(International Print/Online Journal)

SJIF IMPACT FACTOR: 5.565
PUBMED-National Library of Medicine ID-101739732

ISSN (Print): 2209-2870
ISSN (Online): 2209-2862

International Journal of Medical Science and Current Research (IJMSCR)
Available online at: www.ijmscr.com
Volume 4, Issue 3, Page No: 205-211
May-June 2021

Role Of Dexmedetomidine Pre-Medication On Hemodynamic Response To Laryngoscopy, Endotracheal Intubation And Intraocular Pressure Changes Following Suxamethonium Administration In Patients Undergoing Elective Non-Ophthalmic Operations Under General Anesthesia

Dr. Shaista Yaqoob[1,*], Dr. Azka Sunain Khan[2], Dr, Gazala Yaqoob[3], Dr. Abdul Qayoom Lone[4]

[1, 2]Senior Resident, [3]Resident, [4]Professor

[1, 2, 4]Department of Anesthesiology and critical care, Sher-i-kashmir institute of medical sciences, Soura, Jammu & Kashmir, India

[3]Department of Physiology, Government Medical College, Srinagar, Jammu & Kashmir, India

***Corresponding Author:**
Dr. Shaista Yaqoob

[1,*] Senior Resident,Department of Anesthesiology and critical care, Sher-i-kashmir institute of medical sciences, Soura, Jammu & Kashmir, India

Type of Publication: Original Research Paper
Conflicts of Interest: Nil

ABSTRACT

INTRODUCTION Suxamethonium, a depolarising muscle relaxant used for rapid sequence airway management causes an increase in intraocular pressure (IOP). Laryngoscopy and endotracheal intubation further aggravate this rise in intraocular pressure. This rise in intraocular pressure may be harmful in patients with penetrating eye injuries.

AIMS: The purpose of our study was to observe the efficacy of Dexmedetomidine, given as premedication in prevention of rise in intraocular pressure associated with the administration of suxamethonium, laryngoscopy and endotracheal intubation.

METHODOLOGY: One hundred ASA Class I or II patients undergoing general anaesthesia for elective non-ophthalmic surgery were included in this double blind, randomized, prospective, clinical study. Patients were allocated into two groups of fifty each to receive 0.4 µg/kg Dexmedetomidine(group D) or normal saline (group C) intravenously before induction. Heart rate, mean arterial pressure and intraocular pressure were measured before premedication, after suxamethonium injection and after endotracheal intubation.

RESULTS: Dexmedetomidine administration resulted in decrease in HR, MAP and IOP from baseline values. IOP increased after suxamethonium and intubation in both groups but it never increased above baseline values in the study group. Also increase in HR and MAP in the study group after endotracheal intubation was lesser than that seen in the control group.

CONCLUSION: Dexmedetomidine 0.4 µg/kg given intravenously as premedication effectively prevents the rise in IOP associated with the administration of suxamethonium, laryngoscopy and endotracheal intubation and also attenuates the hemodynamic pressor response to laryngoscopy and endotracheal intubation.

Keywords: Dexmedetomidine ; Suxamethonium; Endotracheal intubation; Intraocular pressure.

INTRODUCTION

The perforating injury of the eye has become challenge for anaesthesiologists and surgeons for its management. These patients are considered full stomach, and therefore, require rapid sequence induction (RSI) and intubation without causing rise of intraocular pressure. Precautions have to be taken

to prevent rise of intraocular pressure on one hand and complications of full stomach on the other.[1] When the eye globe is open, any factor that increases the intraocular pressure (IOP) may cause drainage of the aqueous humour or extrusion of the vitreous humour through the wound, which can permanently damage vision.[1] Suxamethonium is used to facilitate rapid tracheal intubation in patients with high-risk for aspiration because of its fast onset time and very good intubating conditions. It is however associated with an increase in the IOP. Laryngoscopy and tracheal intubation further aggravate the rise in IOP.[1,2] Various methods have been tried from time to time to attenuate the effects of succinylcholine on intraocular pressure. These include self-taming or pre-treatment with small dose of non-depolarising neuromuscular blocking agents, lidocaine, narcotics,etc.[3-8] However, no treatment modality for the purpose has been established to be devoid of drawbacks and limitations.

