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Original Research Article

Comparative Analysis of Dexmedetomidine and Lignocaine for Attenuation of Hemodynamic Response during Laryngoscopy and Intubation

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Abstract

Background and Aim: The hemodynamic response to laryngoscopy and endotracheal intubation has been reduced using a variety of pharmaceutical and non-pharmacological techniques. The goal of the current study was to examine the effectiveness of Lignocaine (1 mg/kg), Dexmedetomidine (0.5 mcg/kg), and a combination of these two low doses (Dexmedetomidine and Lignocaine) in reducing the hemodynamic response to intubation.

Material and Methods: After receiving approval from the institutional ethics committee, 150 ASA grade 1 patients ranging in age from 20 to 60 years were included for the study. mg/kg and inject 2 mcg/kg of fentanyl 10 minutes before labour. An aesthesiologist who was not engaged in the trial preloaded the study medicines into coded syringes and diluted them with normal saline to a volume of 10 ml. Dexmedetomidine 1 mcg/kg was infused over 10 minutes and normal saline over 3 minutes for Group A. Group B was given a 10 ml infusion of normal saline over 10 minutes and 1.5 mg/kg of lidocaine over 3 minutes. Dexmedetomidine 0.5 mcg/kg was infused over 10 minutes in Group C, followed by lignocaine 1 mg/kg over 3 minutes.

Results: It was found that the mean HR and BP in group DL remained below baseline value during the entire study period of 15 mins post intubation.

Conclusion: When compared to lignocaine (1.5 mg/kg) alone or a high dose of dexmedetomidine (1 mcg/kg) alone, a combination of low dose dexmedetomidine (0.5 mcg/kg) and lignocaine (1 mg/kg) efficiently reduces the pressor response during laryngoscopy and intubation without causing any hemodynamic adverse effects.

Keywords: Dexmedetomidine, Endotracheal intubation, Laryngoscopy, Lignocaine.

Introduction

Anesthesia induction procedures include laryngoscopy and endotracheal intubation. Hemodynamic reactions can happen during laryngoscopy and endotracheal intubation, which is a well-known risk [1]. The autonomic nervous system, which is heavily innervated by the larynx, pharynx, epipharynx, and trachea stimulated by laryngoscopy, causes a variety of cardiovascular changes, including an increase in heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP),

dysrhythmias, cardiac asystole, and even sudden death [2-6].

The hemodynamic response to laryngoscopy and endotracheal intubation has been reduced using a variety of pharmaceutical and non-pharmacological techniques. There are several pharmacological approaches that have been tested, including inhalational medicines, topical and intravenous local anaesthetics, calcium channel blockers, opioids, and vasodilators [7-9]. Yet, none of the aforementioned strategies or agents have shown to be perfect. Lignocaine, a local anaesthetic and class IB antiarrhythmic medication, has been used intravenously quite frequently to decrease the hemodynamic response following intubation [10]. Alpha-2 adrenergic agonists, primarily Clonidine and Dexmedetomidine, have been used for more than 20 years 6 to reduce the sympathetic response. As a 2-agonist, dexmedetomidine is eight times more selective for 2 receptors than clonidine [11]. It has analgesic effects and decreased the sympathetic activity of the central nervous system [12]. Due to its analgesic and calming effects and lack of respiratory depression, dexmedetomidine is also utilised as a sedative for supervised anaesthetic treatment [13-15].

We suggested that using these two medicines in combination could suppress the intubation response more effectively while also requiring less of each drug. The goal of the current study was to examine the effectiveness of Lignocaine (1 mg/kg), Dexmedetomidine (0.5 mcg/kg), and a combination of these two low doses (Dexmedetomidine and Lignocaine) in reducing the hemodynamic response to intubation.

Material and Methods

After receiving approval from the institutional ethics committee, 150 ASA grade 1 patients ranging in age from 20 to 60 years were included for the study. Patients with reduced autonomic control, such as the elderly, diabetics, people with chronic hypertension, or people with severe cardiac disease, people taking -blockers or calcium channel blockers, pregnant or lactating women, people with a history of allergy to egg proteins, and people who have taken medications, particularly 2 agonists, were not taken into consideration for the study.

Each patient got a full physical examination, baseline tests, and a detailed history of all current and prior ailments as part of the pre-anesthesia checkup. One of the department's senior anesthesiologists examined the patient's airways. After thoroughly explaining the procedure to the patient, a written declaration of valid informed permission was acquired. Three groups of 50 patients each, designated as Group L, Group D, and Group DL, were randomly assigned patients. The pre-operative room was used to capture the patients' baseline vital signs, which included their heart rate (HR), systolic and diastolic blood pressures, mean arterial pressure (MAP), and oxygen saturation. A 20-G venous cannula was used to anchor the i.v. line, and Ringer's lactate infusion (6 ml/kg) was administered over the course of 30 minutes to make up for the fluid deficit caused by hunger. Patients were pre-medicated inside the operating room with i.v. injections of glycopyrrolate, ondansetron, and fentanyl 10 minutes prior to induction.

