Section: Anaesthesiology

Original Research Article

ROLE OF DEXMEDETOMIDINE IN REDUCING MYOCLONIC MOVEMENTS ASSOCIATED WITH ETOMIDATE INDUCTION

Dhivya B.C.[1], Arthy E.[2], B. Megala[3]

[1]Assistant Professor, Department of Anaesthesiology, Government medical College and Hospital, Pudukkottai, Tamilnadu, India.
[2]Assistant Professor, Department of Anaesthesiology, Government medical College and Hospital, Pudukkottai, Tamilnadu, India.
[3]Assistant Professor, Department of Anaesthesiology, Government medical College and Hospital, Pudukkottai, Tamilnadu, India.

Received : 12/06/2025
Received in revised form : 07/08/2025
Accepted : 29/08/2025

Keywords:
Dexmedetomidine; Myoclonus; Etomidate; Ramsay sedation score.

Corresponding Author:
Dr. Dhivya B.C.,
Email: drdhivyabc@gmail.com

DOI: 10.47009/jamp.2025.7.5.12

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2025; 7 (5); 56-62

ABSTRACT

Background: Etomidate is a popular intravenous induction, but myoclonus is observed in 50%–80% of patients who did not receive pretreatment before etomidate administration. Hence the present study was performed to study the effect of dexmedetomidine in the prevention of myoclonus occurring due to Etomidate induction. **Materials and Methods:** A prospective randomized, controlled, double-blind study was conducted on seventy patients aged 18-55 years belonging to ASA I and II scheduled for elective surgery under general anaesthesia. A detailed pre-anaesthetic evaluation was done, and investigations were obtained as indicated. The patients were randomized into two groups, with 35 patients each, receiving either 0.5 µg/kg of dexmedetomidine in 10 ml saline (Group D) or 10 ml of Saline (Group S) for 10 minutes before etomidate induction. **Result:** The incidence of severe myoclonus was significantly less in group D compared to a saline group with p = 0.031 (8.57% in group D and 28.5% in group S). However, both groups had no change in myoclonus incidence (P = 0.237) and pain on injection (p = 0.309). The recovery profile was comparable in both groups. Post extubation Ramsay sedation score was significantly higher in group D compared to group S at the 30th, 60th and 90th minute with p-value = 0.001, 0.001 and 0.051, respectively. There was no significant effect on incidences of nausea and vomiting between both groups. **Conclusions:** Our study shows that pretreatment with dexmedetomidine 0.5 µg/kg IV effectively reduces the severity of etomidate-induced myoclonic muscle movements without affecting myoclonus incidences.

INTRODUCTION

The discovery of IV anaesthetics has long been an important milestone in developing anaesthesia. Before this, induction of general anaesthesia necessarily required inhalation of gases or vapour, which was an unpleasant experience for most patients.[1] Etomidate is a carboxylated imidazole drug used for induction of general anaesthesia and sedation and was introduced into clinical practice in 1973. Etomidate is a popular anaesthetic induction agent because it has a stable haemodynamic profile and results in minimal histamine release. Previous studies reported that at 0.3 mg/kg induction doses, etomidate does not cause significant alterations in heart rate, systolic, diastolic, and mean arterial pressures, right atrial pressure, systemic and pulmonary vascular resistance, stroke volume, cardiac index, systemic blood flow, and shunt flow in paediatric patients undergoing congenital cardiac shunt surgery and in adults.[2]

Two undesirable side effects of etomidate are pain on injection and myoclonus. Pain on injection, venous irritation and haemolysis have been abolished by a new fat emulsion of etomidate (medium-chain triglyceride and soya bean named etomidate -lipuro, Germany). Still, the new solvent has not reduced the incidence of myoclonus after Etomidate injection. The induction dose of etomidate is 0.2 to 0.4 mg/kg. Myoclonus is observed in 50% – 80% of patients who did not receive pretreatment before Etomidate administration. Involuntary myoclonic movements are common during the induction period as a result of subcortical dis-inhibition and are unrelated to cortical seizure activity.[2,3]

Myoclonus may be of clinical significance in various patients undergoing general anaesthesia induction. In theory, in emergency conditions, the myoclonus may increase the risk of regurgitation and aspiration. As a