Pre-treatment with α_2-agonists has also been tried by many anaesthesiologists to control the haemodynamic response to laryngoscopy and endotracheal intubation as well as rise in intraocular pressure following succinylcholine administration.[9] Dexmedetomidine which is a highly specific and selective α_2-adrenoreceptor agonist compared to clonidine has been shown to have intraocular pressure lowering properties besides some well documented perioperative beneficial effects like anxiolysis, sedation, analgesia and sympatholysis with minimal respiratory depression.[10,11]

The aim of this study is to determine the efficacy of 0.4 µg/kg of intravenous (i.v.) dexmedetomidine for prevention of haemodynamic response to laryngoscopy and intubation and rise of IOP by suxamethonium and endotracheal intubation.

MATERIALS AND METHODS: The study was undertaken after obtaining informed consent from all patients. One hundred patients of ASA class I or II, between 20 years to 50 years of age, scheduled for various elective non-ophthalmic surgeries requiring general anaesthesia were selected for the study. Patients less than 20 and more than 50 years of age, with Mallampatti class III and IV, obese (BMI >30), with difficult airway, patients with any contraindication to the study drug and to suxamethonium, having acute /chronic eye disease or with raised IOP, on medication known to alter intraocular pressure, having diabetes mellitus, hypertension, coronary artery disease and other co-existing diseases were excluded from the study.

A routine pre-anaesthetic examination was conducted on the evening before surgery assessing the general condition of the patient, airway examination and a detailed systemic examination especially of the cardio-respiratory system was done and documented. Routine investigations including CBC, KFT, 12 lead electrocardiogram, X-ray chest, fasting blood sugar and urine analysis were done in all patients.

For the purpose of the study, patients were randomly allocated to one of the following groups:

1) Group C (Control group): comprised of 50 patients who received 50 ml normal saline intravenously over a period of 10 minutes, 10 minutes prior to induction of general anaesthesia.

2) Group D (Drug/Study group): again comprised of 50 patients who received 0.4µg/kg of dexmedetomidine diluted in 50 ml of saline over 10 minutes, 10 minutes prior to induction of general anaesthesia.

On arrival to the main operation theatre induction area, IV cannulation was performed with 18-20 G cannula. ECG (Rate and rhythm) and non-invasive blood pressure (systolic, diastolic and mean) were monitored and baseline values were recorded. For the purpose of the study two drops of 4% topical xylocaine was put in each eye to anaesthetise the cornea and baseline intraocular pressure was recorded for each patient using a tonometer. The placebo/drug solutions were prepared and labelled as dexmedetomidine/saline by the OT incharge technologist who was not involved in the study anymore. He confidentially maintained records of every patient and the actual solution used. The study parameters including heart rate, blood pressure and intraocular pressure were observed and recorded by the incharge anaesthesiologist. Standard intraoperative monitoring including pulse oximetry, ECG and non-invasive blood pressure (systolic, diastolic and mean) was performed as a routine as well as for the purpose of the study. Intraocular pressure (IOP) was measured using Schiotz indentation tonometer, after prior instillation of local anaesthetic topical lignocaine 4% eye drops. The

Volume 4, Issue 3; May-June 2021; Page No 205-211

other parameters were measured using automatic multiparameter monitor.

General anaesthesia was standardized in both the groups. After preoxygenation for 3 minutes, anaesthesia was induced with Propofol 2mg/kg and Suxamethonium chloride 1.5 mg/kg. After the fasciculations had ceased, trachea was intubated with appropriate sized cuffed endotracheal tubes (portex) with gentle laryngoscopy using conventional laryngoscopic technique. Proper placement of the tracheal tube was verified by auscultation of the chest for bilateral air entry. If the trachea could not be intubated at first attempt, the patient was excluded from the study. Anaesthesia was maintained with 66% N_2O in O_2 and isoflurane 0.5-1.0% in both the groups. Fentanyl citrate (2 µg/kg body weight) was administered for the purpose of analgesia. Muscle relaxation was achieved using vecuronium bromide (0.1 mg/kg body weight).

Study parameters including **Heart rate** (HR**), mean arterial blood pressure** (MAP) and **intraocular pressure** (IOP) were recorded at the following stages:

S1: just before premedication is started. (Baseline)

S2: 1 minute after suxamethonium administration.

