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Original Research Article

A Study to Evaluate the Efficacy of Dexmedetomidine Pretreatment on the Prevention of Etomidate Induced Myoclonus during Induction of Anaesthesia: A Randomised Double-Blind Prospective Study

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Abstract

Background: Etomidate is a commonly used intravenous agent because of its stable haemodynamic properties but the myoclonus following etomidate induction may be the distressing and clinically undesirable effect.

Aim & Objectives: To evaluate the efficacy of dexmedetomidine pre-treatment in reducing the incidence and severity of etomidate induced myoclonus during induction of general anaesthesia.

Material & method: In this double blind prospective, randomized study of 88 adult surgical patients, allocated into two equal groups: Group I received Dexmedetomidine (0.5ug/kg) pre-treatment and Group II received saline over 10 minutes prior to 0.3 mg/kg Etomidate induction. Patients were observed continuously for 1 minute for the occurrence and severity of myoclonus. Haemodynamics and saturation were recorded at different intervals and complication were noted if occurred. The presentation of the Categorical variables was done in the form of number and percentage (%). The quantitative data were presented as the means ± SD.

Result: Myoclonus was absent in 77.27% of patients in the Dexmedetomidine group versus saline group ($p < 0.0001$). Moderate and severe myoclonus were also markedly lower in the Dexmedetomidine group. Hemodynamic parameters showed favourable trends. While systolic blood pressure (SBP) was significantly lower in the Dexmedetomidine group from 4 minutes post-induction onward ($p < 0.0001$), values remained clinically stable. Diastolic blood pressure (DBP) and heart rate were largely comparable, with a few intervals showing significantly lower values in the Dexmedetomidine group, suggesting its expected hypotensive and mild bradycardia effects. Oxygen saturation (SpO_2) remained stable in both groups throughout.

Conclusion: Dexmedetomidine in a dose of 0.5 µg/kg can be used safely as premedication prior to the induction with etomidate to reduce Etomidate induced myoclonus.

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Introduction

Induction of anaesthesia is a critical phase in providing general anaesthesia. An ideal inducing agent should provide smooth and pleasant induction with good haemodynamic stability and minimal side effects. Etomidate is widely used induction agent as it provides better cardio stability and minimal changes in heamodynamics during general anaesthesia induction than propofol.[1,2] It is preferred in patients with respiratory and airway disease, intracranial hypertension and in patient with shock.[3] Etomidate is a drug of choice in cardiac surgeries and hemodynamically unstable patient posted for surgeries. [4] It is an imidazole derivative has good hemodynamic stability, lesser respiratory depression, favorable toxicity profile and pharmacokinetics and rapid recovery after a

single dose.[5] Though it has been a good choice of induction agent but it has been associated with pain on injection and myoclonus. 6The incidence of myoclonus in non-premedicated patients has been reported to be high as 50-80%.[7] Various drugs are used as a pre-medication to reduce the myoclonic jerks induced by etomidate like midazolam, opioids, magnesium sulphate, lignocaine, dezosine, dexamethasone but none of them succeed to abolish it completely.[8-16] DEx has been recently tried to reduce the incidence and severity of myoclonus due to its anxiolytic properties and by subcortical inhibition. [16-23]. Due to limited availability of literature on dexmedetomidine pretreatment, we had done a research study on the efficacy of dexmedetomidine infusion in reducing the incidence and severity of myoclonus during etomidate induction. The aim was to evaluate the efficacy of dexmedetomidine pre-treatment in reducing the incidence and severity of etomidate induced myoclonus during induction of general anaesthesia with primary objective of comparing the incidence and the severity of myoclonus in the groups. The Secondary objectives were to compare the hemodynamic parameters in both the groups and to observe and evaluate any adverse effect secondary to dexmedetomidine in group-I (Dexmedetomidine).

