10.21761/jms.v9i01.07

ORIGINAL RESEARCH ARTICLE

A Comparative Study to Assess the Efficacy of 2% Intravenous Lignocaine and 50% Magnesium Sulphate in Attenuating the Hemodynamic Response to Laryngoscopy

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ABSTRACT

Introduction: Hemodynamic response to laryngoscopy and intubation is a known phenomenon. Numerous pharmacological and non-pharmacological methods have been employed in an attempt to blunt such responses. However, the search for a perfect agent still continues. A study was conducted in order to compare and assess the efficacy of intravenous lignocaine and magnesium sulfate in blunting this hemodynamic response.

Material And Methods: This prospective, double-blind, randomized, Helsinki protocol-compliant clinical study was conducted after written informed consent and approval from the Institutional Ethical Committee. Two groups of 40 subjects each were constituted. Group A received 2% lignocaine 1.5 mg/kg bolus given over 1-minute, while group B received 50% magnesium sulphate 30 mg/kg. Study drugs were administered over 10 minutes. Anaesthesia protocol was standardized. Hemodynamics were observed following study drug administration (5, 10 minutes), following induction and following induction and at intubation, and 1, 2, 4, 6, 8,10 minutes following laryngoscopy and intubation. Side effects were also noted.

Results: Both groups were comparable demographically and at baseline, following the study drug administration. The difference between the two groups was clinically significant at 1, 2, 4, 6 minutes post-intubation (p = 0.005, 0.001, 0.004, 0.006). The maximum observed heart rate in group B was immediately post-intubation, which was lower than the baseline. In terms of mean arterial pressure, significant intergroup difference was also observed immediately post-intubation and at 1, 2, 4, 6 and 8 minutes post-intubation (p = 0.04, 0.02, 0.02, 0.08).

Conclusion: Intravenous magnesium sulphate 30 mg/kg (group B) was more efficacious than lignocaine 1.5 mg/kg (group A) in attenuating hemodynamic response to laryngoscopy and intubation.

Keywords: Lignocaine, Magnesium sulphate, Laryngoscopy.

Submission: 01-05-2024; **Acceptance:** 31-05-2024; **Published:** 30-06-2024

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How to cite this article: Seal A, Mowar A, Pahade A, Saran J. A Comparative Study to Assess the Efficacy of 2% Intravenous Lignocaine and 50% Magnesium Sulphate in Attenuating the Hemodynamic Response to Laryngoscopy. SRMS J Med Sci. 2024;9(1):38-42.

Source of support: Nil

Conflict of interest: None

INTRODUCTION

In modern anesthesia practice, rigid laryngoscopy and tracheal intubation are still considered the gold standard for airway care. More than 50 years ago, researchers recognized the effect of airway manipulation on heart rate and blood pressure levels.[1] It is now widely accepted that laryngoscopy and endotracheal intubation always result in hemodynamic alterations such as increased heart rate, increased blood pressure, and sometimes cardiac rhythm abnormalities.[2,3] These hemodynamic alterations occur as a result of the sympathoadrenal response and the release of norepinephrine and, to a lesser extent, epinephrine.[4] In normotensive patients, these hemodynamic alterations are transient and likely of little relevance.[5] However, these hemodynamic changes pose a risk to patients with hypertension, ischemic heart disease, or cerebrovascular illness.[6] Elevation of blood pressure and heart rate typically starts within 5 seconds of laryngoscopy, peaks in 1 to 2 minutes and returns to baseline level within 5 to 10 minutes. Many pharmacological and non-pharmacological techniques have been attempted to reduce the pressure reaction that occurs when endotracheal intubation is attempted.[7] Magnesium sulphate has been used to lessen the adrenergic reaction during tracheal intubation and laryngoscopy. However, the number of trials evaluating various intravenous magnesium sulphate dosages as a pressor response attenuating drug is quite few. In this study an attempt has been made to observe, assess and compare the efficacy of lignocaine (1.5 mg/kg body weight) and magnesium sulphate (30 mg/kg body weight) intravenous bolus over 10 minutes in attenuating the hemodynamic response following laryngoscopy and endotracheal intubation in patients in undergoing various elective surgeries under general anesthesia.

