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Comparison of the Efficacy of Intravenous Lignocaine and Intravenous Dexmedetomidine in Attenuation of Hemodynamic Stress Response to Laryngoscopy and Endotracheal Intubation

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ABSTRACT

Objective: To study the effect of intravenous Lignocaine and Dexmedetomidine to attenuate cardiovascular stress reaction to laryngeal manipulation.

Study Design: Quasi-experimental study.

Place and Duration of Study: Department of Anesthesia, Combined Military Hospital, Peshawar Pakistan, from Oct 2019 to Apr 2020.

***Methodology*:** Patients were randomly divided into two Groups by lottery method. Seventy-three were in the intravenous Lignocaine-Group-(A), while 73 patients were in the intravenous Dexmedetomidine-Group-(B). Blood pressure, Pulse rate and ECG were documented at the start of general anaesthesia and after 1, 3 and 5 minutes of intubation. Efficacy was defined if systolic pressure and pulse remained within 20% of the patient's native findings between 1 and 5 minutes after endotracheal intubation.

Results: A total of 146 patients were included in the stud. The mean baseline systolic BP of Group-A was 121.34±4.47mmHg and mean baseline diastolic BP was 80.31±3.57mmHg. While mean Baseline Systolic BP WAS 121.19±4.37mmHg in Group-B and mean baseline diastolic BP was 80.04±3.66mmHg. Efficacy was seen 74% patients of Group-A and 90.4% of Group-B (p=0.009).

Conclusion: Dexmedetomidine 1 microgram/kg blunts the cardiovascular sympathetic reaction to laryngeal manipulation more pronounced than Lignocaine 1.5 milligram/kg without any harmful outcomes.

Keywords: Dexmedetomidine, Endotracheal intubation, Hemodynamic stress response, Laryngoscopy, Lignocaine.

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INTRODUCTION

Laryngeal manipulation is commonly performed in anaesthesia.[1] This technique includes stimulation of pharyngeal and laryngeal structures, which are expansively surrounded by parasympathetic and sympathetic nerves. The discharge of vasoactive mediators practically accompanies direct manipulation of these structures after initiation of anaesthesia.[2] Cardiovascular reaction to laryngeal manipulation causes a rise in pulse rate, blood pressure, central nervous system pressures, intraocular tension and cardiac dysrhythmias.[3] Moreover, it can also lead to cardiac arrest. These clinical findings are observed as a physiological response of laryngeal manipulation, usually enhanced by Light Anesthesia, decreased ventilation, Anxiety and Reflex baroreceptor effect following induction agents like Propofol. Patients with high blood pressure are predisposed to hypertension.[4] Hemodynamic findings are not severe in normal patients but might be dangerous in patients with cardiovascular and central nervous system disorders.[5] Rashmi HD reported the cardiovascular response of intubation during anaesthesia.[6] Much study is being processed to avoid or at least blunt these reactions.

Dexmedetomidine is a different compound having multiple times more similar receptor affiliation as compared to clonidine. Treatment with this drug before anaesthesia reduces the cardiovascular reaction to the stress of intubation.[7]

Prasad SR has shown that the efficacy of intravenous Lignocaine was 78% and 94% with intravenous Dexmedetomidine in reduction of cardiovascular reaction to tracheal manipulation.[8]

No such study has been done earlier on this subject in our local population. Dexmedetomidine is a new drug, so its efficacy must be evaluated in our general population. Therefore we have planned to associate the effectiveness of Lignocaine and Dexmedetomidine in reduction of cardiovascular reaction to tracheal

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manipulation during intubation. If results favour Dexmedetomidine, it can be used routinely in our general population.

METHODOLOGY

This quasi-experimental study was conducted at the Department of Anesthesia, Combined Military Hospital, Peshawar Pakistan, from October 2019 to April 2020. Permission from the Ethical Committee Review Board was take. A sample size of 146 was calculated taking confidence level=95% and alpha=5% (two-sided) with power=80%, using efficacy in (intravenous Lignocaine)=78% and expected efficacy in (intravenous Dexmedetomidine)=94%,[8] the estimated sample size was 146. Seventy-three individuals were in the intravenous Lignocaine-Group (A), and the other 73 patients were in the intravenous Dexmedetomidine-Group (B).The non-probability consecutive technique was used for sampling.

Inclusion Criteria: Patients of either gender with ages ranging from 18-50 years, presenting to the Outpatient Department for elective surgery with ASA Grade I & II, Mallampati Class I-II were included in the study.

