

ATTENUATION OF INTRAOCULAR PRESSURE RISE BY SUCCINYLCHOLINE IN RAPID SEQUENCE INDUCTION BY DEXMEDETOMIDINE- AN OBSERVATIONAL STUDY

Sanjay Choubey¹, Saurabh Mishra², Pragati Garg³, Arindam Sarkar⁴, Aditi Gupta⁵

¹Professor, Department of Anaesthesiology and Critical Care, Era's Lucknow Medical College and Hospital, Sarfarazganj, Lucknow, Uttar Pradesh.

²Resident, Department of Anaesthesiology and Critical Care, Era's Lucknow Medical College and Hospital, Sarfarazganj, Lucknow, Uttar Pradesh.

³Professor and Head of the Department, Department of Ophthalmology, Era's Lucknow Medical College and Hospital, Sarfarazganj, Lucknow, Uttar Pradesh.

⁴Consultant, Department of Anaesthesiology and Critical Care, Era's Lucknow Medical College and Hospital, Sarfarazganj, Lucknow, Uttar Pradesh.

⁵Resident, Department of Ophthalmology, Era's Lucknow Medical College and Hospital, Sarfarazganj, Lucknow, Uttar Pradesh.

ABSTRACT

BACKGROUND

Patients who require endotracheal intubation in the emergency department often require a rapid sequence induction technique to protect against gastric contents aspiration and to facilitate intubation. Rapid Sequence Induction medication practices vary among the anaesthetists throughout the world. The use of succinylcholine causes an undesirable rise in intraocular pressure. Dexmedetomidine is a highly selective centrally acting α_2 -adrenergic agonist that has intraocular pressure lowering properties.

The aim and objective of the present study was undertaken to evaluate the role of dexmedetomidine for attenuation of IOP.

MATERIALS AND METHODS

It was an observational study on 60 patients aged 20-50 years, and of ASA grade I and II, undergoing elective surgery in a tertiary care referral hospital. IOP was measured preoperatively and then just before intubation, after conclusion of premedication, thirty seconds after induction, thirty seconds after succinylcholine, immediately after intubation, two minutes, four minutes and six minutes after induction.

RESULTS

The study findings showed that at all time intervals dexmedetomidine had a better control on mean IOP.

CONCLUSION

Rise of IOP with succinylcholine and rapid sequence intubation can be blunted with I.V. dexmedetomidine premedication.

KEYWORDS

Rapid Sequence Induction, Dexmedetomidine, Succinylcholine, Intra Ocular Pressure.

HOW TO CITE THIS ARTICLE: Choubey S, Mishra S, Garg P, et al. Attenuation of intraocular pressure rise by succinylcholine in rapid sequence induction by dexmedetomidine- an observational study. J. Evid. Based Med. Healthc. 2018; 5(18), 1494-1497. DOI: 10.18410/jebmh/2018/313

BACKGROUND

For emergency surgeries often a rapid sequence induction technique is used to have a speedy and safe access to the airway by means of endotracheal intubation.

Both depolarizing and non-depolarizing muscle relaxants have been used for rapid sequence induction.

Financial or Other, Competing Interest: None.

Submission 04-03-2018, Peer Review 13-04-2018,

Acceptance 24-04-2018, Published 30-04-2018.

Corresponding Author:

Dr. Pragati Garg,

Professor and Head of the Department,

Department of Ophthalmology,

Era's Lucknow Medical College,

Sarfarazganj, Hardoi Bypass,

Lucknow – 226003.

E-mail: drpragati89@gmail.com

DOI: 10.18410/jebmh/2018/313

Among depolarizing muscle relaxants, Succinylcholine is the drug of choice because of its fast onset and short duration of action.¹ But its use causes an undesirable rise in intraocular pressure which is further aggravated by laryngoscopy and endotracheal intubation.¹ This may prove hazardous in patients with penetrating eye injuries and patients with preexisting high intraocular pressure.²

The average increase in intraocular pressure after Succinylcholine 1.0mg/kg is 4-8mmHg. The increase occurs promptly after intravenous injection peaking at 1-2 minutes and lasting as long as the neuromuscular block lasts. The cause is multifactorial, including increase in the choroidal blood volume, extraocular muscle tone and aqueous humor outflow resistance.^{3,4} Various methods have been used to attenuate the effects of succinylcholine on IOP, however, no modality has been devoid of drawbacks and limitations.