Material & Method

A double-blind comparative interventional Prospective study was initiated after obtaining permission from the institutional ethics committee (ECARP/2024/22 letter no). We have taken a total sample size of 88 based on H.F Luan et. al. study, divided into 2 equal groups of 44 each, i.e., Group I (n=44): dexmedetomidine pretreatment with etomidate induction and Group II (n=44): saline pretreatment with etomidate induction as per random allocation using a computer-generated random number table. The inclusion criterias were patients aged between 20-65 years of either sex belonged to ASA (American society of anesthesiology) physical status I/II. Specific exclusion criteria were patient's refusal, allergy to drugs, musculoskeletal disorders, neurological disorders, seizure disorder and patients with significant cardiac, respiratory, hepaticorenal dysfunction.

After confirming nil per oral status and written informed consent for surgery, the patients were taken inside the operation theatre. Baseline pre-operative parameters were recorded after attaching monitors to the patients i.e. electrocardiogram, non-invasive blood pressure monitoring and pulse oximetry. After establishing intravenous excess with 18gauge intravenous cannula, crystalloid infusion was started.

Before anaesthesia induction in group I, 0.5 µg/kg dexmedetomidine in 100mL normal saline was infused over 10 minutes. Infusion was made by anaesthesiologist who was not been observing for myoclonus. In group II, 100 mL NS was infused over 10 minutes. After complete infusion, 0.3 mg/kg etomidate was injected i.v. over a period of 30 seconds. Patients were observed continuously for myoclonus for 1 minute after injection of etomidate by an anaesthesiologist, who was blinded to all group allocations. Intensity of myoclonus was graded as 0 (none), I (mild: movement at the finger or wrist only), II (moderate: involving the face and leg), or III (severe: generalized response or fast abduction of a limb). After administration of etomidate and evaluation of myoclonus, all patients were injected with 2 µg/kg fentanyl [l]. 1 minute after the end of etomidate injection, muscle relaxation was achieved with 0.08mg/kg vecuronium and endotracheal intubation was performed after another 2 minutes. In both the groups myoclonus, haemodynamic parameters (heart rate, systolic blood pressure, diastolic blood pressure, mean arterial blood pressure, oxygen saturation) was recorded before and after induction and intubation. From the onset of action of Dexmedetomidine or normal saline to the injection of etomidate, adverse effects was recorded by one anaesthesiologist, who was blinded to the group allocation and not to be involved further in the study in order to avoid the bias of the outcome assessors who had observed myoclonus. If myoclonus jerk occurs, we had not allowed the seizures to happen in the control group by increasing the dose of anaesthetic agent as rescue drug. After completion of surgery reversal of neuromuscular blockade and extubation was done as per standard protocol. The systolic blood pressure was < 90mmhg or diastolic blood pressure was <60 mmHg, ephedrine 5-10 mg was administered. If the heart rate was <50 beats/min, glycopyrrolate 0.004 mg/kg was administered at once. Complications like nausea, vomiting, hypotension, bradycardia, respiratory depression, agitation, arrhythmia was observed. Patients were observed for 2 hours in PACU (post anaesthesia care unit) for any postoperative complications like nausea, vomiting, hypotension, bradycardia, respiratory depression, agitation, and arrhythmia. Any complications that occurs, was treated with appropriate measures. All data was filled up in the proforma attached.

Statistical Analysis: The analysis was run on Social Sciences (SPSS) software, IBM manufacturer, Chicago, USA, ver 25.0. The presentation of the Categorical variables was done in the form of number and percentage (%). The quantitative data were presented as the means ± SD and as median with 25th and 75th percentiles (interquartile range). The data normality was checked by using Shapiro-Wilk test. The comparison of the variables which were quantitative in nature were analysed using

Independent t test. The comparison of the variables which were qualitative in nature were analysed using Chi-Square test. If any cell had an expected value of less than 5 then Fisher's exact test was used. p value of less than 0.05 was considered statistically significant.