Exclusion Criteria: Patients allergic to Dexmedetomidine or Lignocaine, known cases of hypertension, ischemic heart disease and arrhythmias, pregnancy, oropharyngeal surgery, suspected airway anomalies or anticipated difficult airway, unable to intubate in less than 1 minute and emergency Surgeries were excluded from the study.

At the start of study, baseline demographics (age, gender and weight on weighing machine, ASA score and Mallampati class) were recorded. Written informed consent was taken from patients after clarification about the study.

Randomization was performed by lottery method for both Groups. The Group assignments written on paper were put into an opaque jar and picked when the patient was admitted for the procedure. Seventy-three patients were in the intravenous Lignocaine-Group (A), while 73 patients were in the intravenous Dexmedetomidine-Group (B).

On entrance to the operation theatre, standard vitals such as pulse rate, blood pressure, breathing frequency, and saturation was documented. The patients were preloaded with half litres of lactated Ringer's solution. Group-A was given 100ml of 0.9% saline before the procedure, and after ten minutes,[1.5] milligram /kg of Lignocaine was given IV three minutes prior to laryngoscopy. In Group-B, Dexmedetomidine 1 microgram/kg dissolved in 100ml of 0.9% saline IV in 10 minutes was given; all this was finalized 10 minutes prior to induction. All cases were given injection dexamethasone 0.08milligram/kg IV, injection Nalbuphine 0.1mg/kg IV and injection Midazolam 0.02milligram/kg IV before preoxygenation. All individuals were given injection Propofol. Mechanical ventilation was assisted with 0.5 milligram/kg of Atracurium IV 3 minutes prior to manipulation of the trachea. Laryngoscopy was accomplished by Macintosh laryngoscope, intubated, and attached to a ventilator. After verification of correct placement, the tube was secured. No stress was given for 10 min at the start of the cases. Anaesthesia was sustained by an inhalational agent along with atracurium. On commencement of surgery, all were given an injection of Neopyrolate 0.05mg/kg IV. The endotracheal tube was pulled out on recovering pharyngeal reflexes.

Blood pressure, Pulse rate and ECG were documented on induction and intubation 1,3, and 5 minutes after intubation. Efficacy was defined if Systolic pressure and pulse remained within 20% of the patient's native findings between 1 and 5 minutes after endotracheal intubation.

Data was entered in Statistical Package for the social sciences (SPSS) version 22:00. Mean±SD was shown for markers like age, weight and baseline blood pressure. In addition, frequencies and percentages were obtained for markers like gender, ASA score, Mallampati class and efficacy. The *p*-value lower than or up to 0.05 was considered as significant.

RESULTS

Seventy-three patients that were included in Group-A had a mean age of 36.863±5.49 years, mean weight of 74.36±7.15 Kg, mean Baseline Systolic BP of 121.34±4.47mmHg and mean Baseline Diastolic BP was 80.31±3.57mmHg.

Out of 73 patients in Group-B, the mean age of 38.23±4.56 years, mean weight was 77.08±7.27Kg, mean Baseline Systolic BP was 121.19±4.37mmHg and mean Baseline Diastolic BP was 80.04±3.66mmHg.

The male gender was dominant (54, 74.0%) in Group-A and Group-B (61,83.6%), as displayed in Table-I. In our study, 66(90.4%) Group-B patients who were given injection Dexmedetomidine showed significant efficacy (p=0.009) as compared to 54(74.0%) Group-A patients who were treated with injection Lignocaine, as displayed in Table-II.

Table-I: Descriptive Statistics of Study Parameters in both Groups (n=146)

Study Parameters	Group-A (n=73) (Mean±SD)	Group-B (n=73) (Mean±SD)
Age(years)	36.86± 5.49	38.23± 4.56
Weight (Kg)	74.36±7.15	77.08±7.27
Baseline Systolic BP (mmHg)	121.34±4.47	121.19±4.37
Baseline Diastolic BP (mmHg)	80.31±3.57	80.04±3.66
Gender		
Male	54 (74%)	61 (83.6%)
Female	19 (26%)	12 (16.4%)

Table- II: Comparison of Efficacy of in Both Groups(n=146)

Efficacy	Group-A (n=73)	Group-B (n=73)	*p*-value
Yes	54(74.0%)	66(90.4%)	0.009
No	19(26.0%)	7(9.6%)	

