Original article

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Comparison of inhaled 2% and intravenous 1% lidocaine on hemodynamic response and plasma norepinephrine levels in patients during bronchoscopy by using i-gel® laryngeal mask airway (LMA)

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Abstract

***Background*: Bronchoscopy is commonly performed for the diagnosis and treatment of respiratory tract diseases, but may pose a risk of oxygen desaturation, particularly in patients with certain conditions. The use of the i-gel® laryngeal mask airway (LMA) is a more practical alternative to endotracheal tubes. Inhaled lidocaine, used as a topical anesthetic for the respiratory mucosa prior to bronchoscopy, offers a lower risk of systemic side effects and is thought to be more effective in controlling hemodynamic responses than intravenous lidocaine. This study aimed to compare the effects of inhaled 2% lidocaine and intravenous 1% lidocaine on hemodynamic responses and plasma norepinephrine levels in patients undergoing bronchoscopy with the i-gel® LMA.**

***Methods*: This was a true experimental study with a single-blind, randomized controlled clinical trial design conducted on patients undergoing bronchoscopy at Wahidin Sudirohusodo Hospital, Makassar. Patients who met the inclusion and exclusion criteria were selected using a consecutive sampling method. Group A received inhaled 2% lidocaine, and Group B received intravenous 1% lidocaine. Observed parameters included hemodynamic measurements and plasma norepinephrine levels at three different time points.**

***Results*: A total of 46 patients were included in the study. The mean age of subjects was 54.52±13.29 years in the inhaled 2% lidocaine group and 55.87±13.01 years in the intravenous 1% lidocaine group. There were no statistically significant differences between the groups in baseline characteristics, including age, gender, body mass index (BMI), and American Society of Anesthesiologists Physical Status (ASA PS). However, the group receiving inhaled 2% lidocaine exhibited significantly more stable hemodynamic responses during the bronchoscopy procedure compared to the group receiving intravenous 1% lidocaine. This was evidenced by smaller changes (delta) from baseline values in systolic blood pressure, diastolic blood pressure, heart rate, mean arterial pressure, and plasma norepinephrine levels.**

***Conclusion*: Inhaled 2% lidocaine is more effective than intravenous 1% lidocaine in attenuating hemodynamic responses and reducing plasma norepinephrine levels during bronchoscopy procedures using the i-gel® LMA.**

Keywords: Inhaled lidocaine, intravenous lidocaine, bronchoscopy, hemodynamic response, norepinephrine, i-gel® LMA.

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Introduction

Flexible fiberoptic bronchoscopy (FFB) is a commonly utilized procedure for both the diagnosis and treatment of respiratory tract diseases. During this procedure, continuous monitoring of oxygen saturation and appropriate oxygen supplementation are essential. Certain conditions, such as advanced age (particularly over 80 years) and pulmonary fibrosis, may increase the risk of oxygen desaturation during FFB. Although the use of an endotracheal tube (ET) can provide secure airway control, it is often considered less practical, especially in outpatient settings. As a result, a simpler and less invasive method of airway management during bronchoscopy is warranted. (1–5)

The laryngeal mask airway (LMA), first introduced in 1983, has become a valuable alternative for airway management. Since 1989, it has been widely employed as a safe and effective tool in bronchoscopy procedures for both adult and pediatric patients. Its advantages include the ability to accommodate larger diameter bronchoscopes and to provide effective ventilation, particularly in patients with cervical spine trauma. (6–9)

Lidocaine is the most commonly used local anesthetic from the amide group and exerts its effects by stabilizing cell membranes. It can be administered either intravenously or via inhalation, each route offering distinct advantages and limitations. Intravenous lidocaine reduces the hemodynamic response by inhibiting sodium channels on nerve cell membranes. However, to effectively suppress airway reflexes, plasma concentrations approaching the toxic threshold are often required, which may lead to adverse effects on the central nervous and cardiovascular systems. (10–13)

Alternatively, inhaled lidocaine provides a safer topical anesthetic effect on the airway with a lower risk of systemic side effects. A study by Jokar et al. demonstrated that inhaled lidocaine was more effective than intravenous lidocaine in attenuating the hemodynamic response during intubation; however, the study did not assess its effect on plasma norepinephrine levels. (10,14) Plasma norepinephrine levels reflect sympathoadrenal activity, which plays a key role in hemodynamic responses. Therefore, the present study aimed to compare the effects of inhaled 2% lidocaine and intravenous 1% lidocaine on hemodynamic responses and plasma norepinephrine levels in patients undergoing bronchoscopy with the i-gel® LMA.