It has been demonstrated that dexmedetomidine, which is a highly selective centrally acting α_2 adrenergic agonist, when given in a dose of 0.6 μ g/kg at the induction of anaesthesia abates the haemodynamic changes and increase in IOP in response to laryngoscopy and endotracheal intubation.⁵

So in the present study we tried to evaluate the efficacy of dexmedetomidine at a selective dose of 0.5 μ g/kg in attenuating the increase in IOP due to succinylcholine used in rapid sequence induction.

MATERIALS AND METHODS

The present study was conducted in a tertiary hospital with the collaboration of an ophthalmologist and an anaesthesiologist.

60 adults patients in the age group 20-50 years scheduled for elective surgeries under general anaesthesia, ASA grade I and II physical status (I is normal healthy patient; II is patient with mild systemic disease) were enrolled for the study after proper ethical clearance and informed consent of the patient. Patients with ocular disease with or without increased IOP; with anticipated difficult intubation; Mallampati grade III and IV (Visualization of the soft palate and the base of the uvula; Soft palate is not visible at all respectively); history of cardio respiratory illness; obesity; and contraindication to the use of Succinylcholine or Dexmedetomidine were excluded from the study.

The patients were randomly allocated into two groups of 30 each according to the computer generated randomized tables. Group I (Dexmedetomidine Group) (n=30) received intravenous infusion of dexmedetomidine (0.5 μ g/kg) diluted in normal saline to make a solution of 50ml as premedication, over a period of 10 min. Group II (Control Group) (n=30) received intravenous infusion of 50ml normal saline as premedication, over a period of 10 min. All patients went through relevant history, and ocular and systemic examination was done to rule out any ocular disease and the tendency of getting raised intraocular pressure. Patients were kept fasting overnight and no premedication was given. Preoperative recording of heart rate (HR), noninvasive blood pressure (NIBP) and arterial oxygen saturation (SpO₂) were done. Standard anaesthetic techniques were followed. All patients were preoxygenated with 100% oxygen for 3 minutes. Induction of anaesthesia was carried out with inj. Propofol 1% 2mg/kg intravenously. Succinylcholine was administered at a dose of 2 mg/kg to achieve muscular relaxation for intubation in both the groups.

Preoperative baseline intraocular pressure (IOP) was measured with a Schiotz tonometer after instillation of 4% Lignocaine drops in the right eye by an ophthalmologist. Intraocular pressure was recorded in right eye just before

premedication (T1), after conclusion of premedication (T2), 30 seconds after induction of anaesthesia (T3), 30 seconds after succinylcholine injection (T4), immediately after intubation (T5), 2 minutes after intubation (T6), 4 minutes after intubation (T7), 6 minutes after intubation (T8).

Data so obtained was subjected to statistical analysis using SPSS software version 15.0. Results obtained were analyzed statistically by "t" test, paired and unpaired. P<0.05 was taken as significant value.

RESULTS

This study was done on a total of 60 adult patients in the age group of 20-50 years scheduled for elective surgery under general anaesthesia, after the informed consent and ethical clearance.

They were randomly divided into two groups Group I (n=30) in which intravenous Dexmedetomidine 0.5 μ g/kg diluted in normal saline so as to make a solution of 50ml was used as premedication over a period of 10 minutes; and group II (n=30) which includes patients in whom intravenous infusion of 50ml normal saline was used as premedication over a period of 10 minutes.

Mean age of patients in group I was 27.15 and in group II was 27.33. Male to female ratio was 3: 2 in group I and 2: 1 in group II with females being more in both the groups. (p=0.592) Statistically both the groups were matched for age and gender. All the patients in both the groups were ASA Grade I/II.

On comparing the IOP values between the two groups at different time intervals, we found that at baseline (T1), intraocular pressure of right eye of Group I (12.83 \pm 1.94 mm Hg) was lower than that of Group II (12.98 \pm 1.91 mm Hg) but with no statistical difference. (p=0.764)

At all other time intervals also i.e. from T2 to T8, intraocular pressure of right eye of Group I was found to be lower than that of Group II, but the difference in intraocular pressure was found to be statistically significant only from T2 to T6 (p<0.001).

When the change in IOP from baseline (T1) was observed, we found that in Group I, intraocular pressure at all the recorded time intervals remained lower than its baseline value (T1) at all the recorded time intervals and was statistically significant at all time intervals except at T4 and T5. Maximum change from its baseline value was observed at T8 (4.51 \pm 1.63 mm Hg) and minimum change was observed at T5 (0.40 \pm 1.78 mm Hg).