ISSN (O): 2687-5365; ISSN (P): 2753-6556

result of high intraocular pressure, myoclonus might increase the risk of vitreous prolapse after an open globe injury. In the case of electric cardioversion, continuous electrocardiogram (ECG) recordings may be disturbed due to the patient's myoclonic movements.[3]

Although Etomidate-induced myoclonus's mechanism is still unclear, several drugs have been investigated for their ability to suppress these myoclonic movements. Opioids such as fentanyl, Sufentanil and Remifentanil, benzodiazepines, magnesium sulfate, low-dose Etomidate and rocuronium have been shown to reduce myoclonus to some extent. But even with these drugs, myoclonus was still observed at a rate of 7% - 50%.[4] The carboxylated imidazole etomidate exhibits structural similarities to specific alpha-2 adrenoceptor agonists that belong to the class of imidazole compounds, such as clonidine and dexmedetomidine. Besides the chemical structure, etomidate and alpha-2 adrenoceptor agonists share some clinical similarities, such as inducing sedation/ hypnosis with high cardiovascular stability and only minor respiratory depression. Several authors have tried dexmedetomidine to suppress the myoclonus induced by Etomidate.[5,6]

Thus, the present clinical study was undertaken to investigate the effects of pretreatment with dexmedetomidine on the incidence and severity of myoclonus and injection pain during induction of general anaesthesia with etomidate in elective surgery patients.

MATERIALS AND METHODS

The Present Prospective, randomized control trial, double-blind study was performed on 70 patients in Victoria Hospital and Bowring and Lady Curzon Hospitals attached to Bangalore Medical College and Research Institute between November 2014 and May 2016. All enrolled subjects were divided into Group D (Dexmedetomidine) and Group S (Saline), each with 35 subjects. Institutional ethical committee approval and written consent were taken before the start of the study.

Inclusion Criteria: Patients aged 18-55, ASA physical status I and II, and patients who gave informed written consent were included.

Exclusion Criteria: Patients with cardiovascular, hepatic, renal, epilepsy, adrenal and respiratory diseases. Patients with chronic abuse of alcohol, drugs, and psychotropic agents. Patients who were pregnant and lactating were excluded.

Methodology

Pre-anaesthetic evaluation and preparation: A thorough pre-anaesthetic check-up was done for all patients a day before surgery. No special investigations were required for the study. Preoperative investigations, including complete blood count (CBC), urine examination, blood sugar, serum electrolytes, coagulation profile, liver and renal function tests, electrocardiography and echocardiography, and chest x-ray, were obtained as indicated. They were advised to fast from the night before the day of surgery. Premedication with oral ranitidine hydrochloride 150 mg and alprazolam 0.25 mg were given the night before surgery.

Preparation in operation theatre: Anaesthesia work station was checked. Appropriate size endotracheal tubes, working laryngoscope with medium and large-sized blades, stylet and working suction apparatus were kept ready before the induction of general anaesthesia. Emergency drug trays of atropine, adrenaline and mephentermine were also kept ready for any eventuality.

Upon arriving at the operation theatre, IV cannulations were done with an 18 G cannula and ringer lactate was connected. Patients were connected to ECG, non-invasive blood pressure monitors, pulse oximetry and entropy. The patients were premedicated with IV 50 mg of Inj. ranitidine and 0.2 mg of Inj. Glycopyrrolate. The 31 study drug syringes were prepared by an anaesthetist not involved in the observation. Patients in group D received 0.5 µg/kg of Inj. Dexmedetomidine in 10 ml saline, and group S received 10 ml of Saline for 10 minutes. Oxygen supplementation through a mask was given during this period. Ramsay Sedation Score was noted at baseline, 5th and 10th minute during infusion. Etomidate 0.3 mg/kg was administered over 30 seconds, and pain related to injection was evaluated; 0: no pain, 1: mild pain, 2: moderate pain, 3: severe pain. Also, myoclonus was observed for two minutes following etomidate induction and graded; 0: no myoclonus, 1: mild myoclonus, 2: moderate myoclonus, 3: severe myoclonus. Two minutes after the etomidate injection, Inj. midazolam 0.02 mg/kg, Inj. fentanyl 2 µg/kg and Inj. Vecuronium 0.1 mg/kg was administered. After three minutes, patients were intubated with an appropriately sized, cuffed oral endotracheal tube. Anaesthesia was maintained according to institutional protocol with N2O + O2 + isoflurane. Hypotension, defined as more than a 20% decrease in mean arterial pressure, was treated with fluid boluses and injection ephedrine 6 mg IV. Bradycardia, a heart rate less than 50 beats/min, was treated with an injection of atropine 0.6 mg IV. At the end of the surgery, residual paralysis was reversed with 0.05 mg of Inj. neostigmine and 0.01 mg of Inj. Glycopyrrolate. At the time of extubation, the recovery profile was noted (the time between cut off of the inhalational agent to the opening of eyes), and extubation time (the time between cut off of the inhalational agent to the removal of the endotracheal tube) was recorded. Ramsay sedation score after extubation was recorded.

