

A comparative evaluation of the effect of intravenous dexmedetomidine and clonidine on intraocular pressure after suxamethonium and intubation

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ABSTRACT

Background: In patients with penetrating eye injury and a full stomach, suxamethonium is still used for rapid sequence induction of anesthesia. But its use is associated with the rise in intraocular pressure (IOP) and this can result in permanent vision loss in these patients. Dexmedetomidine and clonidine are two alpha-2 adrenergic agonist drugs which prevent the rise in IOP. The aim of this study is to compare the efficacy of intravenous (i.v.) dexmedetomidine and clonidine in preventing an increase in IOP after administration of suxamethonium and tracheal intubation.

Materials and Methods: Sixty patients undergoing elective nonophthalmic surgery under general anesthesia were included in this clinical study. Patients were randomly assigned into three groups to receive 0.5 mcg/kg dexmedetomidine (Group D), 2 mcg/kg clonidine (Group C) or normal saline (Group S) as premedication i.v. over a period of 10 min before induction. IOP, heart rate, and mean arterial pressure were recorded before and after premedication, after suxamethonium, after intubation and then after 5 min. **Results:** Following administration of dexmedetomidine and clonidine IOP decreased in both groups. After suxamethonium IOP increased in all three groups but it never crossed the baseline in Group D and C. After laryngoscopy and intubation IOP again increased in all three groups but in dexmedetomidine group it never crossed the baseline whereas in clonidine group it was significantly higher than the baseline. **Conclusion:** Single i.v. dose of dexmedetomidine premedication (0.5 mcg/kg) blunt the IOP and hemodynamic response to suxamethonium injection and tracheal intubation more effectively than single i.v. dose of clonidine premedication (2 mcg/kg).

Key words: Clonidine, dexmedetomidine, intraocular pressure, premedication, suxamethonium

INTRODUCTION

Increasing intraocular pressure (IOP) in ophthalmic surgery has always been problematic for the surgeon and it is necessary to prevent the elevation of IOP and control it before, during and after the surgery.^[1]

Anesthesia for a patient with a penetrating eye injury and a full stomach is a challenge to the anesthesiologist. In these cases aim of anesthesia is rapid sequence induction without increasing IOP. Anesthesiologist must weigh the risk of aspiration against the risk of blindness in the injured eye that could result from elevated IOP and extrusion of ocular contents.

Suxamethonium is the most commonly used muscle relaxant to facilitate rapid sequence intubation in patients at risk of aspiration of gastric contents, but it increases the IOP.^[2,3] Laryngoscopy and tracheal intubation further aggravate the rise in IOP.^[4] A variety of methods has been used to prevent suxamethonium induced rise in IOP with limited success. They included self-taming, pretreatment with nondepolarizing muscle relaxant, lidocaine, narcotics

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and use of nitroglycerine.^[5] Dexmedetomidine and clonidine are two alpha-2 adrenergic agonist drugs which have the property of inhibiting rise in IOP following suxamethonium, laryngoscopy, and tracheal intubation.^[6-8]

The aim of this study is to compare the effectiveness of single preinduction bolus of intravenous (i.v.) dexmedetomidine 0.5 mcg/kg and i.v. clonidine 2 mcg/kg in attenuating the rise in IOP after suxamethonium and tracheal intubation.

MATERIALS AND METHODS

After obtaining the approval of institutional ethics committee and written informed consent, 60 adult patients of American Society of Anesthesiologists (ASA) 1 or 2, aged 18-60 years, who were scheduled for elective nonophthalmic surgeries under general anesthesia were included in the study. Exclusion criteria include patients suffering from hypertension, acute or chronic eye disease, any known allergies or contraindication of study drugs, predicted difficulty in intubation and pregnancy. Patients were also not included if they were receiving any drug known to alter IOP.

The patients were randomly allocated into three groups of 20 patients each to receive 0.5 mcg/kg dexmedetomidine (Group D), 2 mcg/kg clonidine (Group C) or normal saline (Group S) i.v. as a premedication. No other sedative premedication was given to patients.

On arrival in the operating room, multichannel monitor (Datex-ohmeda, cardiocap/5, GE healthcare, Helsinki, Finland) with the facility to measure pulse oximeter, noninvasive blood pressure, electrocardiogram, temperature and respiratory gas monitor were attached and patient's baseline heart rate (HR), mean arterial pressure (MAP), SpO₂ and respiratory rate were recorded after 5 min. Topical proparacaine hydrochloride 0.5% eye drop was applied to the cornea in each eye and IOP was measured with a, Schioetz tonometer (made in Germany) by an ophthalmologist who was unaware of the nature of the study (baseline value).