Research methods

This study employed a true experimental design using a randomized, single-blind, controlled clinical trial approach conducted at Wahidin Sudirohusodo Hospital, Makassar. The study commenced in November 2024 and continued until the required sample size was achieved. The study population comprised patients undergoing bronchoscopy procedures with anesthesia techniques using the i-gel® LMA. Subjects meeting the inclusion criteria were selected using a consecutive sampling method. A total of 46 subjects were enrolled and equally divided into two groups: Group A (inhaled 2% lidocaine) and Group B (intravenous 1% lidocaine), with 23 participants in each group.

Inclusion criteria were patients aged 18–65 years, with an American Society of Anesthesiologists Physical Status (ASA PS) classification of I–III and a body mass index (BMI) of 18.5–29.9 kg/m², who provided informed consent to participate. Exclusion criteria included patients who declined participation, were anticipated to have difficulty with LMA insertion, had a known allergy to the study drugs, or had immunological disorders, adrenal dysfunction, or cardiovascular diseases such as arrhythmias or atrioventricular (AV) block. Subjects were withdrawn from the study if complications occurred during the procedure, if LMA insertion required more than one attempt or exceeded 30 seconds, or if the patient voluntarily withdrew from the study.

Participants were randomly assigned to one of two groups. Group A received inhaled 2% lidocaine, while Group B received intravenous 1% lidocaine prior to the bronchoscopy procedure. The observed parameters included hemodynamic responses and plasma norepinephrine levels, which were measured at three time points: five minutes before the procedure (T0), one minute after bronchoscope insertion (T1), and ten minutes after bronchoscope insertion (T2). Data were analyzed using the Shapiro-Wilk test to assess normality, followed by either an independent t-test or the Mann-Whitney U test, depending on the distribution of the data. A p-value <0.05 was considered statistically significant.

Results

Based on **Table 1**, there were no statistically significant differences between the inhaled lidocaine group and the intravenous lidocaine group with respect to age, sex, ASA PS, and BMI ($p>0.05$), indicating comparable baseline characteristics between the two groups.

Table 2 shows that there was no statistically signif-

icant difference in systolic blood pressure (SBP) between the inhaled lidocaine group and the intravenous lidocaine group prior to bronchoscope insertion ($p>0.05$), indicating that baseline SBP values were comparable between the two groups. However, at 1 minute and 10 minutes after insertion, SBP values differed significantly between the groups ($p<0.05$). The changes in SBP from baseline to 1-minute post-insertion and from baseline to 10-minute post-insertion were also significantly different ($p<0.05$), with greater SBP fluctuations observed in the intravenous lidocaine group. In contrast, the difference in SBP change between 1 minute and 10 minutes post-insertion was not statistically significant ($p>0.05$). These findings suggested that the most prominent difference in SBP response between the two groups occurred within the first minute following bronchoscope insertion.

Based on **Table 3**, there was no statistically significant difference in diastolic blood pressure (DBP) between the inhaled lidocaine group and the intravenous lidocaine group prior to bronchoscope insertion ($p>0.05$), indicating that baseline DBP values were comparable between the two groups. However, at 1 minute and 10 minutes after insertion, significant differences in DBP were observed between the groups ($p<0.05$). The changes in DBP from baseline to 1 minute post-insertion and from baseline to 10 minutes post-insertion were also significantly different ($p<0.05$), with greater DBP fluctuations noted in the intravenous lidocaine group. In contrast, the difference in DBP change between 1 minute and 10 minutes post-insertion was not statistically significant ($p>0.05$). These findings suggest that the primary difference in DBP response between the two groups occurred within the first minute after bronchoscope insertion.