An anesthesiologist who was not involved in the trial preloaded the study medicines into coded syringes and diluted them with normal saline to a volume of 10 ml.

Group A- received Dexmedetomidine 1 mcg/kg infusion over 10 mins and Normal saline (for blinding purpose) over 3 mins.

Group B- received 10 ml normal saline (for blinding purpose) infusion over 10 mins and Lignocaine 1.5 mg/kg over 3 mins.

Group C- received Dexmedetomidine 0.5 mcg/kg infusion over 10 mins followed by Lignocaine 1 mg/kg over 3 mins.

The study medicines were preloaded, diluted to a volume of 10 ml with normal saline, and then administered as coded. After study drug infusion, blood pressure and heart rate were monitored and preoxygenated for three minutes. After that, succinylcholine 2.0 mg/kg and inj. propofol 2 mg/kg were used to induce anaesthesia. After succinylcholine injection, manual breathing with 100% oxygen was performed for 90 seconds, and then a direct laryngoscopy with a Macintosh curved blade was performed (no. 3 or 4 as per need). The patients were intubated using cuffed portex endotracheal tubes of the proper size (ETT). All patients had a 45-second time constraint for laryngoscopy and intubation. by a physician who did not take part in the study, an anesthesiologist.

Laryngoscopy and intubation times were recorded using a stopwatch. Following positioning confirmation, an adhesive plaster was used to secure the ET tube. 66% N2O and 33% oxygen were used to sustain anaesthesia in 2 L of fresh gas flow on a circle absorber system. For muscular relaxation, a bolus IV dose of 0.08 mg/kg of vecuronium was administered, followed by an intermittent dose of 0.02 mg/kg. Following surgery, Neostigmine 0.05 mg/kg and Glycopyrrolate 0.008 mg/kg IV were administered to each patient. After a full recovery, patients were extubated and moved to the anaesthesia recovery room for monitoring. Vital parameters such as HR, SAP, DAP and MAP were recorded, at baseline, after study drug administration, after induction, 1, 3, 5, 7, 10 and 15 mins after intubation. No surgical intervention was allowed during this study period of 15 mins.

The hemodynamic abnormalities such a fall in mean arterial pressure (MAP) greater than 20% below the baseline value was treated with principally by pumping a bolus of IV fluid. Drop in systemic arterial pressure (SAP) less than 90 mmHg was treated with inj. Mephenteramine IV bolus, 3 mg, repeated as necessary. An injection of a bolus dosage (0.5 mg/kg) of propofol was used to treat any increase in MAP or SAP of more than 20% or SBP > 140 mmHg. Atropine 0.6 mg IV was used to treat the decrease in HR.

Statistical Analysis

The recorded data was compiled and entered in a spreadsheet computer program (Microsoft Excel 2007) and then exported to data editor page of SPSS version 15 (SPSS Inc., Chicago, Illinois, USA). For all tests, confidence level and level of significance were set at 95% and 5% respectively.

Results

In regard to the distribution of age and gender, there was no statistical difference between the groups. The mean age for group L was 32.2 ± 12.45 years, for group D it was 33.04±11.6 years, and for group DL it was 35.10±11.47 years. ($p>0.05$) among group L. Group L's mean weight of 58.3± 9.1 kg was similar to that of groups D's (58.2±8.10 kg) and group's (58.9±9.10 kg). ($p >0.05$).

In all three groups, the mean baseline HR was essentially the same. ($p >0.05$). After administration of the study drug, mean HR was steady in group L (81.78±9.23), but significantly decreased in group D (57.87±5.56) and group DL (Dexmedetomidine-Lignocaine combination). (70.78 ± 9.35). ($p<0.001$) Even after induction, the decline in HR in D and DL maintained. (group DL=71.12 ± 8.34, group L=79.24 7.26, and group D=58.03 5.48). The mean heart rate in group L considerably increased one minute following laryngoscopy and intubation. Although the HR decreased gradually once more towards the baseline, it did not reach the preinduction value until 15 minutes after the incision.

But it was consistently within the range of clinical normal. In contrast, the mean HR in group D increased from the post-induction value (58.03±5.48) to (mean 68.46 ±8.35) at 1 minute after laryngoscopy and intubation, but it stayed below the baseline value even at (81.90 ±10.34) and after 3, 5, 7, and 10 minutes. In this group, bradycardia affected 6 people. The mean HR in group DL remained below baseline value (85.24 ±12.47) during the entire study period of 15 mins post intubation and no episode of bradycardia or tachycardia was reported in this group throughout the entire study period.