Parameters observed such as Ramsay sedation score at the baseline, after administering study drug and after extubation, Myoclonus grading, Pain grading, time to extubation and eye-opening, and Postoperative RSS, RR, nausea and vomiting every 1/2 an hour for 2 hours.

ISSN (O): 2687-5365; ISSN (P): 2753-6556

Statistical Analysis

The collected data was entered in Microsoft Excel (windows 10), and analysis was done using the statistical package for social sciences (SPSS-15). To find an association between two categorical variables Pearson chi-square test was used. The value of P<0.05 is considered as statically significant.

RESULTS

In the present study, female predominance was reported in both groups. The mean age was 36.4 ± 8.7 years in Group D and 37.69 ± 8.13 years in Group S, which was comparable. The mean weight in both groups was comparable (Group D: 55 ± 8.63 kg; Group S: 55.37 ± 10 kg). Types of surgeries were uniformly distributed in both groups and were comparable. The mean duration of surgery was 67.57±16.91 minutes in group D and 70.57±16.57 minutes in group S, comparable with p = 0.471. Thirty-two patients in group D (91.42%) and 30 patients in group S (85.71%) experienced no pain on injection following etomidate induction. 2 patients in group D (5.71%) and five patients in group S (14.28%) had mild pain on injection. In contrast, one patient in group D (2.85%) had moderate pain on injection. Thus, there was statistically insignificant in comparing the incidence of pain on injection in both the studied groups (p = 0.309) (Table 1).

Twenty patients in group D (57.14%) and 16 patients in group S (45.71%) had no myoclonus following etomidate induction which was statistically insignificant (p = 0.237). Six patients in group D (17.14%) and four patients in group S (11.42%) experienced mild myoclonus (p = 0.367). Six patients in group D (17.14%) and five patients in group S (14.28%) had moderate myoclonus (p = 0.5) following etomidate induction which was statistically insignificant. Three patients in group D (8.57%) and ten patients (28.5%) in group S had severe myoclonus following etomidate induction which was statistically significant with p = 0.031 (Table 1, Fig 1).

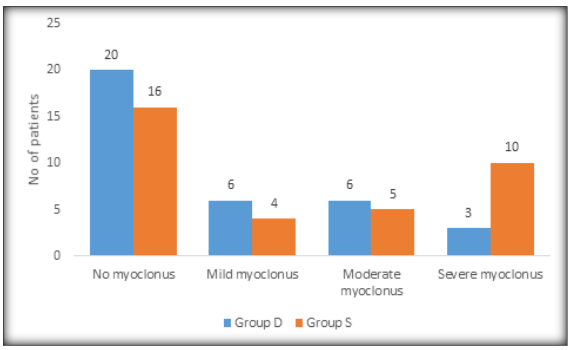

Figure 1: Observation of myoclonus in two groups of patients studied

The time to achieve entropy 50 was 64.23 ± 9.67 seconds in group D and 71.46 ± 15.76 seconds in group S which was statistically significant (p = 0.02). However, both groups found the loss of palpebral reflex and verbal commands comparable (Table 1).

The mean time to extubate from stopping the inhalational agent was 11.89 ± 2.54 minutes in Group D.

In Group S was 12.57 ± 2.91 minutes, which was statistically insignificant (p = 0.298). The eye-opening from stopping an inhalational agent was 10.60 ± 2.58 minutes in group D and 11.34 ± 2.80 minutes in group S was insignificant (p = 0.252) (Table 1).

