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Original Research Article

COMPARISON BETWEEN DEXMEDETOMIDINE AND ESMOLOL AS EFFECTIVE PRETREATMENT REGIMENS ON ATTENUATION OF HAEMODYNAMIC PARAMETRES DURING ELECTROCONVULSIVE THERAPY

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ABSTRACT

Background: Modified Electroconvulsive Therapy (ECT) under anesthesia is an important modality in the treatment of severe persistent depression, bipolar disorder and schizophrenia; specially in case resistant to pharmacological therapy; but this procedure has got many side effects, specifically the hemodynamic alteration is markedly observed. **Aim:** The aim of present study to compare between Dexmedetomidine and esmolol as effective pretreatment regimens on attenuation of hemodynamic parameters during electroconvulsive therapy.
Materials and Methods: After getting approval of the study protocol by the institutional ethics committee (H) of Assam Medical College and Hospital, Dibrugarh and getting written informed consent from the patients and their relatives, fifty cases aged between 20-40 years belonging to ASA grade I and II scheduled for ECT are included in the study and grouped in 2 groups of 25 patients each. Group E received IV esmolol 1mg/kg body weight diluted up to 10ml of normal saline and infused within 2 minutes as premedication. Group D received IV Dexmedetomidine 0.5 mcg/kg diluted to 10ml of normal saline and infused within 10 minutes as premedication. Hemodynamic parameters heart rate (HR), systolic blood pressure, diastolic blood pressure and mean blood pressure at baseline (before induction), after study drug infusion and after ECT application were recorded at different time intervals and presented along with demographic data. Statistical analysis was done with SPSS software using student t-test and Chi square test.
Results: In this study, the baseline systolic blood pressure, diastolic blood pressure and mean blood pressure was statistically insignificant. The heart rate(HR), systolic blood pressure(SBP) and diastolic blood pressure(DBP) was significantly decreased in both esmolol and Dexmedetomidine group after drug administration and at 1,3,5 and 10 min post ECT shock. However, the decrease in MAP was statistically insignificant in both the groups after premedication, at 1,3,5,10 minutes of delivery of ECT shock. The mean time of return of spontaneous respiration was statistically insignificant.
Conclusion: Based on the present hospital based observational study, we came to the conclusion that an intravenous premedication agent in ECT, both esmolol at a dose of 0.1 mg/kg and Dexmedetomidine at a dose of 0.5 mcg/kg results in attenuation of cardiovascular (HR, BP, MAP) effects but more reduction of hemodynamic parameters were seen in the Dexmedetomidine compared to esmolol. The mean blood pressure however was unaffected.
Keywords: Dexmedetomidine, esmolol, modified Electroconvulsive therapy, hemodynamic parameters.

INTRODUCTION

Electroconvulsive Therapy (ECT) is an established therapy for severe depression in patients who do not respond to pharmacotherapy. Nowadays, almost all the ECT procedures are performed under general anesthesia; also known as modified ECT. The cardiovascular response generated by ECT is a brief parasympathetic stimulation followed by sympathetic stimulation during the seizure which markedly increases plasma levels of catecholamines thereby increased heart rate (HR) and Mean arterial pressure. These hemodynamic effects can produce cardiovascular stress and could place a patient with coronary and cerebrovascular disease at the risk of acute coronary or cerebrovascular event. Esmolol is an ultra-short acting beta 1 blocker and because of its negative ion tropic and chronotropic action on cardiovascular system, it is effective in playing a cardio protective role in short procedures like ECT. On the other hand, Dexmedetomidine is an alpha 2 adrenoreceptor agonist which is involved in regulating the autonomic nervous system and cardiovascular system by inhibiting the release of epinephrine and nor epinephrine by acting on blood vessels and sympathetic presynaptic terminals. Baroreceptor reflex is well preserved in patients receiving Dexmedetomidine and reflex heart rate response to a stimulus is augmented. The primary objective in this study was to compare the effects of inj esmolol 1mg/kg and inj Dexmedetomidine 0.5 mcg/kg as pretreatment regimens in attenuation of hemodynamic changes during ECT. Secondary objective is to evaluate postoperative recovery and complications if any.