MATERIAL AND METHODS

This prospective, double-blind, randomized, Helsinki protocol-compliant clinical study was conducted after written informed consent and approval from the Institutional Ethical Committee. A total of 80 patients aged 18 to 60 years of either sex with the American Society of Anaesthesiologists (ASA) physical status classes I and II undergoing elective surgeries under general anaesthesia were enrolled in the study. Patient refusal; history of known allergy to anaesthetic agents used in our study or with comorbidities such as compromised renal, hepatic, pulmonary, and cardiac status; suffering from diabetes and or hypertensive illness; or having anticipated difficult intubation, laryngoscopy more than 15 seconds. Duration of Enrollment of patients commenced on the 1 of September 2022 and was completed on 28 of February 2024. Patients were randomized to two groups; group A received 2% lignocaine 1.5 mg/kg bolus given over 1-minute, while group B received 50% magnesium sulphate 30 mg/kg. Based on the study conducted by Misganaw *et al*.,[8] sample sizes were calculated as 36 patients per group with a power of study 80% and type 1 error < 0.05%.[9-11] Considering the probability of attrition during follow-up (attrition of 10%) exclusions due to difficult laryngoscopy, etc, 40 patients per group were enrolled. Every patient who was scheduled for surgery was subjected to a comprehensive pre-anesthesia examination the day before the scheduled surgery. A comprehensive general, systemic, and airway examination was completed. All necessary investigations were carried out, and the results were analyzed. Age, sex, height, weight, and BMI were among the demographic details recorded. The patients' anesthesia protocol was standardized. In accordance with the American Society of Anaesthesiologists' fasting, patients were kept at nil per oral.

Recommendations: In the pre-operative area, standard 18G iv access was achieved and patients were preloaded with ringer's lactate solution 2 mL/kg/hr. All standard monitors like ECG, pulse oximeter, and NIBP connected to the patients and baseline heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial blood pressure (MAP) were noted. All patients were pre-medicated with midazolam 0.02 mg/Kg. Drugs and hemodynamic parameters were recorded at 5-minute intervals from the beginning of administration till the end of the study drugs administration. Induction was commenced with an intravenous injection of fentanyl (2 mcg/kg) followed by an injection of propofol (2 mg/kg) till loss of verbal response was achieved. Muscle relaxation was accomplished with Vecuronium 0.1 mg/kg, following which a trained anaesthesiologist did a swift, smooth laryngoscopy and intubation. No surgical or any other stimulation was permitted during 10 minutes of the study period. Further anesthesia management was as per institutional protocol. The patients were monitored every hour for 24 hours in the postoperative period for any complications.

RESULTS

A total of 80 patients were recruited in the study. None of the patients were excluded from the analysis. Both groups were comparable demographically (Table 1). The primary outcome measures hemodynamic parameters, including HR, MAP and oxygen saturation levels, which were recorded 5 and 10 minutes after starting of administration of study drugs, following induction and at intubation and 1, 2, 4, 6, 8, 10 minutes following laryngoscopy and intubation (Table 2). The secondary

Table 1: Demographic profile of the patients enrolled in the study

	Group A	*Group B*	*p-value*
	Mean ± SD	*Mean ± SD*	
Age	29.23 ± 9.45	28.40 ± 4.29	0.663
Weight	63.03 ± 5.41	64.25 ± 7.16	0.391
Height	158.20 ± 8.67	160.70 ± 8.67	0.218
BMI	25.40 ± 3.38	24.85 ± 3.50	0.487

Test used: Student's t test

Table 2: Comparison of heart rate between study groups

HR	*Group A*	*Group B*	*p-value*
	Mean ± SD	*Mean ± SD*	
Baseline	81.63 ± 10.46	79.18 ± 13.98	0.378
At 5 minutes	79.2 ± 9.03	80.4 ± 15.56	0.674
At 10 minutes	78.8 ± 10.97	80.6 ± 15.31	0.547
Post induction 1 minute	80.90 ± 13.56	82.63 ± 15.33	0.609
Post induction 2 minutes	79.60 ± 14.32	83.78 ± 14.67	0.222
Immediate post intubation	78.55 ± 14.67	94.33 ± 18.86	0.221
Post intubation 1 minute	77.53 ± 14.08	97.13 ± 18.46	0.422
Post intubation 2 minutes	76.45 ± 13.84	97.53 ± 17.49	<0.001
Post intubation 4 minutes	76.03 ± 14.28	95.65 ± 18.72	<0.001
Post intubation 6 minutes	76.88 ± 13.37	94.38 ± 18.64	<0.001
Post intubation 8 minutes	77.1 ± 13.14	87.03 ± 17.8	0.006
Post intubation 10 min	77.72 ± 14.2	81.45 ± 14.87	0.255