Table 4 reveals no statistically significant difference in heart rate between the inhaled lidocaine group and the intravenous lidocaine group prior to bronchoscope insertion ($p>0.05$), indicating that baseline heart rate values were comparable between the two groups. However, at 1 minute and 10 minutes after insertion, significant differences in heart rate were observed between the groups ($p<0.05$). The changes in heart rate from baseline to 1-minute post-insertion and from baseline to 10-minute post-insertion were also significantly different between the groups ($p<0.05$), with greater fluctuations observed in the intravenous lidocaine group. In contrast, the difference in heart rate change between 1 minute and 10 minutes post-insertion was not statistically significant ($p>0.05$). These findings suggest that the main difference in heart rate responses between the two groups occurred within the first minute after bronchoscope insertion.

Table 5 shows there was no statistically significant difference in total airway resistance (TAR) between the inhaled lidocaine group and the intravenous lidocaine group prior to bronchoscope insertion ($p>0.05$), indicating that baseline TAR values were comparable between the two groups. However, at 1 minute and 10 minutes after insertion, significant differences in TAR were observed between the groups ($p<0.05$). The changes in TAR from baseline to 1-minute post-insertion and from baseline to 10-minute post-insertion were also significantly different between the groups ($p<0.05$), with greater changes observed in the intravenous lidocaine group. In contrast, the difference in TAR change between 1 minute and 10 minutes post-insertion was not statistically significant ($p>0.05$). These findings suggest that the most prominent difference in TAR between the two groups occurred within the first minute following bronchoscope insertion.

Based on **Table 6**, there was no statistically significant difference in plasma norepinephrine levels between the inhaled lidocaine group and the intravenous lidocaine group prior to bronchoscope insertion ($p>0.05$), indicating that baseline norepinephrine levels were comparable between the two groups. However, at 1 minute and 10 minutes after insertion, significant differences in norepinephrine levels were observed between the groups ($p<0.05$). The changes in norepinephrine levels from baseline to 1-minute post-insertion and from baseline to 10-minute post-insertion were also significantly different between the groups ($p<0.05$), with greater increases observed in the intravenous lidocaine group. In contrast, the difference in norepinephrine level changes between 1 minute and 10 minutes post-insertion was not statistically significant ($p>0.05$). These findings suggest that the most substantial difference in norepinephrine response between the two groups occurred within the first minute following bronchoscope insertion.

Discussion

This study included 46 patients undergoing bronchoscopy procedures using the i-gel® LMA, who were assigned to one of two treatment groups: the inhaled 2% lidocaine group and the intravenous 1% lidocaine group. There were no statistically significant differences in baseline characteristics—including age, gender, ASA PS, and BMI—between the two groups (**Table 1**), indicating a homogeneous study population.

In terms of hemodynamic parameters, including SBP, DBP, heart rate, and mean arterial pressure (MAP), the group receiving inhaled 2% lidocaine

demonstrated greater hemodynamic stability compared to the group receiving intravenous 1% lidocaine during the bronchoscopy procedure. These findings were consistent with previous studies reporting that inhaled lidocaine was more effective in attenuating cardiovascular responses triggered by airway manipulation. For instance, studies by Jokar et al. and Saeed et al. demonstrated that nebulized lidocaine more effectively prevented elevations in blood pressure and heart rate during tracheal intubation compared to intravenous administration. (14,15)

The observed hemodynamic stability in the inhaled lidocaine group may be attributed to its local anesthetic effect on the airway mucosa, which effectively blocks afferent impulses from subepithelial receptors. This inhibition reduces the sympathetic response typically triggered by bronchoscope manipulation. Gaurav et al. demonstrated that the administration of inhaled lidocaine prior to induction of anesthesia significantly attenuated the post-intubation increases in SBP, DBP, heart rate, and MAP compared to a control group. (16) These findings supported the notion that inhaled lidocaine mitigates sympathetic stimulation commonly associated with bronchoscopic procedures.

