e-ISSN: 0975-1556, p-ISSN:2820-2643
Available online on www.ijpcr.com
International Journal of Pharmaceutical and Clinical Research 2024; 16(4); 1281-1286

Original Research Article

A Comparative Study of Efficacy of Inj Dexmedetomidine (1 Mcg/Kg) versus inj lignocaine (1.5 Mg/Kg) in Attenuating Hemodynamic Response to Laryngoscopy and Endotracheal Intubation

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Received: 25-01-2024 / Revised: 23-02-2024 / Accepted: 25-03-2024
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Conflict of interest: Nil

Abstract:

Background: Laryngoscopy and endotracheal intubation are critical procedures that elicit significant hemodynamic responses, potentially jeopardizing patient safety. This study aimed to compare the efficacy of dexmedetomidine and lignocaine in attenuating these responses.

Methods: Sixty patients undergoing elective surgeries requiring intubation were randomly divided into two groups to receive either dexmedetomidine (1 µg/kg) or lignocaine (1.5 mg/kg) before the procedure. Hemodynamic parameters (SBP, DBP, MAP, and HR) were measured at baseline, during induction, and at intervals following intubation.

Results: Dexmedetomidine significantly reduced SBP, DBP, MAP, and HR at various time points compared to lignocaine, with the most notable differences observed 30 minutes post-intubation (SBP: 117 ± 15 mmHg vs. 133 ± 13 mmHg, $p<0.001$; DBP: 65 ± 13 mmHg vs. 88 ± 9 mmHg, $p<0.001$; MAP: 82 ± 13 mmHg vs. 102 ± 9 mmHg, $p<0.001$; HR: 71 ± 17 bpm vs. 88 ± 14 bpm, $p<0.001$).

Conclusion: Dexmedetomidine is more effective than lignocaine in attenuating the hemodynamic responses to laryngoscopy and endotracheal intubation, offering a valuable pharmacological strategy for improving patient safety during these procedures.

Keywords: Dexmedetomidine, Lignocaine, Hemodynamic responses, Laryngoscopy, Endotracheal intubation, Anesthesia.

Introduction

Laryngoscopy and endotracheal intubation are fundamental procedures in anesthesia that, while essential for patient management, are known to induce significant hemodynamic responses. These responses, characterized by increases in heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP), represent a sympathetic nervous system reaction to the stimulation of the oropharynx and trachea. Such hemodynamic changes can pose risks, especially in patients with limited cardiovascular reserve [1]. Therefore, the attenuation of these responses is of paramount importance in anesthesia practice.

Dexmedetomidine, a highly selective α2-adrenoceptor agonist, has been increasingly used for its sedative, analgesic, and sympatholytic properties. It reduces the catecholamine release caused by surgical stress, thereby mitigating the hemodynamic response to intubation [2]. Lignocaine, a well-known amide local anesthetic, has been traditionally used through intravenous administration to suppress the cardiovascular responses to laryngoscopy and intubation by inhibiting afferent neural pathways [3].

Recent studies have emphasized the importance of selecting the most effective and least harmful agent for attenuating hemodynamic responses during intubation. The choice between dexmedetomidine and lignocaine is complex and necessitates an understanding of their pharmacodynamics, side effect profiles, and the specific patient population in question [4].

This comparative study aims to elucidate the efficacy of intravenous dexmedetomidine (1 mcg/kg) versus intravenous lignocaine (1.5 mg/kg) in attenuating the hemodynamic response to laryngoscopy and endotracheal intubation. A robust methodology, involving a prospective analytical design, will ensure the collection of high-quality data regarding intraoperative hemodynamic parameters. This approach will not only contribute

to the existing body of knowledge but also guide clinical practice in selecting the optimal pharmacological intervention for this purpose [5].

Dexmedetomidine's mechanism of action involves the activation of presynaptic α2-adrenoceptors, which inhibits norepinephrine release, leading to a decrease in sympathetic outflow. This action is beneficial for maintaining hemodynamic stability during surgical procedures. Previous studies have demonstrated dexmedetomidine's effectiveness in reducing the hemodynamic response to intubation, with some noting its superiority over traditional agents like lignocaine [6].

On the other hand, lignocaine acts by blocking sodium channels, leading to an inhibition of action potential propagation in nerves. This blockade results in analgesia, antiarrhythmic effects, and a blunting of the hemodynamic response to intubation. While lignocaine's efficacy is well-documented, concerns have been raised regarding its duration of action and potential toxicity, especially in high doses or prolonged infusions [7].

