ANAESTHESIA, PAIN & INTENSIVE CARE
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ORIGINAL ARTICLE

Outcome of pretreatment regimens on hemodynamic parameters and emergence during electroconvulsive therapy –a study on dexmedetomidine and esmolol

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Received: 23 January 2019;
Reviewed: 24, 30 January, 7 February 2019;
Revision: 26 January 2019;
Accepted: 13 February 2019

ABSTRACT

Aim: Present study was performed with an aim to observe the effect on hemodynamic parameters and emergence during electroconvulsive therapy using dexmedetomidine and esmolol as pretreatment regimens.

Methodology: In this prospective study, we selected all patients undergoing ECT from January 2017 to February 2018 in our hospital by convenient sampling, and divided them as follows; Group C (Control Group): patients who did not receive any pretreatment. Group D (Dexmedetomidine Group): inj dexmedetomidine 0.5 µg/kg diluted with 10 ml normal saline and administered over 10 min and Group E (Esmolol Group): inj esmolol 1mg/kg diluted in 10 ml normal saline and administered over 2 min during preoxygenation. Pulse rate, systolic, diastolic blood pressure and SpO_2 were measured at baseline value and compared after administration of drugs at various intervals.

Results: We observed statistically significant reduction in mean heart rate and blood pressure in Group E, followed by Group D after administration of drugs. Highly significant rise in hemodynamic parameters (HR, SBP, DBP) from baseline were observed in Group C at 1, 3 and 5 min after ECT shock and returned to baseline value at 10 min of ECT current. Obeying commands and eye opening were significantly delayed in Group D when compared to Group C and Group E. ($p \leq 0.05$)

Conclusion: Attenuation of hemodynamic parameters during ECT are effectively achieved by inj esmolol 1 mg/kg followed by inj dexmedetomidine 0.5 µg/kg, but dexmedetomidine produces delayed recovery and attenuates emergence agitation better than esmolol without affecting seizure duration or any other complications.

Key words: Attenuation, Agitation, Dexmedetomidine, Electroconvulsive therapy, Esmolol

Citation: Modh DB, Parmar MP, Solanki S. Outcome of pretreatment regimens on hemodynamic parameters and emergence during electroconvulsive therapy –a study on dexmedetomidine and esmolol. Anaesth. pain & intensive care 2019;23(1):52-58

INTRODUCTION

The use of electroconvulsive therapy (ECT) was first described in 1938 and continued to be performed without anesthesia for almost 30 years. 'Modified electroconvulsive therapy' is a safe, non-controversial and effective treatment used for resistant psychiatric illnesses when other modalities are failed. Seizures

are produced artificially by placing electrodes over temples and / or forehead and delivering electric current which is characterized by an initial brief period of muscular contractions, followed within 15 sec by a tonic phase, persisting up to 20 sec which is gradually replaced by clonic phase lasting few seconds to over one minute. These noxious stimuli are associated with unpredictable, undesirable and transient cardiovascular and cerebrovascular changes.[1-3]

The typical cardiovascular response to ECT consists of central activation of autonomic nervous system, with initial parasympathetic nervous system stimulation, primarily through direct neuronal stimulation of the hypothalamus to the vagal nerve induced bradycardia lasting 10 to 15 sec, followed immediately by a more prominent sympathetic response that results in hypertension, tachycardia, and arrhythmias etc. The serum levels of epinephrine and norepinephrine rise 15 fold and 3 fold respectively. Metabolic demands of brain, blood flow velocity of the middle cerebral artery, intracranial pressure, cerebral metabolic rate of oxygen ($CMRO_2$) and regional cerebral O_2 saturation are increased following seizure induction which may result in headache, confusion, agitation, restlessness, disorientation, cognitive side effects etc.[4-8]

These changes are usually not hazardous to normal healthy patients of ASA I or II, but may precipitate myocardial ischemia, pulmonary edema, rarely asystole, intracranial hemorrhage, or cerebrovascular accident in old age, cardiac and neurological patients. Cardiovascular complications are the main cause of death during modified ECT with a mortality rate of 0.03%.[7,9] Therefore, anesthesiologists are very conscious regarding attenuation of hemodynamic changes as well as post treatment recovery and that requires ideal pretreatment regimens. No ideal pretreatment regimen has been identified and unfortunately very few studies are available on post procedure recovery and emergence agitation. Resultantly, various agents are used as pretreatment regimens like local anesthetic (lignocaine), ganglionic blocker (trimethephan), β-blockers (esmolol, labetalol), calcium channel blockers (nefidipine, nicardipine), α-2 agonists (clonidine, dexmedetomidine) , direct vasodilators (nitroglycerine, sodium nitroprusside), opioids (fentanyl, remifentanyl, alfentanil etc.) to improve the comfort and safety of modified ECT.[5,10-13]

Our primary objective in this study was to compare cardiovascular effects of inj esmolol 1 mg/kg and inj dexmedetomidine 0.5 μg/kg as pretreatment regimens and assess effectiveness in attenuation of hemodynamic changes during ECT. Secondary objective was to evaluate postoperative recovery and agitation and complications if any.

