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KEYWORDS: Etomidate; Dexmedetomidine; Myoclonus; Midazolam

A COMPARISON BETWEEN DEXMEDETOMIDINE AND MIDAZOLAM FOR PREVENTION OF ETOMIDATE INDUCED MYOCLONUS IN PATIENTS UNDERGOING SURGERIES UNDER GENERAL ANAESTHESIA

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Abstract

Background: Myoclonus is a common problem during induction of anaesthesia with etomidate. A variety of drugs have been used to decrease the incidence of myoclonus. In this study we compared the effects on dexmedetomidine and midazolam pretreatment on the incidence and severity of EIM.

Materials and Methods: Fifty adult patients (18 to 60 years age) of either sex, ASA-PS-I and II undergoing elective general surgeries under general anesthesia were randomly allocated into two groups. Group D patients received Inj. Dexmedetomidine (0.5ug/kg) and Group M received Inj. Midazolam (0.02mg/kg) in 10 ml normal saline over ten minute. After 2 minutes patients were induced with etomidate. The patients were observed for occurrence and severity of myoclonus for 3 minutes from the start of Injection of the induction dose and graded according to clinical severity (Grade 0-III).

Results: In Group D,14 out of 25(56%) patients did not have any myoclonus during induction with etomidate, and none of the patients had grade III (severe) myoclonus. 8 patients of 25 (32%) patients observed grade I and 3 out of 25(12%) had grade II EIM in group D. In group M-9(36%) ,9(36%) ,6(24%),and patients had grade O,I,II and III EIM respectively.

Conclusion: Incidence and severity of EIM among patients who underwent pretreatment with dexmedetomidine was greatly reduced than those who underwent pretreatment with midazolam.

I. Introduction

Etomidate is a carboxylated imidazole derived potent sedative-hypnotic agent,directly acting on gamma-aminobutyric acid(GABA) receptor complex,blocking neuro excitation and producing anaesthesia. (2) It is preferred induction agent especially in hemodynamic compromised patient. Etomidate has two common adverse effects, pain on Injection and myoclonus(5) apart from PONV(Post operative nausea vomiting).

Myoclonus is defined as sudden, involuntary, short either irregular or rhythmic contraction of some muscle fibers of a whole muscle on of different muscles of one group leading to short observable movements of the corresponding body parts.(5) The occurrence of myoclonus in non-premedicated patients may be as high as 80% (2). A number of drugs have been investigated for suppression of etomidate-induced myoclonus.Altering the speed of Injection of etomidate and pre-treatment with drugs lidocaine,benzodiazepam(midazolam),magnesium,opioid (fentanyl,dezocine),dexmedetomidine have been investigated as ways of reducing etomidate induced myoclonus with variable results. (9)

Midazolam,being a benzodiazepine inhibits subcortical neuronal activity and showed some role in decreasing EIM when used as premeditation. Dexmedetomidine is a strong, highly selective alpha 2 adrenoreceptor agonist with a wide spectrum of pharmacological properties. It provides sedation, anxiolysis,hypnosis as well as analgesia, and sympatholytic properties. Studies have shown some role of Dexmedetomidine pretreatment decreasing EIM. This study was designed to compare the efficacy of Dexmedetomidine with midazolam in preventing EIM incidence and its intensity when used as pretreatment.

II. Material And Methods

After approval of the Ethics committee of Burdwan Medical College and Hospital(BMCH) and permission of the West Bengal University of Health Sciences, Kolkata, the present thesis work was carried out under the Departmentof Anaesthesiology, BMCH,Burdwan from January 2020 to September 2020.

Study Design:Institution based prospective observational study.

Study Location: This was a hospital based study done in Burdwan Medical College and Hospital.

Study Duration: January 2020 to September 2020 (9 months).
Sample size: 50 patients.

Sample size calculation: The sample size have been calculated using the formula $n > 2(Z\alpha + Z1\text{-}\beta)2 \times p^*q/d2$, where $p = (p1 + p2)/2$, $q = 1\text{-}p$, and d is p1-p2 Now assuming p value <0.05 to be significant and considering effect to be two sided, we get $Z\alpha = 1.96$; assuming power of study to be 90% we get $Z1\text{-}\beta = 1.28$. taking p1 and p2 as the percentage of Grade 0 myoclonus, in the 2 groups as 55% and 12.5% respectively using the above formula we get n = 22 in each group. Hence minimum 22 patients will be taken in each group.We took 25 patients in each group keeping a margin of safetyfor any adverse effects.