Table 1: Observation of demographic and other evaluation variables of patients in both groups

Variables	Observations N (%)	
	Group D	Group S
Gender		
Female	29 (82.86)	28 (80)
Male	6 (17.14)	7 (20)
Age in Years		
<= 20	1 (2.8)	2 (5.7)
21 – 30	10 (28.6)	4 (11.4)
31 – 40	13 (37.1)	14 (40)
41 – 50	10 (28.6)	14 (40)
51 – 60	1 (2.9)	1 (2.9)
Mean ± SD	36.4 ± 8.7	37.69 ± 8.13
Weight (kg) (mean± SD)	55 ± 8.63	55.37±10.1
Surgeries		
Head and Neck surgeries	10 (28.6)	19 (54.3)
Upper abdominal surgeries	4 (11.4)	3 (8.6)
Breast surgeries	8 (22.9)	3 (8.6)
Laparoscopic surgeries	9 (25.7)	9 (25.7)
Others	4 (11.4)	1 (2.8)
Duration of surgery (min) (mean± SD)	67.57±16.91	70.57±16.57
Pain grading		
No pain	32 (91.42 %)	30 (85.71 %)
Mild pain	2 (5.71 %)	5 (14.28 %)
Moderate pain	1 (2.85 %)	0 (0 %)
Severe pain	0 (0 %)	0 (0 %)
Myoclonus grading		
No myoclonus	20 (57.14 %)	16 (45.71 %)

ISSN (O): 2687-5365; ISSN (P): 2753-6556

Mild myoclonus	6 (17.14 %)	4 (11.42 %)
Moderate myoclonus	6 (17.14 %)	5 (14.28 %)
Severe myoclonus	3 (8.57 %)	10 (28.5 %)
The total incidence of myoclonus	15 (42.86 %)	19 (54.29 %)
Time taken to reach entropy 50 (mean± SD) in seconds	64.23 ±9.67	71.46 ±15.76
Loss of palpebral reflex (mean± SD) in seconds	41.14 ±9.17	43.57 ±6.72
Loss of verbal commands (mean± SD) in seconds	41.14 ±9.17	43.51 ±6.68
Time to extubation **(mean± SD) in minutes**	11.89 2.54	12.57 2.91
Time to eye-opening **(mean± SD) in minutes**	10.60± 2.58	11.34 ±2.80

Ramsay sedation score at the 10th minute of the study drug infusion was significantly higher in group D than in group S (p = 0.001). A total of 10 patients in group D (28.57%) were drowsy; responsive to verbal commands (RSS-3), and one patient in group D (2.85%) was asleep and responsive to light stimulation (RSS-4). Post-extubation Ramsay sedation score was significantly higher in group D compared to group S at the 30th, 60th and 90th minute with p-value = 0.001, 0.001 and 0.051, respectively. However, no patients in group D required intervention for sedation and were easily arousable. There was statistical insignificance between the groups immediately after extubation and at the 120th minute postoperatively (Table 2).

Table 2: Observation of Ramsay sedation score during study drug infusion and after extubation

Ramsay sedation score (RSS)		**Observations N (%)**		**P value by χ2 test**
		Group D	**Group S**	
RSS before extubation				
Baseline	1	6 (17.14%)	2 (5.71%)	0.133
	2	29 (82.85%)	33 (94.28%)	
	3	0 (0%)	0 (0%)	
	4	0 (0%)	0 (0%)	
	5	0 (0%)	0 (0%)	
	6	0 (0%)	0 (0%)	
5 min	1	0 (0%)	0 (0%)	0.151
	2	33 (94.28%)	35 (100%)	
	3	2 (5.71%)	0 (0%)	
	4	0 (0%)	0 (0%)	
	5	0 (0%)	0 (0%)	
	6	0 (0%)	0 (0%)	
10 min	1	0 (0%)	0 (0%)	0.001
	2	24 (68.57%)	35 (100%)	
	3	10 (28.57%)	0 (0%)	
	4	1 (2.85%)	0 (0%)	
	5	0 (0%)	0 (0%)	
	6	0 (0%)	0 (0%)	
RSS post-extubation				
0 min	1	0 (0 %)	0 (0 %)	0.316
	2	0 (0 %)	1 (2.86 %)	
	3	19 (54.29 %)	23 (65.71 %)	
	4	16 (45.71 %)	11 (31.43 %)	
	5	0 (0 %)	0 (0 %)	
	6	0 (0 %)	0 (0 %)	
30 min	1	0 (0 %)	0 (0 %)	0.001
	2	1 (2.86 %)	13 (37.14 %)	
	3	31 (88.57 %)	21 (60 %)	
	4	3 (8.57 %)	1 (2.86 %)	
	5	0 (0 %)	0 (0 %)	
	6	0 (0 %)	0 (0 %)	
60 min	1	7 (20 %)	21 (60 %)	0.001
	2	28 (80 %)	14 (40 %)	
	3	0 (0 %)	0 (0 %)	
	4	0 (0 %)	0 (0 %)	
	5	0 (0 %)	0 (0 %)	
	6	0 (0 %)	0 (0 %)	
90 min	1	0 (0 %)	0 (0 %)	0.051
	2	17 (48.57 %)	25 (71.43 %)	
	3	18 (51.43 %)	10 (28.57 %)	
	4	0 (0 %)	0 (0 %)	
	5	0 (0 %)	0 (0 %)	
	6	0 (0 %)	0 (0 %)	
120 min	1	0 (0 %)	0 (0 %)	0.759