DISCUSSION

Laryngeal manipulation during anaesthesia is the most life-threatening procedure in general anaesthesia as they aggravate transitory but noticeable sympatho-adrenal reaction demonstrating as raised blood pressure and increased heart rate.[9] Numerous writers tried many options for reducing cardiovascular response to laryngeal manipulation.[10] However, they all had restrictions. Many researchers used Lignocaine to decrease the sympathetic reaction. Lignocaine is an inexpensive and harmless option utilized in several centres to reduce the sympathetic reaction of laryngeal manipulation. Compounds like clonidine or Dexme-detomidine weaken the dangerous hemodynamic res-ponses of laryngeal manipulation. Dexmedetomidine, a fresh alpha-2 agonist, has multiple affinities towards alpha-2 receptors compared to clonidine.[11,12] Dexmedetomidine shows distinctive effects on hemodynamic constancy and pronounced benefit in supporting spontaneous ventilation.[13-16] In our study, effectiveness was seen in 74% of patients in Group-A (Lignocaine) as compared to 66(90.4%) in Group-B (Dexmedetomidine) (p=0.009). Research by Prasad *et al*. indicated that intravenous Lignocaine's effectiveness was 78% and 94% with intravenous Dexmedetomidine in reducing cardiovascular reaction to laryngeal manipulation.[8]

Lignocaine 1.5 mg/kg IV was given 3 minutes before laryngeal manipulation. Numerous writers have proved that 1.5 mg/kg of Lignocaine prevents sympathetic activation due to laryngeal manipulation. Dexmedetomidine in this research was one microgram /kg dissolved in 100ml of 0.9% saline. Some writers.[16-20] gave 0.5-1microgram/kg of Dexmedetomidine for reducing sympathetic response to laryngeal manipulation. After using Dexmedetomidine showed an 11% reduction in pulse rate and a 9% reduction in blood pressure. These interpretations were related to other literature and were described as the effect of a reduced central sympathomimetic response.[17-20] A small number of authors.[20,21] Illustrated a substantial decrease in heart rate for the Dexmedetomidine-Group and a minor decrease in blood pressure. All individ-uals in Group-A had a score of sedation as twos in pre-induction times. It happened to owe to injection of Nalbuphine in premedication. The maximum of the individuals in Group-B had a score of sedation. Not a single patient in Group-B had a change in saturations. Numerous researchers,[18,22,23] have described Dexmedetomine as simulating normal sleep and patients can be awakened by voice stimulus without ventilation. These qualities made Dexmedetomidine a superior choice for awake fibreoptic intubations. The amount of Propofol necessary for initiation of anaesthesia was considerably reduced in Group-B relative to Group-A. Bajwa *et al*.[20] described reduced thiopentone necessity in the initiation of anaesthesia in the Dexmedetomidine Group. Lignocaine did not efficiently reduce cardiovascular stress reaction to laryngeal manipulation. A few authors described Lignocaine as reducing cardiovascular reaction, and our interpretations are the same. Matched with Lignocaine, Dexmedetomidine is considerably effective in reducing cardiovascular reaction to laryngeal manipulation but could not completely blunt the reaction. Many researchers,[17-21] described that Dexmedetomidine 0.5-1 microgram/kg considerably reduced cardiovascular reaction to laryngeal manipulation but did not blunt, and our interpretations are the same. None of the patients had decreased heart rate or blood pressure necessitating involvement in Group-B. Dexmedetomidine was given to avoid a decrease in pulse rate or blood pressure associated with the bolus dose.[16] These effects were not seen in cardiac patients, which was a restriction in this research. It would be added beneficial for high-risk cardiac cases.

CONCLUSION

Dexmedetomidine 1microgram/kg IV reduces the car-diovascular reaction to laryngeal manipulation more effi-ciently relative to Lignocaine1.5 mg/kg, lacking harmful properties. Moreover, Dexmedetomidine reduces the amount of Propofol at the initiation of anaesthesia.

Conflict of Intrest: None.

Author's Contribution

Following authors have made substantial contributions to the manuscript as under:

RN: Study design, data analysis, critical review, drafting the manuscript, critical review, approval of the final version to be published.

SHF & MSA: Data acquisition, data analysis, data interpretation, critical review, approval of the final version to be published.

SAUJ & MS: Conception, data acquisition, drafting the manuscript, approval of the final version to be published.

NA: Drafting the manuscript, data interpretation, critical review, approval of the final version to be published.

Authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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