In each of the three groups, the mean baseline SBP was statistically comparable. (L= 122.20 ± 09.45, D=126.2 ± 9.10, DL= 125.23 ± 8.78). Mean SBP in group L (121.10 ± 10.22) remained close to baseline after study medication administration. SBP dropped significantly in group DL (107.10± 6.22), while group D experienced the most drop. (94.09 ± 6.32). After induction a highly significant fall in mean SBP was seen in group L (from 121.10 ± 10.22 to 104.02 ± 7.22) while a moderate fall was seen in group DL, (from 107.10 ± 6.22 to 99.40 ± 4.87). Group D, however had no further decrease in SBP (from 94.09 ± 6.32 to 92.45 ± 5.44). 1 minute after laryngoscopy and intubation the mean SBP increased significantly (to 135.95 ± 7.45) in group L remained above baseline mean SBP (122.20 ± 09.45) post laryngoscopy and intubation throughout the study period of 15 minutes. The mean SBP in group D increased from the post-induction value (92.45 5.44) at 1 minute after laryngoscopy and intubation to 107.22 8.32, although it remained below the baseline value (126.2 ±9.10) even after 3, 5, 7, and 15 minutes (5.34). After 5 or 7 minutes after intubation, 4 patients in this group experienced hypotension, which was successfully managed with injections of mephenteramine (3 to 6 mg). The mean SBP in group DL increased from the post-induction value of (99.40 4.87) 1 minute after laryngoscopy and intubation, showing a tendency similar to that of group D. Moreover, SBP continued to be below the baseline value (125±8.85) for 3, 5, 7, and 10 minutes, just like in group D, although no episodes of bradycardia or hypotension were noted in this group during the course of the trial.

In all three groups, changes in mean and diastolic blood pressure (DBP) exhibited patterns resembling those in SBP. After the study medication, group D experienced the greatest decrease in MAP and DBP. While mean MAP decreased after introduction in all three groups, group L experienced the greatest mean MAP decrease. Group D experienced a small increase from the post-induction value 1 minute later but was still far below the baseline value. In group DL, MAP increased from the post-induction value. However, in group L, the mean MAP considerably jumped 1 minute after laryngoscopy and remained above the baseline mean MAP for the whole 15-minute investigation. SPO2 in each of the three groups remained constant over the course of the trial.

Table 1: Comparison of mean HR between the groups

Time	Group L E MEAN ± SD	Group D MEAN ± SD	Group DL MEAN ± SD	P value
Baseline	81.26 ± 10.12	81.90 ± 10.34	85.24 ±12.47	0.25
After drug infusion	81.78 ± 9.23	57.87 ±5.56	70.78 ± 9.35	0.03*
After induction	79.24 ± 7.26	58.03 ± 5.48	71.12 ± 8.34	0.001*
1	95.24 ± 6.30	68.46 ± 8.35	82.04 ± 8.47	0.02*
3	94.87 ± 7.65	68.4 ± 9.10	78.32 ± 8.47	0.05*

5	90.68 ± 5.47	62.50 ± 7.70	77.58 ± 8.44	0.03*
Post induction 7	90.01 ± 5.22	60.46 ± 8.65	77.22 ± 7.54	0.001*
10	88.54 ± 7.23	60.70 ± 5.87	75.22 ± 8.4	0.002*
15	84.35 ± 8.45	61.22 ± 5.34	70.46 ± 7.64	0.004*

* indicates statistically significance at p≤0.05

Table 2: Comparison of mean SBP between the groups

Time	Group L E MEAN ± SD	Group D MEAN ± SD	Group DL MEAN ± SD	P value
Baseline	122.20 ± 09.45	126.2 ± 9.10	125.23 ± 8.78	0.30
After drug infusion	121.10 ± 10.22	94.09 ± 6.32	107.10 ± 6.22	0.02*
After induction	104.02 ± 7.22	92.45 ± 5.44	99.40 ± 4.87	0.002*
1	135.95 ± 7.45	107.22 ± 8.32	116.18 ± 5.65	0.04*
3	136.9 ± 10.08	104.89 ± 9.23	116.95 ± 5.47	0.06*
5	133.23 ± 8.48	102.21 ± 8.45	114.78 ± 5.78	0.01*
7	129.4 ± 6.64	98.4 ± 6.22	114.90 ± 5.47	0.006*
10	128.79 ± 8.19	99.04 ± 4.5	111.98 ± 4.47	0.005*
15	125.50 ± 7.22	98.10 ± 5.32	109.47 ± 5.46	0.003*

Discussion

A very selective 2 adrenergic agonist is dexmedetomidine. Three different types of 2 receptors—2 A, 2 B, and 2 C—located in the brain and spinal cord are how it works. Sedation, anxiolysis, analgesia, and sympatholysis are the outcomes, with the latter causing bradycardia and hypotension. The brain stem vasomotor center's 2 A receptors are activated, which suppresses the release of norepinephrine and causes bradycardia, hypotension, and hypotension. Sedation results from stimulation of 2A and 2C in the locus ceruleus.

