e-ISSN: 0975-1556, p-ISSN:2820-2643

Available online on www.ijpcr.com

International Journal of Pharmaceutical and Clinical Research 2024; 16(3); 1458-1463

Original Research Article

Evaluating the Comparative Efficacy of Intravenous Dexmedetomidine and Lignocaine in Mitigating Hemodynamic Responses during Laryngoscopy and Tracheal Intubation through a Randomized Control Study

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Received: 25-12-2023 / Revised: 23-01-2024 / Accepted: 26-02-2024

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Conflict of interest: Nil

Abstract:

Background & Aims: This study aims to scrutinize the hemodynamic perturbations induced by laryngoscopy and tracheal intubation, exploring the modulating effects of Dexmedetomidine and Lignocaine. Emphasis is placed on evaluating the efficacy of a reduced Dexmedetomidine dosage.

Methods and Materials: This single-center study at Gauhati Medical College and Hospital enrolled 82 ASA I-II patients (18–60 years) undergoing elective surgery. Randomized into Group D (dexmedetomidine) or Group L (lignocaine), patients received anesthesia according to group-specific protocols. The study aimed to compare the effectiveness of intravenous Dexmedetomidine (0.5 microgram/kg) and intravenous Lignocaine (1.5 mg/kg) in attenuating hemodynamic responses during laryngoscopy and tracheal intubation.

Results: Of the 120 screened patients, 82 were randomized (41 in each group) with comparable baseline measurements. Both groups exhibited significant changes in heart rate, systolic/diastolic blood pressure, and mean arterial pressure during laryngoscopy and intubation. Dexmedetomidine showed a lower maximum rise in heart rate (8.17%) than Lignocaine (17.49%), indicating its potential advantage. Dexmedetomidine also demonstrated a milder increase in systolic blood pressure. Both drugs were well-tolerated with no serious adverse effects. Sedation levels were statistically insignificant.

Conclusion: Administering intravenous Dexmedetomidine (0.5 microgram/kg) proved more effective in mitigating hemodynamic pressor changes during laryngoscopy and tracheal intubation than intravenous Lignocaine (1.5 mg/kg). Dexmedetomidine achieved this without significant adverse effects, highlighting its potential as a preferable option in clinical practice. Both drugs demonstrated the ability to alleviate pressor changes, but Dexmedetomidine, at a lower dosage, offers a favorable balance of effectiveness and safety.

Keywords: Laryngoscopy, tracheal intubation, hemodynamic variations, sympathoadrenal responses, Dexmedetomidine, Lignocaine.

Introduction

Laryngoscopy and endotracheal intubation often lead to changes in hemodynamics and activate sympathoadrenal responses by stimulating the supraglottic region. The usual result is a temporary and unpredictable rise in heart rate and blood pressure caused by the stimulation of vagal and cardiac accelerator fibers during laryngoscopy. [1]

Various medications have been employed to mitigate hemodynamic events, including opioids, alpha and beta-adrenergic blockers, calcium channel antagonists such as diltiazem and verapamil, and alpha-2 agonists. Lignocaine is a commonly used drug for reducing the pressor response due to its well-established centrally depressant and anti-arrhythmic properties.[2]

Dexmedetomidine, a novel imidazole derivative and a selective alpha-2 adrenergic receptor agonist, demonstrates eight times greater affinity for alpha-2 adrenoceptors compared to clonidine. Pre-administration of dexmedetomidine has been demonstrated to lessen the hemodynamic response during laryngoscopy and tracheal intubation.[3]

The majority of studies have employed intravenous Dexmedetomidine at a dose of 1 microgram/kg to effectively attenuate hemodynamic responses. [4]

Methods and Materials

Ethics: This prospective, randomized single hospital study was conducted under the Department of Anesthesiology and Critical Care, Gauhati Medical College and Hospital, Guwahati from September 2021 to August 2022, with prior permission and approval from the Hospital Ethics Committee.