While in Group II, intraocular pressure of right eye was found to be lower than its baseline (T1) value at time intervals T2, T3, T6, T7 and T8 only. Nevertheless the change was found to be statistically significant at all time intervals.

Demographic Variables	Group I (n=30)		Group II (n=30)		Statistical Significance	
	No.	%	No.	%	X ²	'p'
Age Group						
21-30	9	30.00	10	33.33	0.150	0.949
31-40	11	36.67	11	36.67		
41-50	10	33.33	9	30.00		
Gender						
Male:Female	3: 2		2: 1		0.287	0.592

Table 1. Comparison of Demographic Profile of Study Population

Time Intervals	Group I	(n=30)	Group II	(n=30)	Statistical significance	
	Mean	SD	Mean	SD	't'	'p'
Baseline T1	12.83	1.94	12.98	1.91	0.302	0.764
T2	9.91	1.60	12.04	1.53	5.270	<0.001
T3	9.12	1.31	10.52	1.36	4.061	<0.001
T4	12.04	1.56	16.10	3.61	5.655	<0.001
T5	12.43	1.56	16.85	2.95	7.255	<0.001
T6	9.23	0.99	11.38	2.84	3.915	<0.001
T7	8.45	0.96	8.97	2.27	1.156	0.253
T8	8.32	0.92	8.98	2.35	1.432	0.157

Table 2. Comparison of Right Eye Intraocular Pressure (mmHg) at Different Time Intervals

Time Intervals	Group I (n=30)				Group II (n=30)			
	Mean	SD	't'	'p'	Mean	SD	't'	'p'
T1-T2	-2.92	0.65	24.605	<0.001	-0.94	0.60	8.581	<0.001
T1-T3	-3.71	0.93	21.850	<0.001	-2.46	0.77	17.499	<0.001
T1-T4	-0.79	1.63	2.655	0.013	3.12	1.81	9.441	<0.001
T1-T5	-0.40	1.78	1.231	0.229	3.87	1.68	12.617	<0.001
T1-T6	-3.60	1.69	11.667	<0.001	-1.60	1.82	4.815	<0.001
T1-T7	-4.38	1.74	13.787	<0.001	-4.01	1.52	14.450	<0.001
T1-T8	-4.51	1.63	15.155	<0.001	-4.00	1.64	13.359	<0.001

Table 3. Intragroup Change in Intraocular Pressure of Right Eye from Baseline (T1) in the Study Population (Paired 't' test)

DISCUSSION

Patients scheduled for emergency surgeries often require a rapid sequence induction (RSI) technique to have a speedy and safe access to the airway by means of endotracheal intubation.

Succinylcholine is the most commonly used muscle relaxant in rapid sequence induction because of its fast onset and short duration of action when given in the recommended 1.5 mg/kg I.V. dose, although, it can have serious side effects. Though it remains the relaxant of choice in non-fasting patients, yet succinylcholine produces an undesirable rise in IOP.⁶

Though various methods have been used to minimize the IOP rise by succinylcholine like dividing it's dose into two slots where 1/10th dose is given initially followed by the remaining amount, and also use of non-depolarizing neuromuscular blocking agents, lidocaine, opioids, nifedipine or nitroglycerine as premedication, but none was devoid of drawbacks and limitations.

It has been demonstrated that dexmedetomidine (α^2 adrenergic receptor agonist), 0.6 μ g/kg, administered at the induction of anaesthesia abates the haemodynamic changes

and increase in IOP in response to laryngoscopy and endotracheal intubation.^{7,8}

There is no absolute contraindication to the use of dexmedetomidine. Limiting its usefulness in the caution that the drug cannot be bloused, due to concerns about peripheral α^2 receptor stimulation with resulting hypotension and its high cost in comparison to generic medications.⁹

Recent studies have shown its successful use for attenuation of succinylcholine induced rise in IOP among patients undergoing RSI.^{2,5,7,10,11}

We looked for baseline IOP as well as IOP post intubation till 6 minutes in both the groups. IOP at baseline was almost matching with each other in both the groups being 12.83 and 12.98 mmHg respectively.

In group I, IOP remained lower than in group II at all times post premedication but the difference was significant till T6 only upto two minutes after intubation. Thus showing that dexmedetomidine had a better control on mean IOP raised due to succinylcholine.

In group I, mean IOP at all times after premedication remained lower than baseline while in group II, mean IOP

increased from baseline at T4 and T5 i.e 30 seconds after succinylcholine injection and immediately after intubation.