By decreasing the release of substance P, activation of the 2 A and 2 C receptors in the spinal cord directly reduces pain transmission. These pharmacological characteristics have been examined in the past to gauge their impact on patients undergoing laparoscopic surgery in terms of hemodynamic responses. The drug has been administered intravenously both with and without a bolus dosage. Studies have been done on infusion rates ranging from 0.1 to 10 mcg/kg/h [13–15].

According to Kumari and colleagues (2015) [16], dexmedetomidine failed to entirely obtund the hemodynamic response to laryngoscopy and intubation but did reduce the greatest increase in heart rate after intubation by 19.6% in the Dexmedetomidine 0.5 mcg/kg group compared to the placebo group. In contrast, Zhan, Guan, *et al*. [17] discovered that 1 mcg/kg Dexmedetomidine greatly controlled the cardiovascular reactions associated with tracheal intubation, but that it also caused a considerable drop in arterial pressure 5 minutes after intubation. After administering 1 g/kg of dexmedetomidine within 10 minutes of induction, Unlugenc *et al*. [9] saw a significant drop in HR. Also, just like in our study, N. Solanki and colleagues [18-19] mention bradycardia in 2 patients treated with dexmedetomidine (1 mcg/kg). Similar results from our investigation in Group D have been noticed. So, it may be concluded that while modest doses of these drugs may not be entirely beneficial, excessive amounts may be linked to substantial negative effects. This gives justification for mixing these

drugs in small amounts to complete the task with the fewest side effects possible. The effectiveness of the combination of dexmedetomidine (0.25 mg/kg) and lignocaine (1.0 mg/kg) in suppressing the hemodynamic and catecholamine responses during tracheal extubation in sixty hypertensive patients was compared by Moustafa A. *et al*. in a study published in Moustafa A. *et al*. [20]. They discovered that patients receiving the dexmedetomidine-lidocaine combination had considerably lower heart rates, mean arterial pressures, and rate-pressure products after tracheal extubation than patients receiving either dexmedetomidine or lidocaine alone. The efficiency of dexmedetomidine (0.5 g/kg) and intravenous clonidine (3 g/kg) in lowering blood pressure brought on by laryngoscopy and intubation was compared by Sarkar *et al*. Findings indicated that dexmedetomidine and clonidine were useful in this aspect. Both groups' postintubation SBP was lower, but the dexmedetomidine group's HR was greater. [21] In contrast, the dexmedetomidine group in the current study had a lower HR. The impact of intravenous dexmedetomidine on hemodynamic responses to laryngoscopy and intubation was studied by Rashmi and Komala in 2015 [22], Laha *et al*. in 2011 [23], and Kumari *et al*. in 2008-2009 [24]. In these three investigations, dexmedetomidine was successful in reducing the hemodynamic response to laryngoscopy and intubation since HR, DBR, SBP, and MAP decreased in the dexmedetomidine group. Also, due to the specificity of dexmedetomidine, patients frequently experience substantial (>20%) hemodynamic alterations after receiving an injection of this medication. This was also acknowledged in 2019 by Shu and his coworkers [25].

As direct laryngoscopy was a part of our investigation, we kept the lignocaine dosage a little higher, at 0.5 mcg/kg. Many studies indicate that lignocaine may not be sufficient to completely suppress the intubation reaction. To reduce the intubation response, a combination of lignocaine and opioid 17 or doses greater than 1.5 mg/kg are required. The same investigations indicated that a dose of 0.50 mcg/kg of dexmedetomidine had little cardiovascular side effects but was insufficient to stop the tracheal intubation-evoked hemodynamic response. [16,17]

RPP was determined to be within a safer range in Group DL in our study as well; nevertheless, it was found to be elevated to its maximum in Group L and to be too low in Group D. The combination of modest dosages of Lignocaine with Dexmedetomidine provides improved hemodynamic stability, which is seen in this.

Conclusion

When compared to lignocaine (1.5 mg/kg) alone or a high dose of dexmedetomidine (1 mcg/kg) alone, a combination of low dose dexmedetomidine (0.5 mcg/kg) and lignocaine (1 mg/kg) efficiently reduces the pressor response during laryngoscopy and intubation without causing any hemodynamic adverse effects.

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