S3: 1 minute after laryngoscopy and endotracheal intubation.

S4: 2 minutes after endotracheal intubation.

S5: 3 minutes after endotracheal intubation.

S6: 5 minutes after endotracheal intubation.

At the end of surgical procedure, residual neuromuscular block was reversed using neostigmine 80µg/kg and glycopyrolate 10 µg/kg. Side effects like hypotension (fall in mean arterial blood pressure >30% of baseline), bradycardia (heart rate <45 beats /min), or any other were documented.

OBSERVATIONS: The groups were comparable with respect to age, weight and gender of patients (p>0.05)[Table 1].Baseline hemodynamic parameters (heart rate and mean arterial pressure) and baseline intraocular pressure (IOP) were also comparable in both the groups.(p>0.05) Increase in mean heart rate after laryngoscopy and endotracheal intubation was observed in control group (p<0.001) Heart rate remained above baseline at all study stages and the difference was significant at all time points (p<0.001).After premedication with dexmedetomidine, decrease in heart rate was observed in the study group (p<0.001). The mean heart rate remained below basal value at intubation and even at 5 minutes after intubation. Comparison of heart rate between the groups at corresponding study stages showed significantly lower heart rate in the study group at all the stages(p<0.001).[Table 2].Significant increase in mean arterial pressure from baseline value occurred with laryngoscopy and endotracheal intubation in the control group (p<0.001) which was not seen in the study group where mean arterial pressure decreased after dexmedetomidine administration and it remained below baseline at all study stages (p<0.001).[Table 3].In the control group, intraocular pressure (IOP) raised above baseline after suxamethonium administration (p<0.001). IOP further increased after intubation and remained elevated from baseline value upto 5 min after intubation (p<0.001). In the dexmedetomidine group initially there was a decrease in IOP after dexmedetomidine bolus which was statistically highly significant (p<0.001).IOP did not increase after suxamethonium administration in this group. At intubation there was a slight rise in IOP. However, IOP still was significantly lower than baseline value (P<0.01). Even at 5 minutes after intubation, the IOP remained below baseline.[Table 4]. Although heart rate and mean arterial pressure decreased after dexmedetomidine administration, no incidence of hypotension or bradycardia requiring intervention was observed in both groups.

DISCUSSION: The main finding in this study was that dexmedetomidine premedication in a dose of 0.4 µg/kg given over 10 minutes before induction of anaesthesia blunted the rise in the IOP caused by succinylcholine administration and endotracheal intubation. In addition, dexmedetomidine attenuated the haemodynamic response to laryngoscopy and intubation.The results of our study are consistent with previous several researches. Dexmedetomidine infusion as an adjunct to local analgesia in ophthalmic surgery was effective in reduction of the IOP significantly[12]. The administration of intramuscular dexmedetomidine at a dose of 1 µg/kg for premedication in outpatient cataract surgery resulted in sedation, and decrease in intraocular pressure without significant hypotension or

Volume 4, Issue 3; May-June 2021; Page No 205-211

bradycardia[13]. Pal CK in their study observed that dexmedetomidine (both 0.4µg/kg as well as 0.6 µg/kg) prevents rise of IOP associated with administration of suxamethonium and endotracheal intubation. However, they observed significant hypotension with dexmedetomidine 0.6 µg/kg and suggested that dexmedetomidine 0.4 µg/kg might be better in preventing rise of IOP without causing undue hemodynamic fluctuations[14]. Dexmedetomidine was shown to attenuate the sympathetic response to laryngoscopy and intubation in patients undergoing myocardial revascularization[15]. Dexmedetomidine 1.0 µg/kg was shown to provide a consistent, reliable and effective attenuation of pressure responses when compared to esmolol 2.0 mg/kg.[16] Dexmedetomidine (1µg/kg) attenuates hemodynamic response to laryngoscopy and intubation in elective surgery for off pump coronary artery bypass grafting[17].

However, when Lee and colleagues infused dexmedetomidine as a supplement to isoflurane anaesthesia, they found no IOP lowering effect.[18] This may be due to the fact that their patients were pre-medicated with 0.6 mg atropine i.m. which would have prevented the attenuating effect of dexmedetomidine.

