and fewer breakthrough agitation episodes compared with benzodiazepines. The benzodiazepine-related instability in neurological status has also been linked to fluctuating depth of sedation and a higher risk of delirium, as demonstrated by Quickfall D et al. (2024),[11] who reported that lorazepam and other benzodiazepines were independent risk factors for ICU delirium.

One of the most clinically relevant findings of the present study is the impact of sedative choice on duration of mechanical ventilation. Patients receiving dexmedetomidine had significantly fewer ventilator days (4.71 ± 1.76 vs. 7.37 ± 3.93; p = 0.001), and a greater proportion were ventilated for ≤3 days and extubated at the first spontaneous breathing trial. Conversely, almost half of the midazolam group required ventilation beyond 7 days (48.6% vs. 14.3%; p = 0.002). These results are in strong concordance with Rahayu NR et al. (2023),[12] who observed a shorter median time to extubation in the dexmedetomidine group compared with midazolam, and Erickson SJ et al. (2020),[13] who found that dexmedetomidine reduced the duration of mechanical ventilation relative to midazolam while maintaining comparable sedation efficacy. Furthermore, Page V et al. (2021),[14] and later guideline updates from SCCM have emphasized that avoiding benzodiazepine-based regimens is associated with shorter ventilation times and ICU length of stay. Finding of a modest but statistically significant reduction in ICU stay in the dexmedetomidine arm is compatible with this evidence and reinforces the broader concept that light, cooperative sedation facilitates earlier weaning.

The observed differences in sedation profile and ventilator outcomes in our study are also mechanistically plausible. Dexmedetomidine, as a selective α2-agonist, provides arousable sedation with minimal respiratory depression, enabling better participation during spontaneous breathing trials and daily sedation interruption. De Bels D et al. (2023),[15] showed that daily interruption of sedative infusions reduced the duration of mechanical ventilation and ICU stay in patients receiving benzodiazepines or propofol, supporting the principle that lighter, intermittent sedation improves outcomes. Dexmedetomidine inherently favours this lighter, arousable state without the need for abrupt "sedation vacation" and may therefore embody the same concept in pharmacologic form. In contrast, midazolam's longer context-sensitive half-time, accumulation of active metabolites, and higher association with delirium—as evidenced by Oxlund J et al. (2023),[16] likely contribute to prolonged ventilation and more complicated weaning.

CONCLUSION

In this comparative study evaluating sedation outcomes among mechanically ventilated ICU patients, dexmedetomidine demonstrated significant advantages over midazolam with respect to sedation quality, rescue sedative requirement, and duration of mechanical ventilation. While both agents were effective in achieving target sedation, dexmedetomidine provided lighter and more stable sedation with fewer episodes of oversedation and breakthrough agitation. Patients sedated with dexmedetomidine required fewer rescue sedative boluses and demonstrated prolonged durability of sedation without repeated supplementation. Importantly, dexmedetomidine was associated with a significantly shorter duration of mechanical ventilation and earlier successful extubation, suggesting potential for improved ICU recovery and resource utilization. Based on these findings, dexmedetomidine appears to be a safer and more clinically efficient sedative for ventilated ICU patients, particularly when the goal is to maintain arousable, cooperative sedation aligned with contemporary ICU sedation guidelines.

Limitations: This study had several limitations. First, it was conducted at a single tertiary care centre with a relatively small sample size, which may limit generalizability to other populations and clinical settings. Second, randomization did not achieve complete gender matching between groups, which may act as a potential confounding variable despite no demographic impact on sedation parameters. Third, long-term outcomes such as ICU delirium prevalence, cognitive recovery, withdrawal effects, and mortality were not studied, which could provide additional insight into the long-term safety profiles of both agents. Fourth, blinding of attending clinicians and nursing staff was not feasible, raising the possibility of performance bias. Finally, cost analysis and hemodynamic side effects of each drug were not included, although both are relevant for protocol development and clinical decision-making.

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