Subjects & selection method: Subjects were allocated into two groups of equal number of patient(n=25 per group) randomly. No case control cohort is required for the study.

Inclusion criteria:

1. Signed the informed consent
2. ASA-PS I or II undergoing elective surgeries
3. Aged between 18 and 55 years
4. Both sexes
5. BMI: 20-30 kg/meter square
6. Undergoing elective surgery under General Anesthesia.

Exclusion criteria:

1. Geriatric and Pediatric population
2. Pregnant patients
3. Patient's unwillingness to be included in the study.
4. Patients with pre existing adrenal disease or adrenocortical insufficiency.

5. Patients who were receiving or having a history of receiving steroids within the last three months.
6. History of Allergy or hypersensitivity to the study drugs.
7. Patients on sedative, antidepressant medications or having any known psychological disease.
8. History of seizures, convulsion or any known neuromuscular diseases.
9. Serious vision, hearing impairment or other reasons where patient cannot communicate.
10. Sepsis or systemic infections.
11. Need for blood transfusion during surgery.
12. Hepatic Failure
13. Patients with pace-maker,uncontrolled HTN,bradycardia, heart blocks,heart failure.
14. Patients with Asthma, chronic cough, upper respiratory infection during the 2 weeks before surgery.
15. Patients who had received analgesics or sedatives within the 24 hrs.
16. Patients with anticipated difficult airway.

Procedure methodology

After obtaining institutional approval and informed written consent 50 patients who fulfilled the above inclusion criteria were taken up for the comparative study. All patients underwent pre-anesthetic check-up by proper history taking, clinical examination and all routine investigations before performing the procedure as per institutional protocol. Patient's height,weight noted and BMI calculated. All patients kept overnight fasting and premeditated with oral Diazepam (5)and Ranitidine (150) at bedtime on the previous day. Patients were randomly allocated into two groups (D and M) each consisting of 25. Their identification data were collected and written on case record form. Anesthesia Machine, circuit, monitor checked,all necessary medications including emergency drugs made available. Patients were received in O.T, their identification and consent for surgery, anesthesia as well as for the study Gross checked.Intravenous access was secured with 18 Gauze I.V. Cannula and infusion RL started at the rate 4-6ml/kg/hr.Monitors for NIBP,SpO2 ECG were attached to the patients and all baseline vitals were noted. Preoxygenation with 100% oxygen for 5 minutes and premeditation with Inj.Glycopyrrolate 0.4mg/kg I.v was given. Study drugs in each group were given by anesthesiologists who were not involved in this study.

Not Significant

One group of patients (D) received Dexmedetomidine 0.5ug/kgin 10 ml normal saline as infusion over 10 minutes.

Another group of patients (M) received Midazolam 0.02mg/kg in 10 ml Normal Saline as infusion over 10 minutes. After 2 minutes both groups of patients were induced with 0.3mg/kg Etomidate Injection I.v over a period of 30 secs or till the LOC or abolition of eyelash reflex was observed. From giving Dex or Midazolam premedication to Etomidate Injection patients were observed for adverse effects. Following Etomidate Injection mask ventilation with 100% O2 was carried out until intubation. From the starting of Injection Etomidate upto3 minutes all patients were closely observed for the presence of myoclonus and if present, the severity was graded by two experienced anesthesiologists. In case difficulty in mask ventilation due to myoclonus we planned to administer a neuromuscular blocking agent immediately and go for intubation but we didn't faced such episode. After 3 minutes of etomidate Injection and completion of myoclonus evaluation all patients received 2ug/kg I.v. fentanyl Injection and 0.5mg/kg I.v. Atracurium injection to facilitate orotracheal intubation. Anesthesia was maintained using 1.5-2.5% concentration of sevoflurance in a mixture of N2O and O2 (70% and 30%) and patients were put on mechanical ventilation. Residual neuromuscular blockade was antagonized with neostigmine and glycopyrrolate after completion of surgery.Heart rate, SBP,DBP,MAP and peripheral oxygen saturation in capillaries were monitored continuously till the exit of patient from O.T and recorded in frequent intervals up to 30 mins.All patients were monitored thereafter in PACU. If the MAP was below 70mmHg, 5-10 mg I.V.Inj.ephedrine was administered. If the heart rate was <50 beats/min inj.i.v. Atorpin 0.5mg was administered at once.