MATERIAL AND METHODS

After getting clearance from Institutional Ethical Committee (H) of Assam Medical College and getting written informed consent, 50 cases belonging to ASA I and II of age 20-40 years of both sexes diagnosed with major depressive disorder, schizophrenia, catatonia or bipolar disorder were included in the study. Major exclusion criteria were age other than 20-40 years, known allergy to study drugs, pregnant and lactating females, major cardiac illness, renal and hepatic impairment, cerebrovascular accident, acute respiratory disorder. The study population were grouped into 2 groups, Group E and Group D. Each having 25 patients. Group E patients received IV Esmolol at a dose of 1mg/kg of body weight diluted up to 10ml of normal saline and infused within 2 minutes as premedication. Group D patients received IV Dexmedetomidine 0.5mcg/kg of body weight diluted up to 10ml of normal saline and infused within 10 minutes of premedication. Patients were investigated Haemoglobin level, complete blood count, blood urea nitrogen, serum creatinine, random blood sugar, liver function test, X-ray chest PA view, electrocardiography and recorded before the procedure. All patients were allowed for fasting 6 hours before undergoing ECT and were allowed to continue anti-psychotic medicine till day of procedure. Any dentures were removed and loose teeth secured with knot tying. On arrival to the ECT room standard monitors like Spo2 probe, ECG lead, Blood Pressure cuff were fitted and baseline data collected. An intravenous line was secured with 18 G IV canula and 500ml Crystalloid Ringer Lactate was started. The patients were premedicated with Glycopyrolate 4mc/kg IV and ondanseteron 4mg IV prior to the procedure. The patients were Preoxygenated with 100 % oxygen with a tightly fitting mask for 3 minutes before induction with mapelson A circuit. During preoxygenation group E received IV esmolol 1mg/kg IV diluted to 10ml with normal saline and infused within 2 minutes. Group D received IV Dexmedetomidine 0.5 mcg/kg diluted upto 10ml with normal saline and infused within 10 minutes. Patients were induced with Propofol 1mg/kg till loss of verbal response Following the onset of anaesthetic effect depolarizing muscle relaxant succinylcholine 0.5mg/kg were administered for neuromuscular relaxation. After fasciculation subsides patients were ventilated with 100% oxygen with tight fitted face mask. Oropharyngeal airway of adequate size was introduced. Bitemporral electrodes were placed on bifrontotemporal region and brief stimulus for about 2 milliseconds was given to produce seizure. Hemodynamic variables (HR, SBP, DBP and MAP) were recorded before induction (Baseline), after study drug administration, during premedication and 1 minute, 3 minutes, 5 minutes and 10 minutes after seizure. Subsequently, patients were ventilated with 100 % oxygen 14-16 breaths per minute until spontaneous respiration returned and patient recovered fully. The patients data were analyzed using the computer program, Statistical Package for Social Sciences (SPSS for windows version 20.0.0. Chicago, SPSS Inc) and Microsoft Excel 2010.Results on continuous measurement were presented as Mean +/- standard deviation, compared using student t-test. Discrete data were expressed as number (%) and were analyzed using Chi square test and Fischer's Exact test (where the cell count were <5 or 0). Pearson's correlation coefficient (r) was used to measure the associations among continuous variables. For all analysis, the statistical significance was fixed at 5% level (p value <0.05) t.

RESULTS

The demographic data like mean age, weight, gender were comparable in between the groups and shown in table 1. [Table 1]

The baseline hear t rate was comparable between the groups and shown in table2. However the heart rate was significantly decreased in both esmolol and Dexmedetomidine group after drug administration and at 1,3,5 and 10 min post ECT shock. It was

more statistically significant in Dexmedetomidine group. [Table 2]

The base line systolic blood pressure was comparable in both the groups (p< 0.05). It was statistically significantly decreased in both esmolol as well as Dexmedetomidine group during premedication, 1,3,5and 10 min of ECT shock; but more significant statistically in Dexmedetomidine group. [Table 3]

The baseline diastolic pressure was statistically insignificant in both the groups. It was decreased significantly in both esmolol and Dexmedetomidine during preoxygenation, in 1,3,5 and 10 minutes after ECT shock. The fall of diastolic blood pressure was more significant statistically in Dexmedetomidine group. [Table 4]

The Mean Arterial Pressure distribution is shown in Table 5. The baseline MAP was comparable in both the groups. The decrease in MAP was statistically insignificant in both the groups after premedication, at 1,3,5,10 minutes of delivery of ECT shock. [Table 5]

The mean time of return of spontaneous respiration was statistically insignificant and shown in table 6. [Table 6]

Table 1: Demographic data

	Group E	Group D	P value
Age	29.68 +/- 5.53	30.50 +/- 5.57	0.6038
Sex ratio (M:F)	1.08:1	0.092:1	0.773
Weight	50.90 +/- 5.57	50.08 +/- 5.54	0.6041

Table 2: Intergroup comparison of Heart Rate

HEART RATE (Beats/min)	Group E		Group D		P value
	Mean	S.D	Mean	S.D	
Baseline	82.20	1.83	81.92	1.89	0.5968
Preoxygenation	73.56	2.08	72.28	1.88	0.0271
1 min after ECT	74.80	2.87	73.48	1.33	0.0423
3 min after ECT	77.16	1.93	75.72	1.62	0.0063
5 min after ECT	78.28	2.21	76.96	1.77	0.0239
10 min after ECT	81.88	1.54	79.12	0.78	<0.001