Results

Myoclonus was absent in 77.27% of patients in the Dexmedetomidine group versus only 22.73% in the saline group ($p < 0.0001$). Moderate and severe myoclonus were also markedly lower in the Dexmedetomidine group. The incidence of myoclonus was significantly higher in group II as compared to group I. (77.27% vs. 22.73%) (P value < 0.0001). Also the occurrence of grade 2 (47.06% vs 10%) & grade 3 (26.47% vs 0%) myoclonus was significantly higher in group

II as compared to group I.(P value=0.001). Only 9 patients out of 10 patients showed grade-I myoclonus in myoclonus group of group I. Systolic blood pressure was significantly lower in the Dexmedetomidine group from 4 minutes post-induction onward ($p< 0.0001$), values remained clinically stable. Diastolic blood pressure (DBP) and heart rate were largely comparable, with a few intervals showing significantly lower values in the Dexmedetomidine group. Oxygen saturation (SpO2) remained stable in both groups throughout. Baseline characteristics such as age, gender, BMI, and ASA grade were well-matched across groups, confirming successful randomization and minimizing confounders. Regarding complications, Group I had fewer (2.27%) compared to Group II (15.91%), though the difference did not reach statistical significance ($p = 0.058$). Adverse effects like bradycardia, nausea, and vomiting were minimal and statistically comparable between groups, indicating good tolerability of Dexmedetomidine.

Table 1: Mean Age, Height, Weight, and Body Mass Index in both Groups.

Variables	Group 1(n=44)	Group 2(n=44)	P value
Age (yrs)	38.91 ± 10.6	34.61 ± 11.64	0.074‡
Height (cm)	163.07 ± 7.45	160.57 ± 6.96	0.098‡
Weight (kg)	61.14 ± 8.3	58.36 ± 7.2	0.107‡
BMI (kg/m^2)	22.9 ± 1.97	22.6 ± 2.12	0.502‡

‡ Independent t test

Table 2: Systolic Blood Pressure SBP

Time (Minutes)	Group I(n=44) Mean ± SD	Group II(n=44) Mean ± SD	P value
0	121.77 ± 7.45	120.32 ± 6.29	0.325‡
2	119.09 ± 8.79	115.86 ± 7.09	0.061‡
4	117.64 ± 6.89	123.68 ± 7.58	<.0002‡
6	113.64 ± 6.89	119.45 ± 5.51	<.0001‡
8	110.18 ± 7.14	123.95 ± 10.95	<.0001‡
10	106.32 ± 5.1	121.95 ± 9.31	<.0001‡
15	113.95 ± 7.29	126.95 ± 9.62	<.0001‡
20	108.91 ± 7.08	120.68 ± 7.27	<.0001‡
25	109.14 ± 9.17	118.95 ± 7.33	<.0001‡
30	107.55 ± 7.48	118.23 ± 7.54	<.0001‡
40	108.23 ± 8.39	118.45 ± 9.3	<.0001‡
50	108.27 ± 7.4	119.36 ± 7.68	<.0001‡
60	109.45 ± 9.09	117.86 ± 8.45	<.0001‡
90	110.14 ± 8.03	120.18 ± 7.96	<.0001‡
120	113.23 ± 8.43	120.27 ± 10.23	0.0007‡

‡Independent t test

Tables 3: myoclonus and its grade in Group I and Group II

	Group I	Group II	Total	P value
Myoclonus				
No	34 (77.27%)	10 (22.73%)	44 (50%)	<.0001†
Yes	10 (22.73%)	34 (77.27%)	44 (50%)	
Myoclonus grade				
Grade 1	9 (90%)	9 (26.47%)	18 (40.91%)	0.001*
Grade 2	1 (10%)	16 (47.06%)	17 (38.64%)	
Grade 3	0 (0%)	9 (26.47%)	9 (20.45%)	

*** Fisher's exact test, † Chi square test**

Discussion

We concluded that the incidence and severity of Etomidate induced myoclonus, was significantly decreased with 0.5 µg /kg Dexmedetomidine 10 minutes prior to the induction without producing significant adverse effect. Hence Dexmedetomidine can be used safely as premedication prior to the induction with etomidate to reduce etomidate induced myoclonus.

In the present study the primary objective was to assess the incidence and severity of etomidate-induced myoclonus, along with hemodynamic stability and adverse effects as secondary objectives. Baseline characteristics such as age, gender, BMI, and ASA grade were well-matched across groups, confirming successful randomization and minimizing confounders.