Similar to the findings of previous study were the findings of Mowafi et al.⁵ who used dexmedetomidine in a dosage of 0.6µg/kg as premedication. They found that in the dexmedetomidine group IOP rise was not different from the baseline value (p=0.65) and was significantly lower than in the succinylcholine group (p=0.003). According to them, the effect of dexmedetomidine on IOP could be caused by a direct vasoconstrictor effect on the afferent blood vessels of the ciliary body, which results in reduction of aqueous humor production.¹² Moreover, it could increase outflow of the aqueous humor caused by a reduction of the sympathetically mediated vasomotor tone of the ocular drainage system.¹³

Kolarkar et al.¹⁴ in 2014 did the same study and found out a maximum mean change in IOP to the magnitude of 35.7% in control group as compared to 1.4493% in dexmedetomidine group, thus showing a difference of nearly 34.2% between the two groups at a point when the haemodynamic reflex following intubation was at its peak.

There were only two studies till date who have examined the effect of dexmedetomidine on the succinylcholine induced ocular hypotension.^{5,14} Many other researches have been done to observe the effect of dexmedetomidine in attenuating the rise of IOP but not in response to succinylcholine but to other drugs like suxamethonium.^{2, 11}

CONCLUSION

The results in the present study show that dexmedetomidine not only attenuated the haemodynamic effect of succinylcholine but was also helpful to reduce the haemodynamic reflex following RSI which continued to be maintained even after 6 minutes after intubation.

Thus, it is concluded as a safe drug for premedication especially in those patients where rise of IOP can be dangerous. But still further studies are recommended to corroborate the findings.

REFERENCES

- [1] Lee C, Katz R. Clinical implications of new neuromuscular concepts and agents: so long, neostigmine! So long, sux! J Crit Care 2009;24(1):43-49.
- [2] Pal CK, Ray M, Sen A, et al. Changes in intraocular pressure following administration of suxamethonium and endotracheal intubation: influence of dexmedetomidine premedication. Indian J Anaes 2011;55(6):573-577.
- [3] Sinclair RCF, Luxton MC. Rapid sequence induction. Continuing Education in Anaesthesia, Critical Care & Pain 2005;5(2):45-48.
- [4] Jantzen JP. Anaesthesia and intraocular pressure. Anaesthesist 1988;37(8):458-469.
- [5] Mowafi HA, Aldossary N, Ismail SA, et al. Effect of dexmedetomidine premedication on the intraocular pressure changes after succinylcholine and intubation. Br J Anaesth 2008;100(4):485-489.
- [6] Cook JH. The effect of suxamethonium on intraocular pressure. Anaesthesia 1981;36(4):359-365.
- [7] Jaakola ML, Ali MT, Kanto J, et al. Dexmedetomidine reduces intraocular pressure, intubation responses and anaesthetic requirements in patients undergoing ophthalmic surgery. Br J Anaesth 1992;68(6):570-575.
- [8] Scheinin B, Lindgren L, Randell T, et al. Dexmedetomidine attenuates sympathoadrenal responses to tracheal intubation and reduces the need for thiopentone and preoperative fentanyl. Br J Anaesth 1992;68(2):126-131.
- [9] Naaz S, Ozair E. Dexmedetomidine in current anaesthesia practice- a review. J Clin Diagn Res 2014;8(10):GE01-GE04.
- [10] Yavascaoglu B, Kaya FN, Baykara M, et al. A comparison of esmolol and dexmedetomidine for attenuation of intraocular pressure and haemodynamic responses to laryngoscopy and tracheal intubation. Eur J Anaesthesiol 2008;25(6):517-519.
- [11] Shalini A, Srinivas VY, Gurudatt CL. Can dexmedetomidine premedication obtund the intraocular pressure rise after suxamethonium and endotracheal intubation. JEMDS 2014;3(27):7441-7449.
- [12] Marci FJ, Cervario SJ. Clonidine. Effects on aqueous humor formation and intraocular pressure. Arch Ophthalmol 1978;96(11):2111-2113.
- [13] Vartiainen J, MacDonald E, Urtti A, et al. Dexmedetomidine-induced ocular hypotension in rabbits with normal or elevated intraocular pressures. Invest Ophthalmol Vis Sci 1992;33(6):2019-2023.
- [14] Kolarkar P, Badwaik G, Pahuja H, et al. Dexmedetomidine: as novel premedication. JEMDS 2014;3(36):9406-9415.