Study solutions of dexmedetomidine, clonidine and normal saline in 20 ml syringes were prepared by anesthesiologist, who was not involved in data recording. Dexmedetomidine (Dextomid, Neon Laboratories) was prepared by diluting 50 mcg dexmedetomidine in 20 ml of normal saline to make a concentration of 2.5 mcg/ml. Clonidine (Cloneon, Neon Laboratories) was prepared by diluting 200 mcg clonidine in 20 ml normal saline to make a concentration of 10 mcg/ml. In all three groups, 0.2 ml/kg of study solution was administered as a single i.v. bolus over 10 min before induction of anesthesia using a syringe pump

(SP102, Larsen and toubro Ltd). Patients in Group D received dexmedetomidine 0.5 mcg/kg, Group C received clonidine 2 mcg/kg, and Group S received same amount of normal saline.

General anesthesia was standardized in all the groups. Anesthesia was induced with injection thiopental 5 mg/kg and injection suxamethonium was administered at a dose of 1.5 mg/kg to facilitate laryngoscopy and intubation. After cessation of fasciculation laryngoscopy was performed with a Macintosh laryngoscope and trachea was intubated with appropriate number endotracheal tube. The patient was excluded from the study if the trachea could not be intubated at the first attempt. After intubation anesthesia was maintained with N₂O in O₂ (60:40), isoflurane (1%), fentanyl (1 mcg/kg) and vecuronium bromide. All the patients were ventilated mechanically (by Anesthesia workstation: Datex-ohmeda 9100c, serial no-ME14010022, GE healthcare) to maintain the end-tidal carbon dioxide partial pressure between 32 and 35 mmHg. At the end of anesthesia, the neuromuscular blockade was antagonized with neostigmine and glycopyrrolate. Patients were extubated and observed in the postoperative room.

Intraocular pressure, HR, MAP were measured and recorded before premedication (baseline), after 10 min infusion of study drug (after premedication), 30 s after injection of suxamethonium, immediately after intubation and 5 min after intubation.

Statistical analysis

The decision to include 20 patients in each group was based on a power analysis ($\alpha = 0.05$, $\beta = 0.2$), which revealed that at least 20 patients should be included in each group. Difference between the groups in the demographic data and baseline values were analyzed using unpaired *t*-test. For comparison of different observations within and between the groups, data were analyzed by repeated measures analysis of variance. Analysis was performed using software IBM Corp (2011), IBM SPSS statistics for windows, version 20.0 (Armonk, NY). Data were presented as mean \pm standard deviation. A $P < 0.05$ was considered statistically significant.

RESULTS

There were no significant differences among the groups regarding age, weight, ASA physical status of the patients [Table 1]. Baseline HR, MAP, and IOP of the different groups were also comparable.

Significant decrease in IOP was observed in Group D and C following infusion of study drug ($P < 0.001$) compared

with baseline. Following suxamethonium IOP increased in all the three Groups (D, C, S), but it was below baseline in Group D and C and increased significantly from the baseline value ($P < 0.001$) in Group S. After intubation IOP increased in all groups but it was below baseline in Group D, unlike that in Group C and Group S ($P < 0.001$) where it was significantly higher than baseline. IOP was more than the baseline in the Group S even after 5 min following intubation ($P = 0.002$). After intubation IOP in Group, D was lower significantly from Group C and S ($P < 0.001$). In Group D IOP was not higher than the basal value at all the times [Figure 1 and Table 2].

After premedication, decrease in HR and MAP was observed in Group D and C while significant increase in HR and MAP from baseline was recorded following intubation in Group C and S ($P < 0.001$). HR and MAP increased significantly after intubation in Group C and S when compared with Group D (HR and MAP: $P < 0.001$ for Group C and S) [Figures 2 and 3].