Patients data were collected and noted down on case record form and was tabulated on Microsoft Excel Sheet and interpreted by the Statistician.

Statistical analysis

Categorical variables are expressed as Number of patients and percentage of patients and compared across the groups using Pearson's Chi Square test for Independence of Attributes/ Fisher's Exact Test as appropriate. Continuous variables are expressed as Mean, Median and Standard Deviation and compared across the groups using unpaired t test. The statistical software SPSS version 20 has been used for the analysis.

An alpha level of 5% has been taken, i.e. if any p value is less than 0.05 it has been considered as significant.

III. Result

Mean age and BMI between the group D and group M are compared. There was no statistical significant difference for both mean age and BMI between the two groups. Thus both groups are comparable from age and BMI

Distribution for Age, Sex and ASA Physical status I and II across the patients of groups D and M are also compared. There were no statistical significant differences for the age, sex and ASA Physical status between the two groups. So they were comparable.

Mean and standard deviation of HRat 0,1,2,3, 5,10,15, 20,25,30 minutes and Mean and standard deviation of SBP, DBP and MAP at 0,1,3,5, 10,15,20,25,30 minutes of patients of both groups are compared. There are no statistically significant differences for mean HR, SBP, DBP, and MAP between the groups. So the patients of both thegroups were comparable from baseline parameters. Also there was no significant variation in HR along the course of anesthesia due to addition of thedrugs in both groups.Also there was no significant variation in BP due to either drug.

SpO2'/, of patients of both the group D and M at different time intervals are compared. P Value at baseline was 0.122 which is not significant. So both groups SpO2 was matched at initiation. Though there were fall in SpO2 in group M patients at 2,3,4 and 5 minutes intervals with statistical significance, but as the values were within normal range so it was not significant clinically.

The frequency of occurrence and gradation of myoclonus across the patients of groups D and M are measured. In group D, 15 out of 25 patients (60'/,) did not developed EIM at all whereas 8 patients out of 25 (32'/,)patients of group M did not developed EIM. This difference of non occurrence was statistically significant (P value 0.038). Mild grade of EIM happened in 28 and 40 percent patients of group D and M respectively. Similarly moderate grade of EIM occurred in 12 and 24 percent patients of groups D and M respectively. Though mild and moderate grade myoclonus in both groups were statistically not significant, clinically they look significant. In group D, no patient experienced severe EIM whereas 1 among 25 in group M had myoclonus.

The occurrence of adverse effects in the patients of groups D and M is compared. 2 out of 25 patients i.e. 8% patients in group D experienced bradycardia. Both hypotension and hypoventilation were happened in 1 of 25 patients (4%) each in group M patients. No adverse effects came statistically significant.

Table no 1 :Comparisons of mean age between group D and group M

	GROUP			
	DEXMEDETOMIDINE	MIDAZOLAM		

	Mean	Std. Deviation	Mean	Std. Deviation	P valuep Value	Significance level
Age (Yrs)	34.64	9.79	34.68	9.37	0.988	Not Significant
BMI (Kg/M^2)	25.68	2.64	25.16	2.62	0.489	Not Significant

TABLE No 2:

Duration in Minute	GROUP					
	DEXMEDETOMIDINE		MIDAZOLAM			
	Mean	Std. Deviation	Mean	Std. Deviation	p Value	Significance
HR-0	81.00	7.50	79.64	9.42	0.575	Not Significant
HR-1	80.44	7.23	79.80	9.06	0.784	Not Significant
HR-2	81.88	8.19	82.12	9.40	0.924	Not Significant
HR-3	79.88	8.81	84.60	10.10	0.085	Not Significant
HR-5	80.36	10.37	85.52	10.21	0.083	Not Significant
HR-10	80.04	11.40	85.48	10.07	0.080	Not Significant
HR-15	78.48	10.88	83.04	9.82	0.126	Not Significant
HR-20	77.36	9.97	82.04	8.77	0.084	Not Significant
HR-25	78.12	10.22	81.08	8.89	0.280	Not Significant
HR-30	78.08	9.28	81.04	8.83	0.254	Not Significant

Fig 1: LINE DIAGRAM describing the above table for HR among 2groups.