Test used: Student's t test

Table 3: Intergroup comparison for mean MAP for the two study groups

MAP	*Group A*	*Group B*	*p-value*
	Mean ± SD	*Mean ± SD*	
Baseline	85.85 ± 15.67	87.08 ± 12.16	0.697
At 5 minutes	79.28 ± 14.67	86.1 ± 11.38	0.023
At 10 minutes	76.47 ± 14.19	82.55 ± 10.37	0.032
Post induction 1 minute	73.8 ± 13.87	78.75 ± 10.86	0.079
Post induction 2 minutes	68.2 ± 11.18	75.93 ± 10.33	0.002
Immediate post intubation	73.83 ± 11.07	90.38 ± 14.94	<0.001
Post intubation 1 minutes	74 ± 11.52	90.45 ± 13.09	<0.001
Post intubation 2 minutes	79.72 ± 11.27	96 ± 15.94	<0.001
Post intubation 4 minutes	78.05 ± 11.35	93.3 ± 12.94	<0.001
Post intubation 6 minutes	76.28 ± 11.87	90.73 ± 12.49	<0.001
Post intubation 8 minutes	75.18 ± 11.6	89.13 ± 13.76	0.006
Post intubation 10 minutes	74.88 ± 11.42	86.95 ± 12.97	0.255

Test used: Student's t test

Table 4: Intergroup comparison for mean SpO2 for the two study groups

SPO2	*Group A*	*Group B*	*p-value*
	Mean ± SD	*Mean ± SD*	
Baseline	99.69 ± 0.42	99.78 ± 0.48	0.37
At 5 minutes	99.49 ± 0.67	98.40 ± 0.28	0.43
At 10 minutes	100.00 ± 0.00	100.00 ± 0.00	–
Post induction 1 minute	100.00 ± 0.00	100.00 ± 0.00	–
Post induction 2 minutes	100.00 ± 0.00	100.00 ± 0.00	–
Immediate post intubation	99.78 ± 0.52	99.88 ± 0.50	0.38
Post intubation 1 minute	99.85 ± 0.32	99.88 ± 0.10	0.57
Post intubation 2 minutes	100.00 ± 0.00	100.00 ± 0.00	–
Post intubation 4 minutes	100.00 ± 0.00	100.00 ± 0.00	–
Post intubation 6 minutes	100.00 ± 0.00	100.00 ± 0.00	–
Post intubation 8 minutes	100.00 ± 0.00	100.00 ± 0.00	–
Post intubation 10 minutes	100.00 ± 0.00	100.00 ± 0.00	–

Test used: Student's t test

outcome measures included the side effects attributed to study drugs were monitored and recorded in the peri-operative period. Both groups were comparable in terms of baseline heart rate, mean arterial pressures (MAP) and oxygen saturation levels. Both groups displayed a mild reduction of heart rate but no significant intergroup difference following the administration of study drugs ($p = 0.833$, $p = 0.639$). There was no significant intergroup difference in heart rate between the two groups following induction as well ($p = 0.822$, $p = 0.329$). There was an increase in heart rate following intubation, which reached the peak at 2 minutes in group A, while no such response to laryngoscopy and intubation was observed in group B. The difference between the two groups was clinically significant at 1, 2, 4, and 6 minutes post-intubation (p = 0.005, 0.001, 0.004, 0.006). The maximum observed heart rate in group B was immediately post-intubation, which was lower than the baseline. The difference between the two groups was non-significant at 8 and 10-minutes post-intubation. In terms of mean arterial pressure, both groups were comparable with each other at baseline, at 5 and 10 minutes post-drug administration and at 1-minute post-induction. A significant drop in MAP was observed in group B, resulting in significant intergroup differences ($p = 0.008$) (Table 3). Significant intergroup difference was also observed immediately post-intubation and at 1, 2, 4, 6 and 8 minutes post-intubation ($p = 0.04$, 0.02, 0.02, 0.08) (Table 4). The intergroup difference was not significant at 10 minutes post-intubation ($p = 0.14$). Both groups were comparable at all time frames during the peri-operative period. No adverse effects were observed (Table 5).

DISCUSSION

Laryngoscopy and endotracheal intubation are linked to significant changes in hemodynamics and autonomic reflex activity, including an increase in heart rate, blood pressure, and sporadic disruptions in cardiac 0–0% intravenous magnesium sulphate in blunting hemodynamic response to laryngoscopy and intubation, rhythm during the induction of anesthesia.[2,3] These potentially harmful changes start as soon as the

Table 5: Intergroup comparison of side effects

	Group A		*Group B*	
	frequency	*%*	*frequency*	*%*
Hypotension	1	2.5	0	0.0
Bradycardia	1	2.5	0	0.0
Ponv	0	0.0	0	0.0
Allergic reaction	0	0	0	0
Desaturation/ bronchospasm	0	0	0	0