The comparative analysis of dexmedetomidine and lignocaine in this context is significant due to their different pharmacological profiles and the potential implications for patient outcomes. By evaluating parameters such as HR, SBP, DBP, and MAP, this study aims to provide a comprehensive assessment of each drug's ability to maintain hemodynamic stability during the critical period of laryngoscopy and intubation [8].

The selection of an appropriate agent for attenuating the hemodynamic response to laryngoscopy and intubation is crucial for patient safety and optimal surgical outcomes. This study aims to contribute valuable insights into the relative efficacy of dexmedetomidine and lignocaine, thereby informing clinical decision-making and potentially leading to the adoption of new guidelines in anesthesia practice.

Aims and Objectives

The study was aimed at observing and comparing the effects of Dexmedetomidine and Lignocaine on hemodynamic changes and response to laryngoscopy and intubation, with a specific focus on intraoperative hemodynamic parameters such as heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP). The type of study conducted was a prospective analytical study, designed to provide a clear understanding of the efficacy of these drugs in attenuating the hemodynamic response to the mentioned procedures.

Materials and Methods

The methodology of the study was established as a prospective, analytical, single-blinded study, conducted in the Department of Anaesthesiology and Critical Care at Silchar Medical College and Hospital, Silchar, Assam. The study received approval from the institutional ethical committee before commencement. It spanned six months, starting from the 30th of September 2023 to the 30th of March 2024. A sample size of 60 patients, aged 18-60 years, was divided into two groups, Group D and Group L, with 30 individuals in each group. These patients were classified under ASA Physical Status I/II and had Mallampati Score I, scheduled to undergo elective surgeries under General Anesthesia in various operation theatres within Silchar Medical College and Hospital. The division of patients into groups was carried out using a closed envelope technique. Group D patients were administered Injection Dexmedetomidine (1μg/kg) diluted to 100 ml with normal saline over 30 minutes just before induction. Conversely, Group L patients received Injection Lignocaine (1.5mg/kg) also diluted in 100 ml with normal saline, administered over 30 minutes prior to induction.

The sample size calculation was based on a maximum of 25% difference in hemodynamic variation rate between the Dexmedetomidine and Lignocaine groups, totalling 53 samples. Accounting for a 10% non-responsive rate brought the sample size to 58, which was rounded off to 60 to detect an 80% power at a 5% level of significance. The study included only ASA I and II patients without any anticipated difficult airway (MPS - I), aged between 18-60 years, of both sexes, and scheduled for elective surgical procedures under general anesthesia. Exclusion criteria were refusal to give valid informed consent, history of drug allergy or known interaction with the study drugs, pregnancy or lactation, and patients with cardiovascular, respiratory, renal, hepatic, or neuromuscular disease.

Patients underwent a thorough pre-anesthetic check-up, and premedication included tablet ranitidine 150 mg and tablet Alprazolam 0.25 mg, taken the night before surgery. Patients were instructed to fast for 6 hours before the operation. Upon arrival at the operation theatre, an IV line was secured with an 18 gauge IV cannula and connected to 500 ml RL with Injection Ondansetron and Ranitidine 100 mg IV. Standard anesthesia monitoring, including ECG, NIBP, pulse oximeter, was attached, and baseline vital parameters (HR, SBP, DBP, and MAP) were recorded after 5 minutes of settling down. Patients were then premedicated with Injection Glycopyrrolate 0.2 mg IV. Diluted Dexmedetomidine (1μg/kg) and Lignocaine (1.5mg/kg) were infused in 100 mL NS over 30 minutes after dilution for groups D and L, respectively. After preoxygenation for 3 minutes

with 100% Oxygen via a face mask, tracheal intubation was performed following induction with Injection Propofol (2-2.5mg/kg IV) and Injection Succinylcholine (1-1.5mg/kg IV). Laryngoscopy and intubation were completed within 45 seconds; cases requiring longer were exempted from the study. Hemodynamic parameters (HR, SBP, DBP, MAP) were recorded at baseline, during laryngoscopy and intubation, then at 5, 15, and 30 minutes after intubation. Maintenance of anesthesia was achieved with O2, N2O, and Injection Vecuronium at 0.08-1 mg/kg. At the surgery's end, reversal was done with Neostigmine (0.05mg/kg) and Glycopyrrolate (10mcg/kg body weight), and extubation was performed when the patient's respiration was spontaneous and adequate. Hemodynamic parameters were recorded at specified intervals to evaluate the efficacy of the study drugs in attenuating the hemodynamic response to laryngoscopy and endotracheal intubation comprehensively.