The effect of dexmedetomidine on IOP may be due to its direct vasoconstrictor effect on blood vessels of ciliary body leading to reduction of aqueous humour production. It may also facilitate drainage of aqueous humour by reducing sympathetically mediated vasomotor tone of the ocular drainage system[19]. The blood pressure lowering effect of dexmedetomidine can be responsible for reduction of IOP[20].

CONCLUSIONS: From our study we can conclude that dexmedetomidine 0.4 µg/kg bodyweight i.v. given as premedication over 10 minutes before induction of general anaesthesia can effectively attenuate the rise in intraocular pressure associated with succinylcholine administration, laryngoscopy and endotracheal intubation and also attenuate the hemodynamic pressor response to laryngoscopy and intubation without adverse hemodynamic consequences. However, further work and scientific research in this field is desirable.

REFERENCES:

1. Chidiac EJ, Raiskin AO. Succinylcholine and the open eye. Ophthalmology Clinics North America Journal 2006; 19: 279-85.
2. Libonati MM, Leahy JJ, Ellison N: The use of succinylcholine in open eye surgery. Anaesthesiology 1985, 62:637-40.
3. Meyers EF, Singer P, Otto A: A controlled study of the effect of succinylcholine self-taming on intraocular pressure. Anaesthesiology 1980, 53:72-4.
4. Chui CL, Lang CC, Wong PK, Delilkan AE, Wang CY: The effect of mivacurium pre-treatment on intra ocular pressure changes induced by suxamethonium. Anaesthesia 1998, 53,486- 510.
5. *Myers EF, Krupin T, Johnson M, Zink H*: Failure of nondepolarising neuromuscular blockers to inhibit succinylcholine induced increased intraocular pressure, A controlled study. Anaesthesiology 1978, 48:149-51.
6. Chui CL, Jaais F, Wang CY: Effect of rocuronium compared with succinylcholine on intraocular pressure during rapid sequence induction of anaesthesia. Bri J Anaesth 1999; 82: 606-7.
7. Murphy DF, Eustace P, Unwin A, Magner JB.: Intravenous lignocaine pre-treatment to prevent intraocular pressure rise following suxamethonium and tracheal intubation. Bri J Ophth 1986, 70:596- 8.
8. Polarz H, Böhrer H, Fleischer F, Huster T, Bauer H, Wolfrum J : Effects of thiopentone/ suxamethonium on intraocular pressure after pre-treatment with alfentanyl. Euro J Cli Pharm 1992, 43:3,311-3.
9. Polarz H, Bohrer H, Martin E, Wolfrum J, Volcker HE: Oral clonidine premedication prevents the rise in intraocular pressure following succinylcholine administration. German J Ophth. 1993; 2:97-9.
10. Vanda G, Karam Y, Aouad MM: Perioperative uses of dexmedetomidine. M E J Anaesthesia 2006; 18(6):1043-58

Volume 4, Issue 3; May-June 2021; Page No 205-211

11. Afonso J, Reis F: Dexmedetomidine: Current Role in Anesthesia and Intensive Care. Rev Bras Anesthesiol 2012; 62: 1: 118-33.
12. Abdalla MI, Al Mansouri F, Bener A. Dexmedetomidine during local anaesthesia. J Anesth 2006;20:54-6.
13. Virkilla M, Ali Melkkila T, Konto J, Turuner J, Scheinin H. Dexmedetomidine as intramuscular premedication in outpatient cataract surgery: A placebo controlled dose ranging study. Anaesthesia 1993; 4:482-7.
14. Pal CK, Ray M, Sen A, Hajra B, Mukherjee D, Ghanta AK. Changes in intraocular pressure following administration of suxamethonium and endotracheal intubation: influence of dexmedetomidine premedication. Indian J Anaesth 2011;55:573-7.
15. Menda F, Koner O, Sayin M, Ture H, Imer P, Aykac B. Dexmedetomidine as an adjunct to anesthetic induction to attenuate hemodynamic response to endotracheal intubation in patients undergoing fast-track CABG. Ann Card Anaesth 2010;13:16-21.
16. Uysal HY, Tezer E; Türkoğlu M, Aslanargun P, Basar H. Effect of dexmedetomidine on hemodynamic response to laryngoscopy and tracheal intubation in hypertensive patients: a comparison with esmolol and sufentanyl. J Res Med Sci 2012;17(1):22-31.
17. Sulaiman S, Karthekeyan RB, Vakamudi M, Sundar AS, Ravullapalli H, Gandham R. The effects of dexmedetomidine on attenuation of stress response to endotracheal intubation in patients undergoing elective off- pump coronary artery bypass grafting. Ann Card Anaesth 2012; 15(1):39-43.
18. Lee YY, Wong SM, Hung CT. Dexmedetomidine infusion as a supplement to isoflurane anaesthesia for vitroretinal surgery. Br J Anaesth 2009;98:477-83.
19. Vartianinen J, Macdonald E, Urtti A, Ronhianen H, Virtanen R. Dexmedetomidine induced ocular hypotension in rabbits with normal and elevated intraocular pressures. Invest ophthalmology Vis Sci 1992; 33:2019-23.
20. Georgiou M, Parlapani A, Argiriadou H, Papagiannopoulou P, Katsikis G, Kaprini E. Sufentanil or clonidine for blunting the increase in intraocular pressure during rapid sequence induction. Eur J Anaesthesiol 2002; 13: 519-22.