Table 3: Intergroup comparison of systolic Blood pressure

SYSTOLIC BLOOD PRESSURE (mmHg)	Group E		Group D		P value
	Mean	S.D	Mean	S.D	
Baseline	122.96	2.78	122.36	3.00	0.4664
Preoxygenation	114.08	3.03	113.32	2.64	0.3489
1 min after ECT	119.20	2.43	115.28	1.90	<0.001
3 min after ECT	119.00	3.15	115.64	4.95	0.0062
5 min after ECT	119.20	1.87	116.72	3.17	0.0015
10 min after ECT	121.36	2.41	117.88	2.51	<0.001

Table 4: Intergroup comparison of Diastolic Blood Pressure

DIASTOLIC BLOOD PRESSURE (mmHg)	Group E		Group D		P value
	Mean	S.D	Mean	S.D	
Baseline	80.84	4.29	80.44	1.16	0.6545
Preoxygenation	74.76	3.99	73.72	1.17	0.2175
1 min after ECT	78.76	4.18	75.24	11.55	0.1583
3 min after ECT	79.04	4.40	75.88	2.07	0.0021
5 min after ECT	79.48	4.32	77.04	1.81	0.0123
10 min after ECT	80.72	4.08	78.64	1.82	0.0241

Table 5: Intergroup comparison of Mean Arterial Pressure (MAP)

MEAN ARTERIAL PRESSURE (mmHg)	Group E		Group D		P value
	Mean	S.D	Mean	S.D	
Baseline	94.88	3.18	94.41	1.43	0.5066
Preoxygenation	87.87	3.03	86.92	1.21	0.1538
1 min after ECT	92.24	2.87	88.59	7.69	0.0307
3 min after ECT	92.36	3.34	89.13	2.09	0.0002
5 min after ECT	92.72	3.11	90.27	1.37	0.0007
10 min after ECT	94.27	2.89	91.72	1.58	0.0003

DISCUSSION

The hemodynamic alteration is a known complication of ECT. It has been observed in all patients in our study in baseline monitoring. The attenuation of hemodynamic parameters during preoxygenation and at 1,3,5 and 10 minutes of ECT shock has been studied in both esmolol and Dexmedetomidine groups. Our observations were similar to findings of Castelli et al (90), Begec Z et al (96), Aydogan MS et al and Bagle AA et al.

Hemodynamic attenuation was the main parameter of all these studies.

In addition, our results were parallel to the studies carried out by Li X et al, Sharan R et al and Van den Broek WW et al who found significant depression of heart rate and systolic blood pressure during their studies.

Our study was not aligned with Fu W and White PF where they failed to get significant decrease in Peak Blood pressure and Heart rate response after ECT. Hence, they concluded that Dexmedetomidine at a dose of 0.5 to 1 mcg/kg is not beneficial in controlling acute hyper dynamic response in ECT.

Zvara DA et al (1997) studied the use of esmolol during ECT with patients having myocardial ischemia. There were no significant ECG changes in comparison with placebo group Post ECT.

CONCLUSION

Summary

ECT consists of programmed electrical stimulus of Central nervous system to initiate seizure activity. In terms of haemodynamic effects, seizure activity causes an initial parasympathetic discharge later follwed by sympathetic discharge. In cardiovascular compromised patient acute alteration in autonomic nervous system may be hazardous and may bring out fatal complications. The current hospital based observational study was undertaken to compare the effects of intravenous esmolol and Dexmeditomidine as pretreatment regimens on attenuation of cardiovascular response in 50 patients. In our study the haemodynamic status was assessed in terms of HR, SBP, DBP and MAP and the variables were recorded before induction (baseline), at 1 ,3,5,10 minutes after ECT. The baseline observations were statistically insignificant and the SBP and DBP were significantly decreased during the observations. However, the mean BP attenuation was statistically insignificant. Our study also covered post ECT recovery and the time of return to spontaneous respiration was insignificant statistically.

Conclusion

Based on the current hospital based observational study, we came to conclusion that as an intravenous premedication agent in ECT both esmolol at a dose of 1mg/kg and Dexmedetomidine at a dose 0.5mcg/kg results in attenuation of cardiovascular (HR, SBP, DBP) effects but more reduction of hemodynamic parameters were seen with Dexmedetomidine compared to esmolol. There was no significant effect on recovery of spontaneous respiration. The mean blood pressure was also not affected.

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