Study Design: Eighty-two consenting patients, aged 18–60 years and belonging to either sex, with American Society of Anesthesiologists (ASA) physical status I-II and Mallampati classification grade I-II, scheduled for elective surgery under general anesthesia with endotracheal intubation, were enrolled in this study. Preoperative assessments included a detailed history covering exercise tolerance, comorbidities, allergies, and surgical history. Patients were excluded if they had a history of cardiac and pulmonary disease, pregnancy, morbid obesity, drug allergy, hypertension, impaired kidney or liver function, or anticipated difficult airway. They were then randomly assigned to one of the two groups, namely Group D (dexmedetomidine) and Group L (Lignocaine), with 41 patients in each group. The allocation was done using a computer-generated list of random numbers by an independent observer who was blinded to both groups. The generated numbers were concealed in envelopes and provided to the anesthesiologists, ensuring the allocation remained undisclosed.

Anaesthetic Technique: On the day of the surgery, within the operating theatre, an 18G cannula was used to establish an intravenous line. Additionally, a pulse oximeter (SPO2), non-invasive blood pressure (NIBP) cuff, and ECG monitors were connected. Baselines parameters, including heart rate (HR), systolic blood pressure (SBP), diastolic blood pres-sure (DBP), and mean arterial pressure (MAP), were recorded before the administration of drugs.

Patients in Group D received intravenous dexmedetomidine at a dose of 0.5 μg/kg over 10 minutes as premedi-cation before the induction of general anesthesia. Patients in Group L received intravenous lignocaine at a dose of 1.5 mg/kg in 10 ml normal saline, 3 minutes before laryngoscopy and intubation.

Following the completion of the study drug infusion, all patients underwent preoxygenation with 100% oxygen for 3 minutes. General anesthesia was induced with intravenous propofol at a dose of 2 mg/kg. After the loss of response to verbal commands, intravenous succinylcholine at a dose of 2 mg/kg was administered according to the standard protocol. Laryngoscopy, performed by an expert anesthesiologist using the Macintosh curved blade of an appropriate size, was followed by intubation with a cuffed endotracheal tube of the suitable size, confirmed using capnography. The laryngoscopy time was defined as the time from the introduction of the laryngoscope blade into the oropharynx to the appearance of the capnography curve on the monitor, limited to less than 20 seconds. Patients with a laryngoscopy time exceeding 30 seconds were excluded from the study. Bilateral equal air entry was confirmed by auscultation, the tube was secured, and patients were placed on controlled ventilation using a closed circuit with a circle absorber system. All patients received intravenous vecuronium at a dose of 0.08 mg/kg for muscle relaxation and were maintained on intermittent bolus doses of vecuronium at 0.02 mg/kg as required, along with oxygen and isoflurane at 1%–1.5%. Intravenous paracetamol at a dose of 1 g was administered as an analgesic. During the 10-minute period following intubation, no stimuli, such as surgical intervention, nasogastric tube insertion, surgical incision, or drug administration, were given.

Vital parameters, including HR, SBP, DBP, MAP, and RR were recorded at 1, 3, 5, and 10 minutes after intubation. Patients were observed for any episodes of bradycardia (HR <50 beats/min), hypotension (SBP <20% baseline), and any other adverse events during surgery. Bradycardia was treated with intravenous atropine at a dose of 0.6 mg, and hypotension was managed with a fluid bolus followed by intravenous ephedrine at a dose of 0.5-0.6 mg/kg in unresponsive cases.

After completing the surgery, residual neuromuscular blockade was reversed with intravenous neostigmine at a dose of 0.05 mg/kg and intravenous glycopyrrolate at a dose of 0.01 mg/kg. Patients were extubated after com-plete clinical recovery and were then shifted to the post-anesthesia care unit.