The differences in hemodynamic responses between inhaled and intravenous lidocaine may also be explained from a pharmacokinetic standpoint. Inhaled lidocaine achieves higher local concentrations at the airway mucosa while maintaining lower systemic plasma levels, thereby minimizing the risk of systemic side effects. Conversely, intravenous lidocaine produces a more rapid systemic effect but lacks direct action on the airway structures subjected to instrumentation. Groeben et al. reported that inhaled lidocaine effectively inhibits bronchoconstrictive reflexes by achieving high concentrations in the airway without significantly elevating plasma levels, thus ensuring localized efficacy with a favorable safety profile. (17)

In this study, inhaled 2% lidocaine and intravenous 1% lidocaine were selected as the administered doses, considering that more than 50% of the inhaled dose is typically lost during the nebulization process. Previous studies by Gaurav et al. and Kaur et al. have demonstrated that these dosages remained within safe plasma concentration limits (i.e., <5 μg/ml) and were sufficiently effective in attenuating hemodynamic responses without causing systemic toxicity. (16,18)

In addition to its effect on hemodynamic parameters, this study also evaluated plasma norepinephrine levels as an indicator of sympathetic activation. The results showed that the inhaled lidocaine group had more stable norepinephrine levels compared to the intravenous lidocaine group at both 1 and 10 minutes after i-gel® LMA insertion (**Table 6**). The rise in norepinephrine levels typically reflects the physiological stress response to airway manipulation, such as bronchoscope insertion, which activates the hypothalamic-pituitary-adrenal (HPA) axis and the sympathoadrenal system. (19)

The use of inhaled lidocaine was shown to be more effective in suppressing this sympathetic activation. As described by Jordan et al., nebulized lidocaine was evenly distributed throughout the respiratory tract and acts directly on the airway mucosa, thereby reducing afferent sensory input to the central nervous system. This local mechanism likely contributes to the lower norepinephrine response observed in the inhaled group, reflecting reduced systemic stress and improved autonomic stability during the procedure. (20)

This study demonstrated that inhaled 2% lidocaine offered greater benefits in stabilizing hemodynamic responses and suppressing plasma norepinephrine levels compared to intravenous 1% lidocaine during bronchoscopy procedures. Its effectiveness was supported by a localized mechanism of action that attenuated sympathetic impulses while maintaining plasma concentrations within safe systemic limits. Therefore, inhaled lidocaine may serve as a safe and effective alternative for minimizing the physiological stress response associated with airway manipulation.

Strengths and limitations

A major strength of this study lay in the use of plasma norepinephrine levels as an objective biomarker of sympathetic activation, alongside a comparative evaluation of two clinically relevant lidocaine administration routes. However, the study had several limitations. Hemodynamic responses and plasma norepinephrine levels were assessed at a single lidocaine dose, limiting the ability to evaluate dose-response relationships. Additionally, plasma lidocaine concentrations were not measured, which precluded assessment of systemic drug exposure and potential correlations with observed clinical effects.

Conclusion

Inhaled 2% lidocaine is more effective than intravenous 1% lidocaine in controlling hemodynamic responses and plasma norepinephrine levels during bronchoscopy procedures using the i-gel® LMA.

Table 1. Characteristics of research subjects

Characteristics		Inhaled lidocaine	Intravenous lidocaine	p-value
Age (years), mean±SD[a]		54.52±13.29	55.87±13.01	0.730[ns]
Gender[b]	Male, n (%)	13 (56.5)	18 (78.3)	0.116[ns]
	Female, n (%)	10 (43.5)	5 (21.7)	
ASA PS class[b]	I, n (%)	0 (0.0)	1 (4.3)	0.591[ns]
	II, n (%)	13 (56.5)	13 (56.5)	
	III, n (%)	10 (43.5)	9 (39.1)	
BMI (kg/m^2), mean±SD[a]		21.55±2.95	20.75±2.94	0.365[ns]

Legend: SD=standard deviation; ASA PS=American Society of Anesthesiologists Physical Status; BMI=body mass index.
[a]Independent sample t test; [b]chi-square test; [ns]not significant.

Table 2. Comparison of systolic blood pressure between groups

Measurement time	Systolic blood pressure (mmHg), mean±SD		p-value
	Inhaled lidocaine	Intravenous lidocaine	
Before bronchoscopy procedure (T0)[a]	128.26±15.08	129.04±15.49	0.422[ns]
1-minute post bronchoscope insertion (T1)[b]	128.43±10.06	136.52±17.55	0.042[*]
10-minute post bronchoscope insertion (T2)[b]	127.48±11.63	136.65±14.87	0.024[*]
Delta T0-T1[b]	0.17±9.12	7.48±12.66	0.030[*]
Delta T1-T2[b]	-0.96±9.80	0.13±12.18	0.741[ns]
Delta T0-T2[b]	-0.78±10.28	7.61±14.95	0.032[*]

Legend: SD=standard deviation.
[a]Mann-Whitney test; [b]Independent sample t test; [ns]not significant; [*]significant.