DISCUSSION

Suxamethonium is widely used drug in a rapid sequence induction of anesthesia. The use of suxamethonium is limited in the case of perforating eye injury because it increases IOP. There is an increase in IOP about 8 mm Hg from the basal value (10-21.7) after administration of suxamethonium.^[9] There is further increase in IOP due to laryngoscopy and intubation.^[10]

During anesthesia, a rise in IOP can produce permanent visual loss. IOP becomes atmospheric once the eye cavity has been entered, and any sudden rise in pressure may lead

to prolapse of the iris and lens, and loss of vitreous. Thus, proper control of IOP is critical.^[9] Many methods have been advocated to prevent the rise in IOP with limited success. Various studies have focused on the effects of alpha-2 adrenergic agonist in preventing IOP rise. The results obtained from these studies indicate a favorable effect of using alpha-2 agonists dexmedetomidine and clonidine to prevent the IOP increase following the injection of suxamethonium and tracheal intubation.^[11-13]

Dexmedetomidine is a highly selective alpha-2 adrenergic agonist. After a single i.v. dose of dexmedetomidine (0.6 mcg/kg) there was a 34% reduction in IOP.^[14] Mowafi *et al.* have reported attenuation of the increase in the IOP, HR and arterial pressure by a bolus injection of 0.6 mcg/kg dexmedetomidine as a premedication over 10 min before

Table 1: Demographic profile

Demographic profile	Group D (mean ± SD)	Group C (mean ± SD)	Group S (mean ± SD)
Age (years)	33.60±11.00	33±11.71	36.70±10.46
Weight (kg)	57.30±9.78	58.15±9.01	59.45±10.08
ASA (I:II)	17:3	17:3	16:4

$P > 0.05$; ASA: American society of anesthesiologists; SD: Standard deviation

Table 2: Changes in IOP (mmHg)

IOP	Group D (mean ± SD)	Group C (mean ± SD)	Group S (mean ± SD)
T1	15.59±1.89	15.88±2.53	14.92±2.29
T2	11.61±1.47*	12.88±2.27*	14.79±2.25
T3	12.73±1.89*	13.55±2.14*	18.24±2.43*
T4	14.68±1.90**	19.11±3.61*†	21.93±2.93*†
T5	12.60±1.69*	14.41±3.41*	15.88±2.58**

SD: Standard deviation; IOP: Intraocular pressure (mmHg); * $P < 0.001$ and ** $P < 0.05$ in comparison with T1; † $P < 0.001$ versus Group D

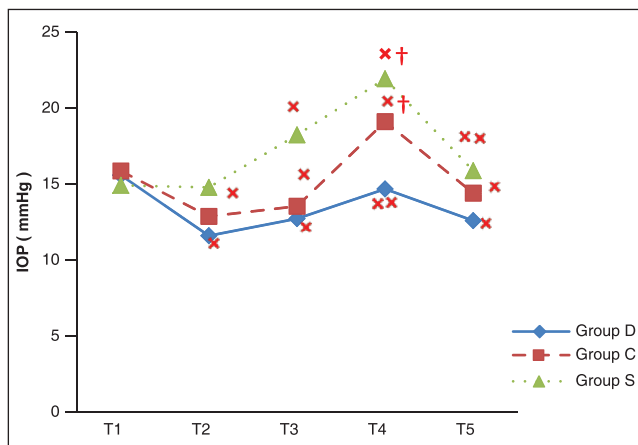


Figure 1: Changes in intraocular pressure in Groups D, C and S. Measurements were recorded before premedication (T1), after premedication (T2), 30 s after suxamethonium (T3), after intubation (T4) and 5 min after intubation (T5). * $P < 0.001$ and ** $P < 0.05$ in comparison with T1, † $P < 0.001$ versus Group D

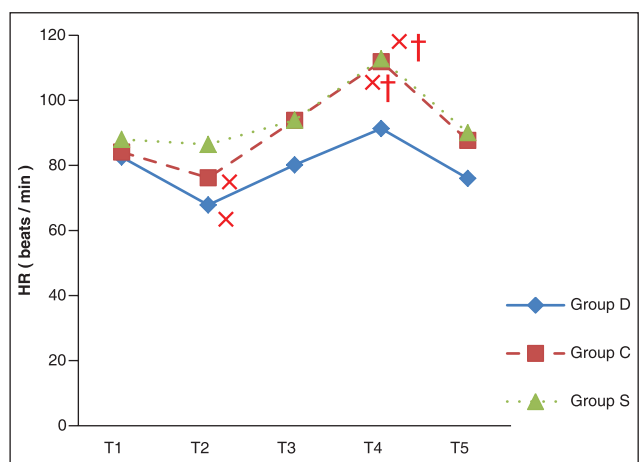


Figure 2: Changes in heart rate in Groups D, C and S. Measurements were recorded before premedication (T1), after premedication (T2), 30 s after suxamethonium (T3), after intubation (T4) and 5 min after intubation (T5). * $P < 0.001$ in comparison with T1, † $P < 0.001$ versus Group D