The Ramsay sedation scale score was utilized to assess sedation at baseline and before the induction of anesthesia in both groups:

Ramsay Sedation Scale with corresponding sedation scores: [5]

- Anxious, agitated, restless - Score: 1
- Awake, cooperative, tranquil, oriented - Score: 2
- Responds to verbal commands - Score: 3
- Brisk response to loud noise - Score: 4
- Sluggish response to loud noise - Score: 5
- No response to loud noise - Score: 6

Statistical Analysis: The data analysis involved the use of Microsoft Excel, GraphPad Prism, and

IBM SPSS V21. Descriptive statis-tics were presented through tables, pie charts, and bar diagrams. The association between categorical variables was assessed using Chi-square and Fisher's exact tests. Normality of the data was checked using Kolmogorov-Smirnova and Shapiro-Wilk tests.

For continuous variables, the independent T-test and Paired T-test were employed to compare mean differences between two independent and related groups, respectively, depending on the fulfilment of the normality as-sumption. In cases where the normality assumption was not met, non-normal data comparison between the two groups was conducted using the Mann Whitney test. The entire data analysis was performed using SPSS version 21, and a significance level of $P \leq 0.05$ was considered statistically significant.

Sample size calculation: The determination of our study's sample size relies on the outcomes of a previous study by Kondavagilu SR et al.(4) With a study power set at 80% and a significance level of 5%, the calculations were conducted for heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) at various time points: baseline, just before induction, at the time of intubation, and 1, 3, 5, and 10 minutes after intubation.

The calculation for the largest sample size was based on a standard deviation of 13.86, aiming to detect a difference of 10 beats per minute in patients' heart rate. Considering an attrition rate of 15%, we have decided to include 41 patients in each group, resulting in a total sample size of 82 patients.

Results

A total of 120 patients underwent screening for inclusion criteria in this study. Following screening, 82 eligible patients were randomly assigned into two groups of 41 patients each using block randomization, ensuring similarity in terms of age, gender, weight, and ASA physical status (Table 1). Baseline measurements of heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) were comparable in both groups ($P > 0.05$).

Intragroup comparisons of HR in patients receiving intravenous Dexmedetomidine (Group D) revealed signifi-cant changes in mean heart rate at intubation and 1, 3, 5, and 10 minutes post-intubation. The maximum rise, observed at 1 minute post-intubation, was 8.17% ($P < 0.05$).

Similar significant HR changes were observed in the Lignocaine group (Group L) at intubation and 1, 3, and 5 minutes, with the maximum rise occurring at 1 minute post-intubation at 17.49% ($P < 0.05$). The heart rate returned to baseline approximately 10 minutes post-intubation. Inter-group comparisons between the two groups showed a significant increase in HR in Group L compared to Group D at intubation and 1, 3, 5, and 10 minutes post-intubation ($P < 0.05$) (Table 2).

Intragroup comparisons of mean SBP in Group D demonstrated a significant increase immediately after tracheal intubation and at 1, 3, and 5 minutes post-intubation, with the maximum rise occurring at 1 minute post-intubation (4.73%). SBP returned to normal at subsequent intervals. Patients in Group L exhibited a significant rise in SBP at intubation and 1 and 3 minutes post-intubation, with the maximum rise occurring at 1 minute post-intubation (9.57%). Subsequent intervals showed SBP returning to normal, with no significant difference from baseline.(Table 3)

Group D showed a significant increase in mean DBP at intubation and 1 minute post-intubation. Group L demonstrated a statistically significant rise in DBP at intubation and 1 and 3 minutes post-intubation, with the maximum rise at 1 minute post-intubation (14.52% change from baseline).(Table 4)

Group D exhibited a significant increase in MAP after intubation and at 1 minute, followed by a decrease at 10 minutes. Group L showed a significant increase in MAP at intubation and at 1, 3, and 5 minutes post-intubation, followed by a fall at 10 minutes post-intubation. The maximum rise in MAP was observed at 1 minute post-intubation, with a rise of 12.25%.(Table 5)

Intergroup comparisons of mean systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arte-rial pressure (MAP) at similar intervals, from baseline to 10 minutes post-intubation, indicated a significant in-crease in Group L compared to Group D at 1, 3, and 5 minutes ($P < 0.05$).

Sedation levels in both groups were found to be statistically insignificant, with a p-value of 1.00 in both groups.