ISSN (O): 2687-5365; ISSN (P): 2753-6556

	2	29 (82.86 %)	28 (80 %)	
	3	6 (17.14 %)	7 (20 %)	
	4	0 (0 %)	0 (0 %)	
	5	0 (0 %)	0 (0 %)	
	6	0 (0 %)	0 (0 %)	

Table 3: Observation of nausea and vomiting among all patients at different time points

		Observations N (%)		P value by $\chi 2$ test
		Group D	Group S	
Nausea				
0 min	0	27 (77.14%)	27 (77.14%)	0.232
	1	5 (14.29%)	2 (5.71%)	
	2	0 (0%)	3 (8.57%)	
	3	3 (8.57%)	3 (8.57%)	
30 min	0	32 (91.43%)	28 (80%)	0.423
	1	2 (5.71%)	3 (8.57%)	
	2	0 (0%)	2 (5.71%)	
	3	1 (2.86%)	2 (5.71%)	
60 min	0	32 (91.43%)	30 (85.71%)	0.786
	1	1 (2.86%)	3 (8.57%)	
	2	1 (2.86%)	1 (2.86%)	
	3	1 (2.86%)	1 (2.86%)	
90 min	0	33 (94.29%)	32 (91.43%)	0.389
	1	1 (2.86%)	1 (2.86%)	
	2	0 (0%)	2 (5.71%)	
	3	1 (2.86%)	0 (0%)	
120 min	0	34 (97.14%)	35 (100%)	0.314
	3	1 (2.86%)	0 (0%)	
Vomiting				
0 min	0	32 (91.43 %)	32 (91.43 %)	0.721
	1	1 (2.86 %)	2 (5.71 %)	
	2	1 (2.86 %)	0 (0 %)	
	3	1 (2.86 %)	1 (2.86 %)	
30 min	0	34 (97.14 %)	33 (94.29 %)	0.555
	1	0 (0 %)	0 (0 %)	
	2	0 (0 %)	0 (0 %)	
	3	1 (2.86 %)	2 (5.71 %)	
60 min	0	33 (94.29 %)	34 (97.14 %)	0.602
	1	1 (2.86 %)	1 (2.86 %)	
	2	0 (0 %)	0 (0 %)	
	3	1 (2.86 %)	0 (0 %)	
90 min	0	34 (97.14 %)	34 (97.14 %)	0.368
	1	0 (0 %)	1 (2.86 %)	
	2	0 (0 %)	0 (0 %)	
	3	1 (2.86 %)	0 (0 %)	
120 min	0	34 (97.14 %)	35 (100 %)	0.314
	1	0 (0 %)	0 (0 %)	
	2	0 (0 %)	0 (0 %)	
	3	1 (2.86 %)	0 (0 %)	