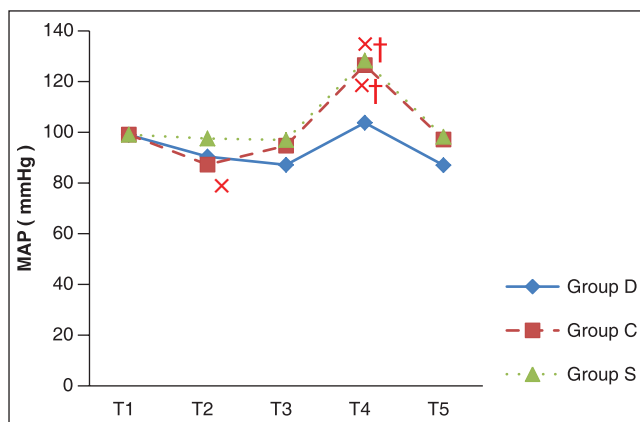


Figure 3: Changes in mean arterial pressure in Groups D, C and S. Measurements were recorded before premedication (T1), after premedication (T2), 30 s after suxamethonium (T3), after intubation (T4) and 5 min after intubation (T5). * $P < 0.001$ in comparison with T1, † $P < 0.001$ versus Group D

induction of anesthesia.^[7] Pal *et al.* in a study on 66 patients showed that the dexmedetomidine (0.6 mcg/kg as well as 0.4 mcg/kg) effectively prevents rise of IOP following suxamethonium and intubation. But hemodynamic stability is better with a lower dose (0.4 mcg/kg).^[15]

As with dexmedetomidine, pretreatment with clonidine attenuates responses to tracheal intubation, decreases IOP, and increases the likelihood of hypotension. Ghignone *et al.* studied the effect of oral clonidine and found that it effectively blunts the IOP and hemodynamic responses to laryngoscopy and intubation following injection of suxamethonium.^[16] Weigert *et al.* reported pronounced drop in IOP and MAP after administration of 0.2 mcg/kg/min clonidine i.v. over 10 min.^[17] Similar effects were reported by Lemes *et al.* by using i.v. clonidine (2.5 mcg/kg) during cataract surgery.^[18]

None of the previous workers compared the effect of using i.v. dexmedetomidine and i.v. clonidine premedication over IOP during general anesthesia. There is a risk of hypotension and bradycardia when higher dose of clonidine and dexmedetomidine are used. So we preferred single — bolus low dose for both drugs in our study.

In our study reduction in IOP was noticed following administration of dexmedetomidine and clonidine (Group D and C). IOP was increased after administration of suxamethonium in all three groups but in dexmedetomidine and clonidine groups this increase was up to the baseline value whereas in saline group IOP increase was significantly more than the baseline. Also, after laryngoscopy and intubation IOP was increased in all three groups but only in the dexmedetomidine group it still remained less than the baseline value whereas in clonidine and saline group it was significantly more than the baseline. We also

observed significant attenuation of pressure response to laryngoscopy and intubation using dexmedetomidine (0.5 mcg/kg) whereas clonidine (2 mcg/kg) was shown to be ineffective in attenuation of pressure response at this dose.^[19,20]

Thus, single i.v. dose of dexmedetomidine premedication (0.5 mcg/kg) blunt the IOP and hemodynamic response to suxamethonium injection and tracheal intubation. However, i.v. clonidine premedication, with the dose of 2 mcg/kg used in this study was shown to be effective in preventing rise of IOP after suxamethonium only but was ineffective in the attenuation of IOP and hemodynamic responses to laryngoscopy and tracheal intubation.

We conclude that i.v. dexmedetomidine is more effective than i.v. clonidine in attenuating the IOP and hemodynamic response to laryngoscopy and tracheal intubation. Therefore, dexmedetomidine should be used as premedication before rapid sequence induction of anesthesia where raised IOP is dangerous for the patients. In order to further evaluate the effects of i.v. clonidine over IOP during general anesthesia, additional studies should be planned to assess the optimum dose, mode and delivery timing of this drug.

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