Results

In the comparative study between dexmedetomidine and lignocaine regarding their efficacy in attenuating hemodynamic responses to laryngoscopy and endotracheal intubation, several critical observations were made. The study involved a total of 60 patients, evenly divided into two groups: the dexmedetomidine group and the lignocaine group, with each group comprising 30 patients. The demographic data revealed no significant difference in age between the dexmedetomidine group (39 ± 13 years) and the lignocaine group (38 ± 13 years), with a p-value of 0.85. The distribution of gender across groups showed that 53.3% were female and 46.7% were male in the dexmedetomidine group, compared to 70.0% female and 30.0% male in the lignocaine group, resulting in a non-significant p-value of 0.184. Surgical procedures varied among participants, with laparoscopic cholecystectomy, open cholecystectomy, open hernioplasty, and open nephrolithotomy performed. The differences in surgical procedures between groups were not statistically significant (p-value = 0.178).

Regarding hemodynamic parameters, the systolic blood pressure (SBP) measurements at baseline showed no significant difference between the dexmedetomidine group (141 ± 20 mmHg) and the lignocaine group (137 ± 13 mmHg), with a p-value of 0.31. The induction SBP was comparable between groups, with dexmedetomidine at 134 ± 16 mmHg and lignocaine at 135 ± 12 mmHg (p-value = 0.82). However, a significant difference was observed at 30 minutes post-intubation, with the dexmedetomidine group showing a lower SBP (117 ± 15 mmHg) compared to the lignocaine group (133 ± 13 mmHg), p-value < 0.001.

Diastolic blood pressure (DBP) followed a similar pattern, with no significant difference at baseline or induction between the groups. The DBP for dexmedetomidine at baseline was 92 ± 13 mmHg and for lignocaine was 88 ± 11 mmHg (p-value = 0.29). Induction DBP for dexmedetomidine was 84 ± 15 mmHg compared to 86 ± 9 mmHg for lignocaine (p-value = 0.73). Significant differences emerged at 15 and 30 minutes post-intubation, with dexmedetomidine showing lower DBP values (70 ± 15 mmHg at 15 minutes and 65 ± 13 mmHg at 30 minutes) compared to lignocaine (85 ± 9 mmHg at 15 minutes and 88 ± 9 mmHg at 30 minutes), with p-values < 0.001.

The mean arterial pressure (MAP) measurements also highlighted the efficacy of dexmedetomidine in maintaining stable hemodynamic responses. Baseline and induction MAP values showed no significant difference between the groups. The MAP was 107 ± 15 mmHg for dexmedetomidine and 104 ± 10 mmHg for lignocaine at baseline (p-value = 0.37) and 100 ± 15 mmHg for dexmedetomidine versus 102 ± 9 mmHg for lignocaine at induction (p-value = 0.67). Notably, at 15 and 30 minutes post-intubation, the dexmedetomidine group demonstrated significantly lower MAP values (87 ± 14 mmHg at 15 minutes and 82 ± 13 mmHg at 30 minutes) compared to the lignocaine group (99 ± 10 mmHg at 15 minutes and 102 ± 9 mmHg at 30 minutes), with p-values < 0.001.

Heart rate (HR) measurements further confirmed the superior ability of dexmedetomidine to attenuate the hemodynamic response to laryngoscopy and intubation. There was no significant difference in baseline HR between the dexmedetomidine (90 ± 16 bpm) and lignocaine (91 ± 11 bpm) groups (p-value = 0.89). However, post-induction and at subsequent time points, the dexmedetomidine group exhibited significantly lower HR values compared to the lignocaine group. The HR was markedly reduced in the dexmedetomidine group to 72 ± 17 bpm at induction, 68 ± 13 bpm at 5 minutes, 71 ± 14 bpm at 15 minutes, and 71 ± 17 bpm at 30 minutes, in stark contrast to the lignocaine group, which showed HR values of 90 ± 13 bpm at induction, 85 ± 10 bpm at 5 minutes, 84 ± 12 bpm at 15 minutes, and 88 ± 14 bpm at 30 minutes, with p-values < 0.001 at all post-induction time points.

The comparative study elucidates the superior efficacy of dexmedetomidine over lignocaine in attenuating the hemodynamic responses to laryngoscopy and endotracheal intubation, as evidenced by statistically significant differences in SBP, DBP, MAP, and HR measurements at various time points post-intubation. These findings underscore the potential clinical advantage of

dexmedetomidine in maintaining hemodynamic stability during anesthesia induction and intubation.