DISCUSSION

Etomidate is widely used as an anaesthetic induction agent in clinical practice. Several desirable properties, such as rapid onset, brevity of action, lack of cardiovascular depression, and protection of intracranial pressure, make it an attractive agent for rapid sequence intubation. However, etomidate is also associated with two side effects, pain on injection and myoclonus. Etomidate is weak water soluble, formulated with propylene glycol, lipid emulsions, and polyethylene and phosphate buffers.[7] This formulation causes pain, inflammation, and sometimes phlebitis and thrombosis at the injection site. Pain on injection has been largely eliminated by using a lipid formulation of etomidate, but myoclonus remains a common problem during anaesthesia induction. Etomidate-induced myoclonus can have serious consequences, such as vitreous prolapse in a patient with an open eye injury, and ECG leads may become detached during myoclonic movements.[8]

Several studies have reported myoclonic activity in 50% to 80% of patients receiving etomidate.[8-9] Though various drugs have been tried to reduce the incidence of myoclonic movements after etomidate administration, the mechanism by which this effect is achieved remains unclear. Sievert D et al. reported that myoclonus after etomidate is caused by subcortical disinhibition.[10]

Many drugs have been reported to prevent myoclonus associated with etomidate. Schwarzkopf et al. observed decreased myoclonic incidence by 20% after intravenous administration of 0.015 mg/kg midazolam when given 90 minutes before etomidate administration.[11] However, intravenous midazolam injection could induce respiratory depression and

ISSN (O): 2687-5365; ISSN (P): 2753-6556

sedation. Stockam et al. revealed that 100 µg fentanyl did not decrease myoclonus incidence; higher doses decreased myoclonic activity but caused apnea.[12] In a study by Salman et al., pretreatment with dexmedetomidine 0.5 µg/kg and midazolam 0.25 mg/kg decreased myoclonus associated with etomidate use. In their research, severe myoclonus was not observed in both groups. Only mild myoclonus was more common in the dexmedetomidine group (16.7% vs 40%) ($p<0.05$).[13]

In the present study, Group D had a significantly lower incidence of severe myoclonus than Group S, with only three patients having severe myoclonus compared to.[10] Thus, dexmedetomidine significantly reduced myoclonus's severity, consistent with the other studies. However, unlike the other studies, there was no change in the incidence of myoclonus following etomidate induction.

In a study conducted by Salman et al., the pain on injection was significantly less in the midazolam group compared to the dexmedetomidine group following etomidate induction; no pain; $p< 0.001$, mild pain; $p = 0.005$, moderate pain; $p = 0.002$, severe pain; $p<0.001$.[13]

In contrast to the above studies, in our study, the incidence of pain on injection was less in both the study groups. Thirty-two patients in group D (91.42%) and 30 patients in group S (85.71%) did not have any pain on injection following etomidate induction ($p = 0.309$). 2 patients in group D (5.71%), and five patients in group S (14.28%) had mild pain on injection. In contrast, one patient in group D (2.85%) had moderate pain on injection. This may be because we had used a lipid formulation of etomidate.

In the present study, the mean loss of palpebral reflex following etomidate induction was 41.14 ± 9.17 seconds in group D, similar to the Gunes Y et al. observation. Unlike them, we did not find any delay in induction in group S (43.57 ± 6.72 seconds).14 However, it is possible that patients might be in lighter planes of anaesthesia or sedation, which could have been deep, that they did not respond to the request. This clinical assessment of the depth of anaesthesia is ambiguous and does not have high sensitivity or specificity.

Gunes Y et al. observed decreased BIS values after pretreatment with midazolam and dexmedetomidine.[14] In our study, we used entropy to measure the depth of anaesthesia. Entropy is a useful tool to quantify the anaesthetic drug effect and is comparable to established processed EEG parameters like BIS. There was a significant fall in RE ($p = 0.007$) and SE ($p = 0.006$) values from the baseline in the dexmedetomidine group compared to a saline group at the end of the dexmedetomidine infusion consistent with Gunes Y et al. observation.

The Ramsay sedation score was comparable at baseline and 5th minute but significantly higher at the end of dexmedetomidine infusion. Post-extubation sedation scores were comparable but significantly higher in group D. Recovery profile, time to extubation, respiratory rate after extubation, 30th, 60th, 90th and 120th minute postoperatively were comparable.