METHODOLOGY

A prospective study was carried out at Department of Anesthesiology, GMERS Medical College Hospital, Dharpur- Patan from January 2017 to February 2018. Inclusion criteria were adult patients (18 to 40 y), ASA-I and II, and Mallampati grade I and II. We excluded pediatric, geriatric age group and pregnant patients, patients with difficult intubation, obesity, systemic diseases and allergy. Ethical approval was taken from the Institutional Ethical Committee.

Pre anesthesia evaluation of patients was done in the morning on the day of electroconvulsive therapy. Routine blood investigations, ECG and chest x-ray were carried out in all patients. Patients were kept nil by mouth for 10 h before procedure. In pre-operative room patients were examined clinically, and heart rate (HR), systolic BP (SBP), diastolic BP (DBP), and SpO_2 were noted with Schiller multipara monitor. Agitation score was noted. Intravenous line secured with 20G IV cannula and infusion was started slowly.

All the patients were premeditated with inj glycopyrrolate 0.2 mg and inj ondansetron 4 mg IV 10 min before procedure and shifted to ECT room. Patient was shifted on table and Schiller multiparameter monitor attached. Baseline heart rate, SBP, DBP and SpO_2 recorded as 'B'.

Patients received inj dexmedetomidine 0.5 μg/kg diluted with normal saline up to 10 ml and administered intravenously slowly over 10 min in Group D While in Group E, inj esmolol 1 mg/kg diluted with normal saline up to 10 ml was administered IV within 2 min, when preoxygenation started. Patients did not receive any drug in Group C (Control group). Hemodynamic parameters e.g. heart rate, SBP, DBP, and SpO_2 were recorded as 'P'.

Patient was pre oxygenated with 100% oxygen for 3 min and patient was induced with inj Propofol 1 mg/kg intravenously & inj Succinylcholine 0.5 mg/kg. An oral soft bite block was placed and electroconvulsive therapy shock current was delivered after 1 min from induction. All patients received same protocol of ECT shock. The effectiveness of ECT current was verified by appearance of tonic – clonic seizures and time. Ventilation with 100% O_2 continued until adequate respiration. HR, SBP, DBP and SpO_2 were measured and compared after administration of drugs , 1 min, 3 min, 5min and 10 min after ECT shock and recorded as E1, E3, E5, and E10 respectively.

Post op recovery parameters were assessed by time to return spontaneous breathing, time required to obey command and eye opening as well as agitation score. Timing noted after administration of succinylcholine. Agitation score was evaluated at 5 and 15 min after

return of spontaneous respiration. [Emergence agitation score- 1. Sleeping, 2. Awake and calm, 3. Irritable and crying, 4. Inconsolable crying, 5. Severe restlessness and disorientation].

Patients were observed for hypotension, bradycardia, arrhythmias, broncholaryngospasm, nausea, vomiting and other complications.

Statistical analysis:

The data were coded and entered into Microsoft Excel spreadsheet. Analysis was done using SPSS version 15 (SPSS Inc. Chicago, IL, USA) software program. The variables were assessed for normality using the Kolmogorov-Smirnov test. Descriptive statistics were calculated.

RESULTS

As shown in Table 1, mean age, sex ratio and weight were statistically comparable in all groups. Baseline HR, SBP and DBP were also statistically comparable in all of the three groups. There was significant reduction in mean values of HR, SBP and DBP in Group D and in Group E after administration of drugs, whereas no changes were seen in Group C (Control Group).

There was marked increase in hemodynamic parameters recorded in Group C (heart rate by 42 bpm, SBP by 23 mmHg and DBP by 10 mmHg) followed by dexmedetomidine Group which showed near baseline values of hemodynamic parameters followed by Group E in which heart rate and blood pressure remain all time low from baseline after one minute of ECT shock.