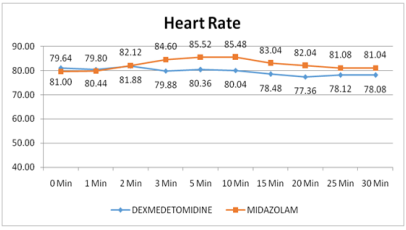
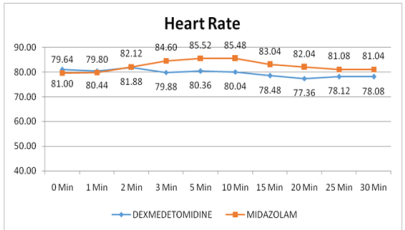


TABLE 2 showing SBP at 0 ,1,3,5 10,15,20,25,30 minutes (mean and standard deviation) among the 2 groups.

	GROUP					
	DEXMEDETOMIDINE		MIDAZOLAM			
	Mean	Std. Deviation	Mean	Std. Deviation	p Value	Significance
SBP-0M	124.52	12.10	128.60	12.27	0.242	Not Significant
SBP-1M	124.08	11.34	126.56	9.91	0.414	Not Significant
SBP-3M	121.48	9.05	122.96	9.59	0.577	Not Significant
SBP-5M	121.20	10.19	123.84	9.95	0.359	Not Significant
SBP-10M	122.00	10.15	122.56	12.07	0.860	Not Significant
SBP-15M	120.88	9.68	122.72	11.64	0.546	Not Significant
SBP-20M	122.16	10.41	122.16	11.41	1.000	Not Significant
SBP-25M	122.64	10.22	122.36	11.38	0.927	Not Significant
SBP-30M	122.32	10.49	122.72	10.37	0.893	Not Significant



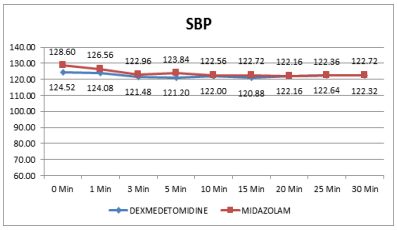


TABLE 3 for comparison of mean and standard deviation of DBP at 0,1,3,5,10,15,20,25 and 30 minutes.

	GROUP					
	DEXMEDETOMIDINE		MIDAZOLAM			
	Mean	Std. Deviation	Mean	Std. Deviation	p Value	Significance
DBP-0 M	76.80	8.15	78.88	10.08	0.426	Not Significant
DBP-1M	75.76	9.46	77.40	10.01	0.554	Not Significant
DBP-3M	72.04	6.24	76.36	11.12	0.097	Not Significant
DBP-5M	72.56	8.35	76.16	11.38	0.208	Not Significant
DBP-10M	74.40	8.93	74.92	10.17	0.849	Not Significant
DBP-15M	73.76	7.29	74.84	9.07	0.645	Not Significant
DBP-20M	74.48	6.10	74.48	7.85	1.000	Not Significant
DBP-25M	75.28	6.45	75.08	8.01	0.923	Not Significant
DBP-30M	76.60	6.30	76.40	8.25	0.924	Not Significant

LINE DIAGRAM for DBP of 2 groups describing the table 8:

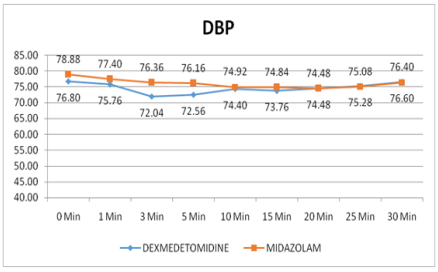


TABLE 9 : Depicting mean and standard deviation of MAP among the 2 groups at 0M,1M,3M,5M ,10M,15M,20M,25M and 30M intervals.