Table 3. Comparison of diastolic blood pressure between groups

Measurement time	Diastolic blood pressure (mmHg), mean±SD		p-value
	Inhaled lidocaine	Intravenous lidocaine	
Before bronchoscopy procedure (T0)[a]	80.04±11.47	80.78±8.58	0.806[ns]
1-minute post bronchoscope insertion (T1)[b]	78.69±9.22	90.30±10.14	<0.001[*]
10-minute post bronchoscope insertion (T2)[b]	79.13±9.22	89.22±11.23	0.003[*]
Delta T0-T1[b]	-1.35±6.56	9.52±8.73	<0.001[*]
Delta T1-T2[b]	0.43±11.52	-1.09±6.92	0.590[ns]
Delta T0-T2[a]	-0.91±12.90	8.43±10.68	0.010[*]

Legend: SD=standard deviation.
[a]Mann-Whitney test; [b]Independent sample t test; [ns]not significant; [*]significant.

Table 4. Heart rate distribution between groups

Measurement time	Heart rate (beats/min), mean±SD		p-value
	Inhaled lidocaine	Intravenous lidocaine	
Before bronchoscopy procedure (T0)[a]	81.43±13.57	87.17±10.75	0.050[ns]
1-minute post bronchoscope insertion (T1)[b]	81.00±11.67	95.26±15.93	0.001[*]
10-minute post bronchoscope insertion (T2)[b]	77.61±8.30	94.74±12.24	<0.001[*]
Delta T0-T1[a]	-0.43±9.48	8.08±14.30	0.034[*]
Delta T1-T2[b]	-3.39±8.12	-0.52±12.32	0.356[ns]
Delta T0-T2[a]	-3.83±11.79	7.56±11.09	0.002[*]

Legend: SD=standard deviation.
[a]Mann-Whitney test; [b]Independent sample t test; [ns]not significant; [*]significant.

Table 5. Comparison of mean arterial pressure between groups

Measurement time	Mean arterial pressure (mmHg), mean±SD		p-value
	Inhaled lidocaine	Intravenous lidocaine	
Before bronchoscopy procedure (T0)[a]	96.11±11.69	96.87±9.23	0.404[ns]
1-minute post bronchoscope insertion (T1)[b]	95.49±8.17	104.03±12.21	0.013[*]
10-minute post bronchoscope insertion (T2)[b]	94.86±9.24	103.59±9.42	0.004[*]
Delta T0-T1[a]	-0.63±11.86	7.15±10.46	0.030[*]
Delta T1-T2[a]	-0.63±7.91	-0.44±6.67	0.933[ns]
Delta T0-T2[a]	-1.25±10.56	6.71±10.68	0.037[*]

Legend: SD=standard deviation.
[a]Mann-Whitney test; [b]Independent sample t test; [ns]not significant; [*]significant.

Table 6. Comparison of plasma norepinephrine levels between groups

Measurement time	Plasma norepinephrine level (ng/ml), mean±SD		p-value
	Inhaled lidocaine	Intravenous lidocaine	
Before bronchoscopy procedure (T0)[a]	258.07±165.57	241.81±158.04	0.621[ns]
1-minute post bronchoscope insertion (T1)[b]	264.92±169.83	358.07±185.44	0.047[*]
10-minute post bronchoscope insertion (T2)[b]	254.40±167.64	343.20±161.28	0.04[*]
Delta T0-T1[b]	6.85±14.88	116.85±93.35	<0.001[*]
Delta T1-T2[b]	-10.52±16.17	-15.46±95.12	0.733[ns]
Delta T0-T2[a]	-3.66±13.78	101.39±135.55	0.011[*]

Legend: SD=standard deviation.
[a]Mann-Whitney test; [b]Independent sample t test; [ns]not significant; [*]significant.

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