Table 1: Demographic data

Variables	Group C	Group D	Group E
Age (years) (Mean ± SD)	32.31 ± 4.26	35.22 ± 5.85	32.52 ± 4.17
Gender (M : F)	21:9	23:7	22:8
Weight (kg) (Mean ± SD)	53.34 ± 6.38	52.12 ± 7.06	54.11 ± 3.24

Mean hemodynamic parameters were significantly higher at 3 and 5 min after delivering current during ECT in Group C while mean HR decreased by 1 and 4 bpm, SBP reduced by 6 and 10 mmHg and DBP by 1 and 4 mmHg at 3 and 5 min after ECT shock respectively in Group D. We observed significantly lower mean HR and BP in Esmolol group at 3 and 5 min of ECT current from baseline. At 10 min of ECT shock, hemodynamic parameters returned to baseline values in Group C & E, while in Group D, HR and BP (systolic and diastolic) remained low from baseline which was statistically significant.

Mean time to return of spontaneous respiration was comparable in Group C, D and E, e.g. 121.24 ± 36.39 min vs. 126.44 ± 54.50 min vs. 124.54 ± 44.0 min respectively.

Response to verbal commands and eye opening were comparable in Group C and E, while in Group D a significant delay in above parameters was seen as compared to Group C and E.

Transient bradycardia in 4 and hypotension in 2 patients were observed in esmolol group. Emergence agitation score > 2 was observed in 4 patients and 5 patients of Group C and E respectively, while we did not observe emergence agitation in Group D patients.

DISCUSSION

Appropriate anesthesia management is a critical part of successful ECT. Anesthesiologist aims to provide musculoskeletal relaxation with airway protection, attenuation of hemodynamic and cerebrovascular changes, prevention of psychological and physiological trauma as well as smooth and prompt recovery without compromising benefits of ECT. Hence, full knowledge and understanding of physiology of ECT, as well as the mechanism and systemic effects during and after procedure, allow the anesthesiologist to strike a balance between under-medication and over-sedation, as well as to be able to anticipate problems and limit complications.

We observed a significant reduction in mean hemodynamic parameters after administration of esmolol which might be due to negative inotropic and chronotropic action on heart. Blockage of beta adrenergic receptors on SA node and AV node, reduction of conduction velocity and increase in refractory period in AV nodal tissue properties are therapeutic for sinus tachycardia and dysrhythmias and may be responsible for cardiac stability following ECT. Esmolol, an ultrashort acting β1 blocker, provides the added dimension of 'titrability' due to its pharmacokinetic properties, which make it suitable for rapid on and off effects during short procedures like ECT. Because of pharmacokinetics, negative inotropic and chronotropic action on cardiovascular system, it effectively attenuates cardiovascular changes besides playing a cardioprotective role.[14-16] Rapid bolus and higher doses of esmolol may cause bronchospasm, severe transient bradycardia and hypotension and may affect seizure duration.

But for dexmedetomidine, the optimal dose which can keep balance between attenuation of hemodynamic changes and recovery following delivery of electric current without affecting quality of seizures is still under question.[17-24] In our study, we used inj dexmedetomidine 0.5 μg/kg infusion, and found significant reduction in mean values of HR, SBP and

Table 2: Comparative mean heart rate (beats/min) in three groups

Reading	Group C	Group D	Group E	p value
B	84.2 ± 5.13	82.5 ± 4.24	82.6 ± 3.88	**0.54**
P	84.4 ± 5.21	74.3 ± 6.44	64.5 ± 4.53	**0.05***
E1	126.9 ± 12.84	83.1 ± 3.06	76.73 ± 9.35	**0.001***
E3	110.6 ± 8.42	81.4 ± 2.60	74.22 ± 7.19	**0.02***
E5	94.56 ± 4.06	78.66 ± 2.74	70.86 ± 5.08	**0.45**
E10	83.50 ± 3.92	76.80± 3.51	82.44 ± 2.74	**0.01***

** indicates statistically significance at p ≤ 0.05 Test applied one-way ANOVA*

Table 3: Comparative mean systolic blood pressure (mmHg) in three groups

Reading	Group C	Group D	Group E	p value
B	124.93 ± 5.67	122.0 ± 4.29	124.26 ± 3.53	0.47
P	125.14 ± 4.94	114.0 ± 7.18	102.19 ± 6.28	0.04*
E1	148.46 ± 11.47	128.46± 6.24	115.11 ± 7.09	0.12
E3	136.18 ± 12.39	116.20 ± 7.79	115.21 ± 8.11	0.006*
E5	132.0 ± 12.64	112.6 ± 9.5	117.9 ± 4.6	0.001*
E10	126.74 ± 9.81	114.14 ± 5.08	121.4 ± 6.5	0.05*