	GROUP					
	DEXMEDETOMIDINE		MIDAZOLAM			
	Mean	Std. Deviation	Mean	Std. Deviation	p Value	Significance
MAP-0M	91.04	9.18	92.72	9.12	0.519	Not Significant
MAP-1M	89.76	9.47	90.04	9.47	0.917	Not Significant
MAP-3M	85.64	6.26	88.68	10.49	0.220	Not Significant
MAP-5M	86.52	8.83	88.16	11.30	0.570	Not Significant
MAP-10M	88.08	8.48	87.68	10.21	0.881	Not Significant

MAP-15M	87.00	6.91	87.40	8.70	0.858	Not Significant
MAP-20M	89.04	6.84	87.76	8.54	0.561	Not Significant
MAP-25M	88.64	5.23	88.24	7.66	0.830	Not Significant
MAP-30M	89.68	5.68	88.08	7.47	0.398	Not Significant

LINE DIAGRAM for MAP describing the above table – 9.

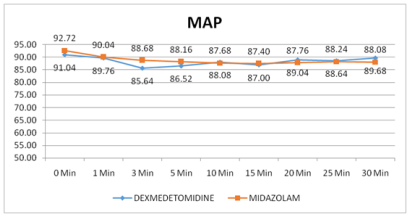


TABLE 10 : Showing SpO2% of patients of group D and M at different time intervals.

	GROUP					
	DEXMEDETOMIDINE		MIDAZOLAM			
	Mean	Std. Deviation	Mean	Std. Deviation	p Value	Significance
SPO2-0 M	99.12	0.83	99.44	0.58	0.122	Not Significant
SPO2-1M	99.20	0.96	98.80	1.15	0.189	Not Significant
SPO2-2M	99.20	1.04	98.36	1.78	0.047	Significant
SPO2-3M	99.16	1.03	98.32	1.91	0.049	Significant
SPO2-4M	99.20	0.87	98.40	1.68	0.040	Significant
SPO2-5M	99.60	0.71	98.64	1.55	0.007	Significant
SPO2-7M	99.80	0.50	99.36	1.19	0.094	Not Significant
SPO2-10M	99.72	0.68	99.68	0.85	0.855	Not Significant
SPO2-15M	99.88	0.44	99.76	0.72	0.482	Not Significant
SPO2-30M	99.80	0.50	99.76	0.60	0.798	Not Significant

LINE DIAGRAM describing the above table for SpO2 percentage among 2 groups.

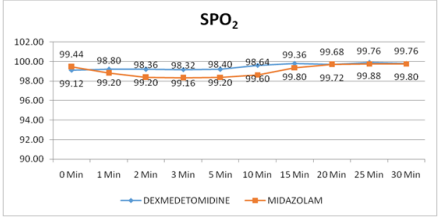


TABLE 11: Depicting frequency of occurrence and gradation of myoclonus across the patients of groups D and M.

	GROUP			Total		
	DEXMEDETOMIDINE	MIDAZOLAM			p Value	Significance
MYOCLONUS	ABSENT	15(60)	8(32)	23(46)	0.038	Significant
	MILD	7(28)	10(40)	17(34)	0.367	Not Significant
	MODERATE	3(12)	6(24)	9(18)	0.264	Not Significant
	SEVERE	0(0)	1(4)	1(2)	0.307	Not Significant
Total	25(100)	25(100)	50(100)			

Fisher's Exact Test

BAR DIAGRAMdescribing table11. Distribution of different grades of myoclonus across the patients of 2 groups are shown.

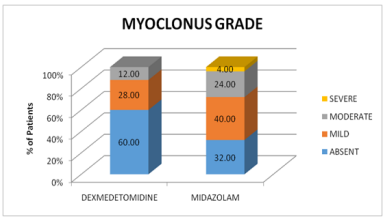
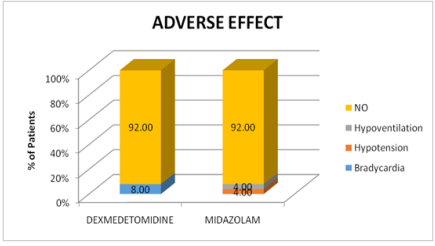


TABLE 12: Showing occurrence of adverse effects in the patients of groups D and M.