Mizrak et al. found no statistically significant difference between the group incidences of nausea and vomiting. In our study, the findings were consistent with the above study. The incidence of nausea and vomiting after extubation, 30th, 60th, 90th and 120th minute postoperatively was comparable between groups D and S ($p > 0.05$). None of the patients experienced hypotension, bradycardia, hypertension, arrhythmia or other side effect of the drug during the study.[15]

CONCLUSION

Our study shows that pretreatment with dexmedetomidine 0.5 µg/kg IV effectively reduces the severity of etomidate-induced myoclonic muscle movements without any haemodynamic side effects. However, dexmedetomidine does not significantly affect the incidence of myoclonus following etomidate induction.

REFERENCES

1. Bassett DC, Stokes KJ, Thomas WR. Wound infection with Pseudomonas multivorans: a water-borne contaminant of disinfectant solutions. Lancet. 1970; 1:1188-91.
2. Cruse PJ. Surgical wound infection. In: Wonsiewicz MJ, ed. Infectious Diseases. Philadelphia: W.B. Saunders Co., 1992, 758-64.
3. Suryawanshi P, Khan AQ, Saiyyad Altaf G, Patil A. Analysis of Organisms Found at Incision Site Intra-Operatively and its Implications with Postoperative infections. Int J Sci Res, 2014; 4: 1-15.
4. Krizek TJ, Robson MC. Evolution of quantitative bacteriology in wound management. Am J Surg 1975; 130:579-84.
5. Henderson B, Poole S, Wilson M. Microbial/host interactions in health and disease: who controls the cytokine network? Immunopharmacology 1996; 35:1-21.
6. Salman N, Gurbuz HA, Oguzalp H, Ucar HI, Yorgancioglu C, Sekerci S. Comparison of dexmedetomidine and midazolam in prevention of myoclonus occurring due to etomidate in coronary bypass surgery. J Cardiovasc Surg. 2013; 1:52-6.
7. Sema Aktolga, Yasemin Gunes, Murat Gunduz, Geylan Isik. A Comparison of Midazolam and Dexmedetomidine for the Prevention of Myoclonic Movements and Pain Following Etomidate Injection. J Anaesth Clin Pharmacol 2010; 26: 162-6
8. Luan HF, Zhao ZB, Feng JY, Cui JZ, Zhang XB, Zhu P, et al. Prevention of etomidate-induced myoclonus during anesthetic induction by pretreatment with dexmedetomidine. Braz J Med Biol Res 2015; 48:186–90.
9. Hueter L, Schwarzkopf K, Simon M, Bredle D, Fritz H. Pretreatment with sufentanil reduces myoclonus after etomidate: Pretreatment with sufentanil reduces myoclonus after etomidate. Acta Anaesthesiol Scand 2003;47:482–4.
10. Sievert DM, Ricks P, Edwards JR, Schneider A, Patel J, Srinivasan A, et al. Antimicrobial-resistant pathogens associated with healthcare-associated infections: summary of data reported to the National Healthcare Safety Network at the Centers for Disease Control and Prevention, 2009-2010. Infect Control Hosp Epidemiol. 2013; 34:1-14.
11. Schwarzkopf KR, Hueter L, Simon M, Fritz HG. Midazolam pretreatment reduces etomidate-induced myoclonic movements. Anaesth Intensive Care 2003; 31: 18-2.
12. Stockham RJ, Stanley TH, Pace NL, Gillmor S, Groen F, Hilkens P. Fentanyl pretreatment modifies anaesthetic

ISSN (O): 2687-5365; ISSN (P): 2753-6556

induction with etomidate. Anaesth Intensive Care. 1988; 16:171–6.
13. Salman N, Gurbuz HA, Oguzalp H, Ucar HI, Yorgancioglu C, Sekerci S. Comparison of dexmedetomidine and midazolam in prevention of myoclonus occurring due to etomidate in coronary bypass surgery. J Cardiovasc Surg 2013; 1: 52-6.
14. Gunes Y, Aktolga S, Gunduz M, Isik G. A comparison of midazolam and dexmedetomidine for the prevention of myoclonic movements and pain following etomidate injection. J Anaesthesiol Clin Pharmacol 2010;26:162.
15. Mizrak A, Koruk S, Bilgi M, Kocamer B, Erkutlu I, Ganidagli S, et al. Pretreatment with dexmedetomidine or thiopental decreases myoclonus after etomidate: a randomized, double-blind controlled trial. J Surg Res 2010; 159: e11-e16.

ISSN (O): 2687-5365; ISSN (P): 2753-6556