** indicates statistically significance at p ≤ 0.05 Test applied one-way ANOVA*

Table 4: Comparative mean diastolic blood pressure (mmHg) in three groups

Reading	Group C	Group D	Group E	p value
B	78.8 ± 4.23	78.6 ± 5.81	79.9 ± 6.15	0.009
P	80.4 ± 3.32	74.3 ± 3.17	70.3 ± 4.67	0.45
E1	88.5 ± 7.15	80.4 ± 4.32	73.2 ± 4.13	0.6
E3	86.2 ± 6.24	79.3 ± 5.12	72.8 ± 5.40	0.002*
E5	83.5 ± 6.57	74.4 ± 6.06	72.4± 5.09	0.01*
E10	80.0 ± 7.49	75.8 ± 4.27	76.8 ± 3.42	0.003*

** indicates statistically significance at p ≤ 0.05 Test applied one-way ANOVA*

Table 5: Comparison of post procedure recovery parameters

Parameters	Group C	Group D	Group E	P value
Spontaneous respiration (sec)	121.24 ± 36.39	126.44 ± 54.50	124.54±44.0	0.05*
Eye Opening (sec)	315.43 ± 83.91	404.52 ± 122.47	294.57 ± 92.41	0.002*
Obeying commands (sec)	305.56 ± 66.24	388.01 ± 125.18	290.34 ± 86.62	0.04*

** indicates statistically significance at p ≤ 0.05 Test applied one-way ANOVA*

DBP. Mean hemodynamic parameters were found near baseline soon after delivering ECT, but remained low from baseline at 3, 5, and 10 min of ECT. Variable results were seen in various studies, which found that dexmedetomidine at dose of 0.2 μg/kg attenuated hemodynamic insult. Moshiri F and colleagues found 0.5 μg/kg dose less effective than alfentanil, while Fu W and colleagues concluded that dexmedetomidine was ineffective to block the acute hemodynamic responses to ECT, but Aydogan MS recorded different result than Fu W and colleagues.[25-33,34-38]

Proper administration timing and dosage in relation to the stimulus is important. Due to pharmacokinetic properties, we administered esmolol 1 mg/kg diluted in normal saline in 2 min, and dexmedetomidine 0.5 μg/kg in 10 min. Distribution half-life of esmolol is about 2 min and it may contribute to its rapid onset and offset. Distribution half-life of dexmedetomidine is about 6 min. It will take 10-15 min for onset of action. Similar pattern of administration of drugs has been observed in various studies.[12,16,29,31,32,35,36,38]

Dexmedetomidine decreases perioperative catecholamine release. The α2 adrenoreceptors are involved in regulating the autonomic nervous system and cardiovascular system. It inhibits release of epinephrine and norepinephrine by acting on blood vessels and sympathetic presynaptic terminals. A reduction of tonic levels of sympathetic out flow and an augmentation of vagal activity results in decrease in heart rate and cardiac output. α agonists produce hyperpolarization of noradrenergic neurons and suppression of neuronal firing in the locus ceruleus leading to decreased systemic noradrenaline release. Baroreceptor reflex is well preserved in patients who received dexmedetomidine and reflex heart rate response to a stimulus is augmented, It prevents transmural redistribution of blood flow by specific epicardiac vasoconstriction effect leading to improvement in endocardial perfusion and lower heart rate. Thus It plays cardioprotective role.[17,18,23,24]

High dose of inj dexmedetomidine with rapid administration may cause severe reflex bradycardia, severe hypotension as well as irregular ventilation and apnea.[19,20]

Time to return of spontaneous respiration following ECT was comparable in all groups. Delay in eye opening and obeying verbal command was observed

with dexmedetomidine as compared to other groups, due to anxiolytic and hypnotic properties of dexmedetomidine. Dexmedetomidine decreases cerebral blood flow velocity but its ratio with cerebral metabolic rate i.e. flow metabolism coupling appears to be preserved and plays neuroprotective role. All these effects may attenuate agitation following ECT by dexmedetomidine and produce excellent recovery, which is in similarity with studies done by various authors.[22,25-27,32-36]

Seizure duration is important indicator and seizure time > 25 sec is sufficient to produce therapeutic effects. We did not found any patient with < 25 sec seizure duration which means esmolol and dexmedetomidine in given doses do not affect seizure duration.[8,32]

Transient bradycardia or hypotension was observed in esmolol group, but did not require any treatment. Emergence agitation was managed by inj midazolam. We didn't observe agitation score greater than 2 in any patient of Group D.

CONCLUSION

Attenuation of hemodynamic parameters during ECT are effectively achieved by esmolol 1 mg/kg or dexmedetomidine 0.5 μg/kg IV. Dexmedetomidine produces slightly delayed recovery but attenuates emergence agitation better than esmolol without affecting seizure duration and without any complications.

Conflict of interest: None declared by the authors.

Authors' contribution:

DM: Concept, analysis

MP: Data collection, drafting

SS: Statistical analysis, manuscript drafting

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