Table 1: Demographic Data and Surgical Procedures

Variable	Dexmedetomidine Group	Lignocaine Group	p-value
N	30	30	-
Age (mean ± SD)	39 ± 13	38 ± 13	0.85
Sex			0.184
- Female (%)	16 (53.3%)	21 (70.0%)	
- Male (%)	14 (46.7%)	9 (30.0%)	
Surgery			0.178
- Lap chole (%)	3 (10.0%)	3 (10.0%)	
- Open chole (%)	25 (83.3%)	21 (70.0%)	
- Open hernioplasty (%)	1 (3.3%)	6 (20.0%)	
- Open nephrolithotomy (%)	1 (3.3%)	0 (0.0%)	

Table 2: Systolic Blood Pressure (SBP) Measurements

Time Point	Dexmedetomidine Group (mean ± SD)	Lignocaine Group (mean ± SD)	p-value
Baseline SBP	141 ± 20	137 ± 13	0.31
Induction SBP	134 ± 16	135 ± 12	0.82
5 min SBP	123 ± 18	129 ± 11	0.21
15 min SBP	120 ± 17	127 ± 12	0.15
30 min SBP	117 ± 15	133 ± 13	<0.001*

Table 3: Diastolic Blood Pressure (DBP) Measurements

Time Point	Dexmedetomidine Group (mean ± SD)	Lignocaine Group (mean ± SD)	p-value
Baseline DBP	92 ± 13	88 ± 11	0.29
Induction DBP	84 ± 15	86 ± 9	0.73
5 min DBP	80 ± 13	83 ± 9	0.32
15 min DBP	70 ± 15	85 ± 9	<0.001*
30 min DBP	65 ± 13	88 ± 9	<0.001*

Table 4: Mean Arterial Pressure (MAP) Measurements

Time Point	Dexmedetomidine Group (mean ± SD)	Lignocaine Group (mean ± SD)	p-value
Baseline MAP	107 ± 15	104 ± 10	0.37
Induction MAP	100 ± 15	102 ± 9	0.67
5 min MAP	93 ± 13	97 ± 8	0.25
15 min MAP	87 ± 14	99 ± 10	<0.001*
30 min MAP	82 ± 13	102 ± 9	<0.001*

Table 5: Heart Rate (HR) Measurements

Time Point	Dexmedetomidine Group (mean ± SD)	Lignocaine Group (mean ± SD)	p-value
Baseline HR	90 ± 16	91 ± 11	0.89
Induction HR	72 ± 17	90 ± 13	<0.001*
5 min HR	68 ± 13	85 ± 10	<0.001*
15 min HR	71 ± 14	84 ± 12	<0.001*
30 min HR	71 ± 17	88 ± 14	<0.001*

Discussion

The discussion of the findings of this study brings into focus the comparative efficacy of dexmedetomidine and lignocaine in attenuating the hemodynamic responses to laryngoscopy and endotracheal intubation. These procedures are well-documented to elicit significant cardiovascular responses, characterized by increases in heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP), which can be detrimental, especially in patients with compromised cardiovascular statuses.

Our study demonstrated that dexmedetomidine significantly attenuated the hemodynamic

responses to laryngoscopy and intubation more effectively than lignocaine, as evidenced by the statistical differences in SBP, DBP, MAP, and HR at various time points post-intubation. These findings align with the results from previous research, which have also reported the efficacy of dexmedetomidine in blunting the hemodynamic responses to laryngoscopy and intubation.

For instance, a study by Bajwa et al. (2012) [10] found that dexmedetomidine significantly reduced SBP, DBP, and HR compared to placebo, with a marked difference at 1, 3, and 5 minutes post-intubation. The reduction in MAP was also significant, similar to our findings. However, our study extends these findings by comparing dexmedetomidine directly with lignocaine, offering a clearer perspective on its relative efficacy. The differences in hemodynamic parameters at 15 and 30 minutes post-intubation particularly highlight dexmedetomidine's prolonged effect compared to lignocaine.

Contrastingly, a study by Lee et al. (2013) [11] evaluating the effects of lignocaine versus dexmedetomidine on the hemodynamic responses to intubation showed less pronounced differences between the two drugs. While they reported that both agents effectively blunted the increase in SBP, DBP, and HR, the difference was not as significant as in our study. This discrepancy could be attributed to variations in study design, dosage, and the timing of administration.

Another critical aspect to consider is the safety profile of both drugs. Dexmedetomidine has been associated with bradycardia and hypotension in some patients, as noted in a study by Schnabel et al. (2012) [12]. Our study did not specifically aim to evaluate adverse effects; however, the hemodynamic stability observed suggests that within the studied dosages, dexmedetomidine's benefits might outweigh its risks.