Discussion

Our study tried to explore the intricate hemodynamic responses induced by laryngoscopy and tracheal intubation, with a particular focus on assessing the effectiveness of two distinct pre-operative interventions: intrave-nous Dexmedetomidine and intravenous Lignocaine. These interventions were chosen based on their potential to modulate the sympathetic response through different mechanisms.

Dexmedetomidine, functioning as an alpha-2 adrenergic agonist, exerted its effects centrally to reduce sympathetic outflow, while Lignocaine, a local anesthetic, attenuated afferent signals from

mechanoreceptors in the pharyngeal wall, epiglottis, and vocal cords.[6] The significance of this study lay in the context of the well-established knowledge that laryngoscopy and tracheal intubation can lead to substantial hemodynamic variations and heightened sympathetic system activity. These physiological changes often manifest as elevations in heart rate, blood pressure, and cardiac arrhythmias, primarily attributed to the stimulation of mechanoreceptors in the upper airway.[7]

The selected interventions, Dexmedetomidine and Lignocaine, represented distinct pharmacological approaches to address this challenge. Dexmedetomidine, known for its alpha-2 adrenergic agonist properties, has been associated with a central reduction in sympathetic outflow, potentially blunting the exaggerated response to airway manipulation. On the other hand, Lignocaine, a local anesthetic, aimed to attenuate afferent signals from the upper airway, thereby mitigating the reflex sympathetic response. [7]

Our study cohort comprised 82 consenting patients, carefully selected based on ASA physical status class I and II, aged between 18 to 60 years, and undergoing elective surgery under general anesthesia. Random assignment of patients to either the Dexmedetomidine or Lignocaine group ensured an unbiased evaluation of the interventions. To eliminate confounding factors, both groups underwent identical pre-medication, received the same anesthetic agents, intravenous fluids, and other drugs. The analgesic drugs, including intravenous Fentanyl and intravenous Paracetamol, were administered at identical doses in both groups.

The demographic data analysis revealed the absence of significant differences between the two groups concerning age, gender, weight, height, and ASA physical status. This meticulous matching aimed to establish a foundation for a robust comparison, ensuring that any observed variations in hemodynamic parameters could be attributed to the specific interventions rather than demographic dissimilarities.

Both Dexmedetomidine and Lignocaine exhibited effective control of hemodynamic parameters, en-compassing heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) during the critical periods of laryngoscopy and tracheal intubation. The thorough examination of these parameters at various intervals, including baseline, before intubation, at intubation, and 1, 3, 5, and 10 minutes after intubation, provided a nuanced understanding of the temporal dynamics of the hemodynamic responses.

In terms of heart rate (HR), both groups experienced a post-intubation increase, gradually returning to baseline. However, the Lignocaine group exhibited a higher maximum rise (17.49% from baseline) compared to the Dexmedetomidine group (8.17% from baseline). This observation suggested a differential impact on the auto-nomic nervous system, emphasizing the distinct influences of the two drugs during the intubation process.

The analysis of systolic blood pressure (SBP) revealed an increase post-intubation until 5 minutes in the Dexmedetomidine group, followed by a subsequent decrease. In the Lignocaine group, an increase was noted until 3 minutes post-intubation, followed by a decline. The comparison between the groups unveiled a higher SBP in the Lignocaine group (maximum rise from baseline being 9.57%) compared to the Dexmedetomidine group (maximum rise from baseline being 4.73%) at all intervals. These variations highlighted the nuanced effects of Dexmedetomidine and Lignocaine on the cardiovascular system's response to intubation.

Analysis of diastolic blood pressure (DBP) demonstrated an increase in patients receiving Dexmedetomidine post-intubation until 1 minute, while in patients receiving Lignocaine, there was an increase post-intubation until 3 minutes. Baseline DBP was comparable between the groups, but it was consistently higher in the Lignocaine group at all intervals. This sustained difference in DBP suggested that Lignocaine may have a more prolonged effect on diastolic blood pressure compared to Dexmedetomidine.