		GROUP		Total	p Value	Significance
		DEXMEDETOMIDINE	MIDAZOLAM			
ADV-EFFECT	Bradycardia	2(8)	0(0)	2(4)	0.140	Not Significant
	Hypotension	0(0)	1(4)	1(2)	0.307	Not Significant
	Hypoventilation	0(0)	1(4)	1(2)	0.307	Not Significant
	NO	23(92)	23(92)	46(92)	1.000	Not Significant
Total		25(25)	25(25)	50(50)		

Fisher's Exact Test

BAR DIAGRAM for the table 12 stating distribution of adverse effects among the patients of groups D and M.



IV. Discussion

This study compared the effects of dexmedetomidine and midazolam pretreatment on the incidence and severity of etomidate induced myoclonus (EIM). The study demonstrated that dexmedetomidine significantly decreased the occurrence of EIM in comparison to Midaz. Though both Dex and Midaz decrease the severity of myoclonus, Dex is more powerful than midaz. Etomidate, an imidazole derivatives short acting intravenous induction agent used in the clinical practice. Several desirable properties, such as rapid onset (81),brevity of action (82),lack of cardiovascular depression (83),minimal histamine release and protection of intracranial pressure could make it a good i.v Induction agent if the undesirable side effects like pain on injection ,myoclonus, increased

PONV can be attenuated. Also etomidate may cause temporary inhibition of steroid synthesis after single dose and infusion (84,85).Pain on Injection has been largely eliminated by the use of lipid formulation. Since EIM can have serious consequences discussed earlier, many physical methods and drugs have been reported for trying to prevent EIM.

In 2003,Schwarzkopf and colleagues compare the effect of pretreatment with midazolam versus placebo on the incidence and severity of myoclonus following etomidate induction. They concluded that pretreatment of midazolam is effective in reducing the incidence of myoclonic movements while preserving the advantage of etomidate, that is, cardiovascular stability and short duration of action. (87).

Huter et al. Investigated the effects of low dose intravenous midazolam pretreatment 0.015mg/kg on the incidence and severity of myoclonus during induction of anaesthesia with etomidate for elective cardioversion in non pre-medicated patients and it was found that 10% of patients in midazolam group had myoclonus movements as compared to 50% receiving placebo. (88).

Remifentanyl was reported to reduce the myoclonus but use was limited by its side effects like chest rigidity and bradycardia (90,91).

Alka Kewalramani et al.did a prospective, randomized study to compare butorphanol and fentanyl pretreatment with midazolam to reduce EIM and concluded that both can reduce the incidence as well as severity of EIM.(6)

Etomidate Injection technique also influences the incidence of EIM.Mullick et al.did a study designed to clarify which of the two Injection techniques -slow Injection (received etomidate-2mg/ml induction dose over 2 min) or priming (received pretreatment with 0.03mg/kg etomidate, followed after 1 min by an etomidate induction dose over 20 sec.) with etomidate-is more effective in reducing myoclonus. They concluded thatpriming is more effective than slow injection in reducing the incidence of myoclonus, but their effects on the severity of myoclonus are comparable. (9). So the search for a good preventive measure for EIM continues. In our study we took two drugs namely dexmedetomidine and midazolam as pretreatment in two groups and compared between two for controlling incidence and severity of EIM. Dexmedetomidine is a highly selective alpha-2 adrenoreceptor agonist. Its mechanism of action is at the locus ceruleus which shows one of the highest densities of alpha-2A adrenoreceptor in the brain. (94)

Therefore, the effect of dexmedetomidine in relieving myoclonus may be related to its sedative and analgesic effects.(79) Midazolam is a water-soluble benzodiazepine. Its pharmacological characteristics includes fast-acting, Short half life,rapid and complete absorption by muscle tissue and mucous membranes, and bioavailability >90% (97).

The inhibitory effects of midazolam on the central nervous system act through GABA- A receptor (98).