We have chosen Dexmedetomidine 0.5 µg/kg to obtain an optimal attenuation. In the present study, the incidence of Etomidate-induced myoclonus in the Dexmedetomidine group was in concordance with the findings of studies by Mizrak et al., [19] Luan et al., [20] and Miao et al. [22] where it was compared to other agents (midazolam, Thiopentone and placebo) and in a dose of 0.5 µg/kg. This denotes that Dexmedetomidine in a dose of 0.5 µg/kg showed almost uniform prevention of myoclonus across different populations.

In our study in Group 1, 77.27% of patients did not experience any myoclonus, while only 22.73% found myoclonus and none develop severe myoclonus in stark contrast to Group 2, and, where 77.27% of patients experienced myoclonus and only 22.73% remained unaffected ($p < 0.0001$). Dey & Kumar [23] in their study, compared Dexmedetomidine (0.5 µg/kg, Group D) with midazolam (0.015 mg/kg, Group M), In their study the Dexmedetomidine group, 55% of patients had no myoclonus and none developed severe (grade III) myoclonus. In contrast, in the midazolam group, 47.5% experienced moderate (grade II) and 15% developed severe (grade III) myoclonus. Miao S et al. [22] in their study, found the incidence of myoclonus in group 1 (Dexmedetomidine) vs. group 2 (Saline) 26% vs. 64%. Pre-treatment with Dexmedetomidine 0.5 µg /kg allows for a 38% reduction in the incidence of Etomidate-induced myoclonus, as well as reducing the severity of myoclonus without inducing any adverse effects. Luan et al. [20] in their study they found the incidence of myoclonus 36.7% vs. 63.3% in Dexmedetomidine vs normal saline group in their study. Ghodki et al. [24] also noted the incidence of myoclonus 40% vs 66.7% in Dexmedetomidine vs. saline, ($P = 0.023$). Our findings concur with the above investigators.

The severity of Etomidate-induced myoclonus was significantly lower in the Dexmedetomidine group (Group 1) compared to the saline group (Group 2). Further analysis of myoclonus severity revealed that moderate (Grade II) and severe (Grade III) myoclonus were markedly lower in Group 1, with Grade II observed in only 10% of patients compared to 47.06% in Group 2, and no cases of Grade III myoclonus in Group 1 compared to 26.47% in Group 2 ($p = 0.001$). Dey & Kumar [23] Observed 10% of patients in the Dexmedetomidine group had Grade II myoclonus as compared to 47.5% in the midazolam group. 7.5% of patients in midazolam group experienced Grade III myoclonus whereas none of the patients in the Dexmedetomidine group with grade III myoclonus. Miao S et al.[22] revealed moderate grade (grade II) in 5/50 vs. 10/50 patients and severe grade (grade III) in 1/50 vs. 14 /50 patients in Dexmedetomidine vs. normal saline group. Luan et al. [20] revealed moderate grade (grade II) in 2/30 vs. 6/30 patients and severe grade (grade III) in 0/30 vs. 3 /30 patients in Dexmedetomidine vs. NS group. Therefore, the results of our study are in concordance with the above studies which finds Dexmedetomidine efficacious in suppressing Etomidate-induced myoclonus.

The hemodynamic parameter such as systolic blood pressure (SBP) was comparable between the two groups at baseline (0 min) and 2 minutes post-induction. However, from 4 minutes onwards, Group 1 (Dexmedetomidine) consistently showed significantly lower SBP compared to Group 2 (Saline), with p-values < 0.0001 at most time points up to 120 minutes. This sustained reduction in SBP in Group 1 highlights the hypotensive effect of Dexmedetomidine, likely due to its central sympatholytic and peripheral vasodilatory actions. Despite this, values remained within clinically acceptable limits, suggesting hemodynamic stability rather than compromise. Overall, diastolic blood pressure remained comparable between the two groups at most time points. Group 1 had a significantly higher DBP at baseline (77 ± 7.16 mmHg vs. 73.64 ± 7.03 mmHg, $p = 0.029$) and at 2 minutes (75.3 ± 6.91 mmHg vs. 69.73 ± 7.84 mmHg, $p = 0.0007$), possibly due to pre-induction variability. Group 1 showed a significantly lower DBP at 8 minutes ($p = 0.016$) and 50 minutes ($p = 0.003$) compared to Group 2, indicating the onset of Dexmedetomidine hypotensive effect. However, no significant difference was observed at most other intervals ($p > 0.05$), reflecting overall hemodynamic stability in both groups. Dey and Kumar [23] in their study observed both Dexmedetomidine and midazolam infusions led to a decrease in the mean BP from the baseline at various time intervals, but Dexmedetomidine group showed significant reduction ($P < 0.05$) from the baseline mean BP as compared to the midazolam