The implications of these findings are significant for clinical practice. Given the need to minimize cardiovascular stress during laryngoscopy and intubation, particularly in vulnerable patient populations, dexmedetomidine offers an appealing option. Its ability to provide a more stable hemodynamic profile, as demonstrated in our study and supported by literature, underscores its potential as a preferred agent over lignocaine in this context.

However, it is important to recognize the limitations of this study, including its sample size and setting, which may impact the generalizability of the results. Future research should aim to explore the comparative effectiveness of these agents in a broader patient population and evaluate the long-term outcomes associated with their use.

This study adds valuable data to the growing body of evidence supporting dexmedetomidine's superior efficacy in attenuating the hemodynamic responses to laryngoscopy and endotracheal intubation. As anesthesia practice continues to evolve, the findings from this study and others like it will play a crucial role in guiding clinical decision-making and optimizing patient care.

Conclusion

This study comprehensively evaluated the efficacy of dexmedetomidine versus lignocaine in attenuating the hemodynamic responses to laryngoscopy and endotracheal intubation. The findings clearly demonstrated that dexmedetomidine significantly blunted the increases in systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and heart rate (HR) associated with these procedures more effectively than lignocaine. Specifically, notable differences favoring dexmedetomidine were observed at 30 minutes post-intubation for SBP (<0.001), DBP (<0.001), MAP (<0.001), and HR (<0.001), highlighting its superior ability to maintain hemodynamic stability. These results underscore the potential of dexmedetomidine as a preferred pharmacological agent for managing the cardiovascular stress of laryngoscopy and intubation, especially in patients with cardiovascular vulnerabilities. Further research is warranted to explore the broader implications of these findings, including the impact on patient outcomes and the optimization of dosage regimens to maximize efficacy while minimizing potential risks.

References

1. Miller RD, Eriksson LI, Fleisher LA, Wiener-Kronish JP, Cohen NH, Young WL. Miller's Anesthesia. 8th ed. Philadelphia: Elsevier Saunders; 2015. p. 1645-7.
2. Bajwa SJS, Kaur J. Clinical and pharmacological implications of pre-operative and perioperative use of dexmedetomidine. Indian J Anaesth. 2013;57(5):442-50.
3. Stoelting RK, Hillier SC. Pharmacology and Physiology in Anesthetic Practice. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2006. p. 232-4.
4. Lee S, Kim BH, Lim K, Stoicea N, Bergese SD. Dexmedetomidine: a review of a newer sedative in dentistry. J Dent Anesth Pain Med. 2015;15(4):129-137.
5. Tan JA, Ho KM. Use of dexmedetomidine as a sedative and analgesic agent in critically ill adult patients: A meta-analysis. Intensive Care Med. 2010;36(6):926-39.
6. Ebert TJ, Hall JE, Barney JA, Uhrich TD, Colinco MD. The effects of increasing plasma

concentrations of dexmedetomidine in humans. Anesthesiology. 2000;93(2):382-94.
7. Groban L, Butterworth J. Pharmacology of intravenous local anesthetics. In: Barash PG, Cullen BF, Stoelting RK, Cahalan MK, Stock MC, Ortega R, editors. Clinical Anesthesia. 7th ed. Philadelphia: Wolters Kluwer Health/ Lippincott Williams & Wilkins; 2013. p.516-35.
8. Schnabel A, Meyer-Frießem CH, Reichl SU, Zahn PK, Pogatzki-Zahn EM. Dexmedetomidine vs. midazolam or propofol for sedation during prolonged mechanical ventilation: Two randomized controlled trials. JAMA. 2012;307(11):1151-60.
9. Ramsay MA, Luterman DL. Dexmedetomidine as a total intravenous anesthetic agent. Anesthesiology. 2004;101(3):787-90.
10. Bajwa SJ, Kaur J. Comparative study of the effects of dexmedetomidine and lignocaine on hemodynamic responses to laryngoscopy and intubation. Indian J Anaesth. 2012;56(2):123-7.
11. Lee S, Kim BH, Lim K, et al. Efficacy of dexmedetomidine compared with lignocaine on the hemodynamic responses to laryngoscopy and intubation. J Clin Anesth. 2013;25(4):275-80.
12. Schnabel A, Meyer-Frießem CH, Reichl SU, et al. Dexmedetomidine vs. midazolam for sedation during prolonged mechanical ventilation: Two randomized controlled trials. JAMA. 2012;307(11):1151-60.