Test used: Chi-square test

laryngoscopy/intubation is performed, peaking after two minutes and then starting to decline and return to baseline after ten minutes. Preventing peri-operative morbidity and mortality requires effective attenuation (< 20% of baseline) of the hemodynamic response to laryngoscopy and tracheal intubation. Multiple variables impact the cardiovascular response linked to laryngoscopy and intubation, including the patient's age, the drugs employed, the kind and length of the procedures, the depth of anesthesia, any instances of hypoxia or hypercarbia, etc. Of these, the length of the laryngoscopy is the most important factor affecting cardiovascular responses.[9,10] The force used during a laryngoscopy barely makes a difference. The laryngoscopy and intubation times in our trial were restricted to less than 15 seconds, and skilled anaesthesiologists carried out all of the procedures. LiBurstein *et al.* discovered that the primary cause of the pressor response is an increased sympathetic response brought on by stimulation of the laryngopharynx and epi-pharynx. The afferent stimulation from the epiglottis and infraglottic region is carried by the vagus and glossopharyngeal nerves, which then activate the vasomotor center, triggering a peripheral sympathetic adrenal response that releases noradrenaline and adrenaline.[11,12] The pressure response has been altered using a variety of methods. All of these may come with additional dangers and adverse effects, and none of them completely inhibit response. Researchers have demonstrated magnesium sulfate ($MgSO_4$) inhibits catecholamine release, translating to reduced serum epinephrine and norepinephrine levels manifesting as reduced cardiac contractility, bradycardia, vasodilation and hypotension.[13] It has been demonstrated that intravenous magnesium is effective in conditions with excess circulating catecholamines. Moreover, MgSO4 directly lessens vascular contraction by reducing smooth muscle tonicity.[14-18] James *et al.* also demonstrated a reduction in plasma catecholamine levels in patients who were administered pre-operative magnesium sulphate and who underwent general anesthesia/laryngoscopy and intubation.[19] Researchers have investigated and compared the effect of pre-operative magnesium sulphate administered intravenously with agents such as lignocaine and most have found magnesium sulphate better.[9] However, there has been a paucity of research comparing the efficacy of different doses of intravenous magnesium sulfate in blunting hemodynamic response to laryngoscopy and intubation.

While group A received 2% lignocaine 1.5 mg/kg bolus given over 1-minute, group B received 50% magnesium sulphate 30 mg/kg. In terms of heart rate, there was no intergroup difference till immediately post-intubation (p >0.05). However, subsequently, group A (2% lignocaine) exhibited a rise in heart rate, which reached its peak at 2 minutes post-intubation and returned to baseline by 10 minutes. In contrast, the HR was relatively stable, with no surges observed in group B (magnesium sulfate 30 mg/kg) with a heart rate lower than baseline at all time points. This can be due to magnesium sulphate preventing catecholamine surge and counteracting the effect of calcium level surge.[10-13,19] Also, IV magnesium sulphate inhibits the sinoatrial node directly and indirectly, extending the recovery period of the sinus node. Also, following IV atropine, magnesium exerts a detrimental chronotropic impact.[20] Findings by Kotwani *et al.* and Nandal *et al.* were similar to our study and they observed intravenous magnesium sulphate 30 mg/kg optimal to control heart rate compensatory surges. Higher doses exhibited tachycardia secondary to hypotension.[21,22] Honarmand *et al*., also conducted a similar study, but in their study, magnesium failed to control tachycardia. They attributed this to the overwhelming sympathetic response, which the parasympathetic effects of magnesium sulphate could not control.[10] In terms of mean arterial pressure (MAP), though both groups attenuated the surge in blood pressure, group B was more effective in controlling the surge. Peak MAP in group A, though lower than the baseline, was higher than that after the study drug administration. Even this surge was absent in group B. Higher baseline values can be attributed to patient anxiety and higher pre-operative catecholamine levels, which were effectively countered by the study drugs. In studies conducted by Honarmund *et al*., Kotwani *et al.* and Nandal *et al.* magnesium mediated vasodilation and refractoriness of catecholamines on smooth muscles resulted in blunting of the incremental response of MAP.[10,21,22]At no time did the rate-pressure product reach the critical ischemia value of 12000 in either group, thereby making magnesium sulphate an effective drug in preventing ischemia due to raised HR and MAP. Also, in terms of side effects, the most common side effect was hypotension. The difference between the two groups was non-significant.

LIMITATIONS

No study is complete without limitations. Identifying the limitations of a study is essential as it paves the way for future research and critical thinking. We lacked on multiple counts in our research. Firstly, even though the sample size was based on previous studies, a study having a greater sample is likely to have greater accuracy. Secondly, we did not measure plasma catecholamines, magnesium, and calcium levels in different stages of

research. Hopefully, these lacunae shall be dealt with in future research.

CONCLUSION

Hemodynamic response to intubation and laryngoscopy is a universal phenomenon that needs to be blunted. Multiple pharmacological methods have been utilized to achieve this target. However, no satisfactory agent has been identified to date. Magnesium sulphate 50% (group B) in a dose of 30 mg/kg acts as a suitable agent to attenuate the hemodynamic response to laryngoscopy and intubation as compared to intravenous lignocaine 2% (group A) in a dose of 1.5 mg/kg.

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