group. However, Rautela R et al. [25] did not find any significant difference in mean blood pressure with a P-value of 0.268 between the groups.

Heart rate remained largely comparable between the two groups across the various time intervals ($p > 0.05$), indicating overall hemodynamic stability. However, Group 1 exhibited significantly lower pulse rates at: 8 min (77.34 ± 6.31 bpm vs. 79.93 ± 5.1 bpm, $p = 0.037$), 25 min (75.75 ± 5.86 bpm vs. 78.39 ± 5.91 bpm, $p = 0.039$), 40 min (75.59 ± 5.76 bpm vs. 78.16 ± 5.45bpm, $p = 0.034$), and 50 min (74.64 ± 5.61 bpm vs. 78.11 ± 5.66 bpm, $p = 0.005$). These findings suggest a slight decrease in heart rate which was statistically not significant in Dexmedetomidine Group 1, consistent with its known pharmacodynamic profile. Dey and Kumar [20] found both Dexmedetomidine and midazolam infusions led to a decrease in the heart rate from the baseline at various time intervals, but Dexmedetomidine group showed significant reduction ($P < 0.05$) from the baseline heart rate as compared to the midazolam group.

There was no statistically significant difference in SpO2 values between Group 1 and Group 2 at any time point (all $p > 0.05$). Both groups maintained consistently high oxygen saturation levels throughout the observation period, indicating adequate respiratory function and oxygenation irrespective of pre-treatment with Dexmedetomidine.

In our study, the adverse effects following etomidate induction were assessed and compared between Group 1 (Dexmedetomidine pre-treatment) and Group 2 (Saline pretreatment). The findings revealed no statistically significant difference in the incidence of adverse effects between the two groups. Bradycardia was reported in 1 patient (2.27%) in Group 1, while no cases were observed in Group 2 (0%) ($p = 1.000$). Nausea occurred in 2 patients (4.55%) in Group 1 and 3 patients (6.82%) in Group 2 ($p = 1.000$). Vomiting was seen in 1 patient (2.27%) in both groups ($p = 1.000$). No patient reported to experience dizziness, respiratory depression. Miao et al. [22] also found one patient with hypotension and two patients with bradycardia in the Dexmedetomidine group in their study. Luan et al. [20] studied 3 groups in which group 1 of normal saline and group 2 with 0.5 µg /kg of Dexmedetomidine and group 3 with 1 µg /kg Dexmedetomidine for prevention of etomidate induced myoclonus and he found the incidence of low blood pressure 6.7%, 10% and 13.3% and bradycardia 0%, 6.7% and 24.0% in groups 1, 2 and 3 respectively. He concluded that the dose of Dexmedetomidine with 1 µg/kg was associated with the higher incidence of hypotension and bradycardia. In our study the Dexmedetomidine 0.5 µg/kg in the form of a single injection over 10 minutes did not produce statistically significant bradycardia and in the pharmacological literature of Dexmedetomidine, it has been reported that the bradycardia is a dose and duration dependent known adverse effect. Our study design had some limitations. We did not measure plasma cortisol and adrenocorticotropic hormone levels. We only recorded the incidence and severity of Etomidate induced myoclonus except the duration of myoclonus that might help detect differences in the severity of myoclonus.

Conclusion

Dexmedetomidine in a dose of 0.5 µg/kg can be used safely as premedication prior to the induction with etomidate to reduce Etomidate induced myoclonus.

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