Table 1: Showing comparison of demographic parameters between the two groups.

Demographic criteria	Group C (Control) n=50	Group D (Study) n=50	p- value
	Mean ± SD	Mean ± SD	
Age (yrs)	35.70 ± 7.335	36.80 ± 8.019	0.460*
Gender distribution(M/F)	28/22	29/21	0.840*
Weight (kg)	62.92 ± 9.739	61.00 ± 8.692	0.301*

* - *not significant*

Volume 4, Issue 3; May-June 2021; Page No 205-211

Table 2: Comparison of Heart Rate (Mean±SD) between the two groups at different stages

TIME	Heart Rate (beats/min) Mean ± SD		p-value
	GROUP C n=50	GROUP D n=50	
S1 (baseline)	87.28±7.502	88.66±8.050	0.377*
S2 (1min after suxamethonium**)**	84.84±7.533	73.30±5.905	0.000**
S3 (1 min after intubation)	117.20±10.13	85.42±6.302	0.000**
S4 (2 min after intubation)	107.22±7.487	83.36±6.043	0.000**
S5 (3 min after intubation)	101.26±6.493	82.00±6.243	0.000**
S6 (5 min after intubation)	96.14±6.411	80.16±6.339	0.000**

**-not significant (p>0.05) **- highly significant (p<0.01)*

Table 3: Comparison of mean arterial pressure (Mean ±SD) between the two groups at different study stages

TIME	MAP (mm Hg) Mean ± SD		p-value
	GROUP C n=50	GROUP D n=50	
S1 (baseline)	89.04±6.101	88.98±6.006	0.961*
S2 (1min after suxamethonium**)**	80.66±7.507	70.72±4.463	0.000**
S3 (1 min after intubation)	107.18±7.572	83.04±6.247	0.000**

Volume 4, Issue 3; May-June 2021; Page No 205-211

S4 (2 min after intubation)	102.48±5.881	81.56±6.142	0.000**
S5 (3 min after intubation)	99.68±5.787	80.36±5.992	0.000**
S6 (5 min after intubation)	97.40±5.707	79.00±5.657	0.000**

*-not significant (p>0.05) **- highly significant (p<0.01)*

Table 4: Comparison of intraocular pressure (Mean±SD) between the two groups at different study stages

Time	IOP (mm Hg) Mean ± SD		p-value
	GROUP C n=50	GROUP D n=50	
S1 (baseline)	14.526±1.7613	15.14±1.659	0.0759*
S2 (1min after suxamethonium)	16.62±1.68898	11.02±1.469	0.000**
S3 (1 min after intubation)	21.70±1.7642	13.866±1.458	0.000**
S4 (2 min after intubation)	20.50±1.8323	13.584±1.458	0.000**
S5 (3 min after intubation)	19.22±1.785	12.83±1.469	0.000**
S6 (5 min after intubation)	17.98±1.55	12.47±1.383	0.000**

*-not significant (p>0.05) **- highly significant (p<0.01)*

Volume 4, Issue 3; May-June 2021; Page No 205-211