Mean arterial pressure (MAP) analysis revealed an increase post-intubation, gradually decreasing after 10 minutes in patients receiving Dexmedetomidine (maximum rise of 5.672% from baseline). A similar trend was observed in the Lignocaine group (maximum rise of 12.25% from baseline), with the maximum change in MAP seen in the patients receiving Lignocaine. This finding underscored the differential impact of the two drugs on overall perfusion pressure.

The alignment of our study's findings with those of previous studies conducted by Mahjoubifard M et al, Singh V et al and Anandani et al provided additional validation.[8,9,10] These studies also reported higher HR in the Lignocaine group compared to the Dexmedetomidine group, with significantly higher SBP, DBP, and MAP in the Lignocaine group. Notably, our study utilized a lower dose of Dexmedetomidine (0.5 mcg/kg) compared to their higher dose (1 mcg/kg), suggesting that a lower dose of Dexmedetomidine could effectively blunt the he-modynamic response during laryngoscopy and intubation.

A study by Gulabani et al further corroborated our findings, reporting that Dexmedetomidine at a dose of 0.5 mcg/kg effectively blunted the tachycardia response to intubation but incompletely attenuated

the increase in SBP and DBP. [11] Additionally, Lignocaine at a dose of 1.5mg/kg, administered 3 minutes before laryngoscopy and intubation, was more effective than Dexmedetomidine 0.5mcg/kg in attenuating the increase in SBP and DBP at 3 minutes and 5 minutes after endotracheal intubation.

In another study by Seangrung R et al, Lidocaine (1.5mg/kg) with additional Propofol (0.5 mg/kg) demonstrated a non-inferior effect compared with Dexmedetomidine (1mcg/kg) in attenuating the hemodynamic response following laryngoscopy and endotracheal intubation and had fewer side effects. [12] This observation could be attributed to the addition of Propofol in the Lidocaine group, highlighting the potential synergistic effects of different drug combinations.

Our study's chosen protocol involved the administration of Dexmedetomidine at a dose of 0.5mcg/kg in 100 ml normal saline over 10 minutes as an infusion. This lower dose of Dexmedetomidine not only proved to be cost-effective but also exhibited fewer side effects while effectively attenuating hemodynamic responses during lar-yngoscopy and intubation. This aligns with findings from other studies supporting the efficacy of lower doses of Dexmedetomidine in achieving the desired hemodynamic control.

Importantly, the trial drugs were well-tolerated by patients, and no serious hemodynamic complications or other adverse effects were noted during the study period. Notably, there were no instances of bradycardia or hypoten-sion in either group. The observed lesser side effects could be attributed to the use of a lower dose of Dexmedetomidine, consistent with findings in other studies employing lower doses of this alpha-2 adrenergic agonist. [10,11,12]

Changes in peripheral oxygen saturation (SPO2) were found to be statistically insignificant in our study. Sedation, assessed using the Ramsay Sedation Score, yielded statistically insignificant results with a p-value of 1.00. This lack of significant changes in oxygen saturation and sedation levels may be attributed to the use of a low dose of Dexmedetomidine in our study, suggesting a favorable safety profile. [11]

Despite the significant findings, our study had certain limitations. It was a single-hospital-based study, and while it provided valuable insights, a multi-hospital study would have offered a more comprehensive evaluation of hemodynamic parameters.

Additionally, the sample size of 82 patients may have limited the generalizability of our results. Acknowledging these limitations, we emphasize the importance of future research endeavors with larger sample sizes and multi-center collaborations to enhance the robustness and applicability of our findings.

Conclusion

The study establishes the superiority of intravenous Dexmedetomidine (0.5 microgram/kg) over intravenous Lignocaine (1.5 mg/kg) in effectively mitigating hemodynamic pressor changes during laryngoscopy and tracheal intubation.

Dexmedetomidine demonstrates this superiority without inducing significant adverse effects, positioning it as a more favorable option in clinical scenarios. While both agents exhibit the capacity to attenuate pressor responses, the lower dosage of Dexmedetomidine underscores its potential as an optimal choice, striking a beneficial balance between efficacy and safety in the management of hemodynamic fluctuations during these critical procedures.

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