Small doses of midazolam have been found to readily suppress the effect of stimulation of the medullary reticular formation on electrochemical responses, leading to relaxation of central muscle, thus inhibiting or mitigating myoclonus. (87). In our study two group of the study D-Dexmedetomidine and M-Midazolam were taken. The demographic profile, weight etc were comparable across the group. Group D patients received 0.5ug/kg of Dexmedetomidine infusions in 10 ml NS over 10 mins and group M patients received 0.02mg/kg of Midazolam i.v. After 2 minutes induction done with 0.3mg/kg Etomidate over30 secs and after giving etomidate patients were observed for EIM upto 3 minutes till muscle relaxant was injected.

Myoclonus:

In our study 15 patients among 25 of group D i.e 60% didn't experienced myoclonus whereas 8 patients of 25 patients in group M i.e only 32% patients didn't have EIM.This difference was significant,p value being 0.038.So patients of group D experienced EIM in much less frequency (40%) than group M(68%).(80)

Grade-I i.e mild EIM was observed in 7 persons of D group and 10 persons of M group, which is comparable and low. Moderate, i.e grade II EIM was experienced in 3(12%) patients in group D and 6(24%) patients in group M. No patient in group D had severe myoclonus, Whereas only 1 patient (4%) in group M had severe (grade III) myoclonus. Gunes etal.compared midazolam and dexmedetomidine for the prevention of EIM movements and concluded pretreatment with both midazolam and dexmedetomidine reduced EIM.(74) Swarnendu Dey et.al compared dexmedetomidine and midazolam for prevention of EIM and concluded that incidence of EIM among patients who underwent pretreatment with Dex was significantly lesser than those who underwent pretreatment with midazolam. (2). This supports the result of our study. Luan et al. in their study concluded that pretreatment with 0.5 and 1ug/Kg dexmedetomidine significantly reduced the incidence of EIM during anaesthesia induction, however, 0.5ug/kg is the recommended dose because it has lesser side effects (1). Therefore, the results of our study are in Concordance with the other studies which finds dexmedetomidine efficacious in suppressing EIM. In our study, however, midazolam didn't suppress EIM as effectively as dexmedetomidine,which may be due to taking low dose of midazolam.

Hemodynamic and respiratory parameters: In our study 2 patients from group D developed bradycardia (HR<55min) and one patient of group M developed hypotension (Map<65 or >20% fall from baseline). But with respect to hemodynamic parameters there were no statistical significance between the two groups. One patient of group M developed low Spo2 (90%),but that was not significant. Though there was statistically significant differences seen in Spo2 during 2 minutes to 5 minutes interval in between two groups, but it is not so important as fall in Spo2 in M-midazolam group was within normal limit(in the range of 98%) Adverse reaction: In our study no serious adverse reaction noted that needed prompt and vigorous interruption. 2 patients of D group experienced mild bradycardia (HR @50/min and 51/min.)However, they didn't need any treatment (eg-Atropin injection). One patient from group M developed hypotension (MAP<65 Hg) who was managed with single dose of i.v. ephedrine. Another 1 patient's spo2 of group M decreased to 90%, which may due to sedation caused by midazolam. However, no specific intervention was needed, recovery was uneventful. No patient in either group experienced nausea-vomiting, sedation or dizziness. The low doses used in both groups may be the reason for less adverse effects.

V. Conclusion

From our study we conclude, Dexmedetomidine pretreatment is better in prevention of etomidate induced myoclonus than Midazolam pretreatment. Although both dexmedetomidine and midazolam pretreatment reduces severity of etomidate induced myoclonus, dexmedetomidine is much better in that respect also.
None of the drugs caused any severe adverse effect (only some level of bradycardia, hypotension and hypoxia in few cases) which needed specific management. These lesser side effects may be due to lower doses taken in our study. However search for ideal drugs and their dosing will continue.

VI. Limitations

Small study group, exclusion of ASA-PS-III, IV, may have hindered better evaluation of our objective.Also we did not include pediatric, geriatric and pregnant patients in our study. I think if we could observe for EIM for longer duration, during surgeries and anaesthesia and if we could record EEG, EMG during observation for EIM, the result would be more accurate. Also testing on different doses of dexmedetomidine and midazolam can detect more effective pretreatment for EIM.

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