ORIGINAL ARTICLE ASIAN JOURNAL OF MEDICAL SCIENCES

A comparative study of single-dose esmolol and single-dose lignocaine in the prevention of intubation-induced tachycardia and hypertension during general anesthesia

Bandana Paudel[1], Samir Ghimire[2], Sumitra Paudel[3]

[1]Assistant Professor, [3]Medical Officer, Department of Anaesthesiology, Critical Care and Pain Management, Nobel Medical College Teaching Hospital, Biratnagar, [2]Medical Officer, Department of Internal Medicine, Kathmandu Medical College Teaching Hospital, Kathmandu, Nepal

Submission: 09-03-2022 Revision: 20-03-2022 Publication: 01-04-2022

ABSTRACT

Background: During laryngoscopy and endotracheal intubation, hemodynamic stress response is a great concern. **Aims and Objectives:** This study aimed to compare single-dose esmolol with single-dose lignocaine in the prevention of intubation induced tachycardia and hypertension during general anesthesia. **Materials and Methods:** On 60 patients, a prospective comparative study was done. Patients ranging in age from 20 to 60 years old were included in the study. Individuals with physical Status I and II as per the guidelines of the American Society of Anesthesiologists scheduled for elective surgeries under general anesthesia with endotracheal intubation were divided into two groups randomly. Two minutes before intubation, 1.5 mg/kg esmolol and 1.5 mg/kg lignocaine bolus doses were given diluted in 10 ml normal saline. Measurement of the mean heart rate (HR), the mean systolic blood pressure (SBP), and the mean diastolic blood pressure (DBP) was taken at the base level, during intubation and 1st, 2nd, 3rd, 5th, and 10th min after intubation. Furthermore, mean arterial pressure (MAP) was calculated based on these values. **Results:** The mean HR, systolic, diastolic, and mean blood pressure before starting anesthesia were similar in the lignocaine group and esmolol group ($P > 0.05$). The mean HR (79.37 ± 5.46 in lignocaine group and 74.63 ± 5.411 in esmolol group), mean SBP (127 ± 5.387 in lignocaine group and 114.50 ± 6.317 in esmolol group), mean DBP (83.07 ± 3.028 in lignocaine group and 65.10 ± 2.77 in esmolol group), and MAP (98.03 ± 2.883 in lignocaine group and 81.57 ± 2.812 in esmolol group) at intubation and 1, 2, 3, 5 and 10 min after intubation showed a significant decrease in the values in the esmolol group. **Conclusion:** Esmolol when given intravenously as a 1.5 mg/kg bolus dose is proven to be superior and efficient in dampening the vasopressor response to laryngoscopy and endotracheal intubation in comparison with lignocaine given intravenously as 1.5 mg/kg bolus dose during general anesthesia without inducing unexpected hypotension and bradycardia.

Key words: Endotracheal intubation; Esmolol; Hemodynamic response; Laryngoscopy; Lignocaine

Access this article online

Website: http://nepjol.info/index.php/AJMS

DOI:10.3126/ajms.v13i4.43723

E-ISSN: 2091-0576

P-ISSN: 2467-9100

INTRODUCTION

Throughout the practice of anesthesiology, laryngoscopy and intubation during general anesthesia form the basis of controlling the patient's airway.[1] Larynx, epipharynx, pharynx, and trachea stimulation, which are innervated by the autonomic nervous system, including parasympathetic innervation through the vagus and glossopharyngeal nerves, and sympathetic innervation through the superior cervical ganglion are all stimulated by laryngoscopy and endotracheal intubation.[1] The cardiovascular stress reactions to the laryngoscopy and the endotracheal intubation are short-lived in most of the patients and might have fewer negative effects.[2] Both laryngoscopy

Address for Correspondence:
Dr. Bandana Paudel, Assistant Professor, Department of Anaesthesiology, Critical Care and Pain Management, Nobel Medical College Teaching Hospital, Biratnagar, Nepal. **Mobile:** +977-9827314712. **E-mail:** dr.bandana.nobel@gmail.com

and endotracheal intubation are unpleasant sensations that can trigger a stress response that includes tachycardia, hypertension, and cardiac arrhythmias.[3] These types of hemodynamic responses are tolerated in a thriving manner in otherwise healthy people, but then again, they can have serious consequences in people with coronary artery disease, cerebrovascular disease, hypertension, and intracranial aneurysms, such as cerebral hemorrhage, left ventricular failure, ventricular dysrhythmias, pulmonary edema, and myocardial ischemia.[3] The stimulation of the larynx, trachea, and bronchi, increases catecholamine release, causing these potentially catastrophic extubation consequences.[4] As a result, especially in high-risk patients, thorough surveillance of the hemodynamic response to stress following extubation may be required.[5,6] Notably, it was shown that the risk of respiratory problems after extubation is greater than it was during anesthetic induction or endotracheal intubation.[7] Although numerous techniques are being used to regulate cardiorespiratory reactions to airway management throughout intubation, no standardized protocol or recommendations for preventing the cardiovascular responses during the peri-extubation phase have been identified.[8-10] Pharmaceutical therapies such as beta-blockers, N-methyl D-aspartate antagonists, local anesthetics, and alpha-2 agonists have long been recognized as efficient in reduction of the rate of undesirable consequences associated with tracheal intubation.[8-12] Throughout this context, preventative administration of the beta-blockers during the peri-extubation phase has been suggested as a possible intervention to reduce hemodynamic reactions and negative outcomes such as airway manipulation reflexes.[12]

Beta-blockers reduce endogenous sympathetic chronotropic action by binding to beta-adrenoreceptors in the sinoatrial node, resulting in a negative chronotropic impact.[13] Esmolol, a cardioselective short-acting beta-adrenergic antagonist, allows for quick titration to a desired amount of beta-blockade after delivery, making it an ideal perioperative drug.[14] It has a muscle-relaxing impact on the heart; hence, its role in the treatment of cardiovascular risk population is still unclear.[15] Esmolol has long been known to reduce reflex hypertension and arrhythmia caused by severely noxious stimulus during crucial moments of anesthesia and surgery, supporting the notion of generally controlled anesthesia. Human blood pressure, like the predominance of hypotension, rises linearly with aging.[16,17] Esmolol dosages from 0.5 to 2 mg/kg range were utilized in the prior studies. The previous research found that 1.5 mg/kg esmolol dose reduced the pulse rate, the systolic blood pressure (SBP), the diastolic blood pressure (DBP), the mean arterial blood pressure (MAP), and the rate pressure product.[18] According to Singh et al., esmolol 2 mg/kg suppresses the sympathomimetic response effectively and without negative effects. Nonetheless, there was no consensus among scholars on using an exact dosage of esmolol for cardiovascular response attenuation.[19] Lignocaine is an amide synthetic local anesthetic, which is used in the management of ventricular dysrhythmias and as a prophylactic measure in ventricular tachyarrhythmia.[1] It has cardio stabilizing action.[1] A higher threshold for pulmonary stimulation and centralized suppression of sympathetic transmissions appeared to be the basis of IV local anesthetics.[15] Increased lignocaine dosage may cause irregular heartbeat, low blood pressure, and oxygen deprivation.[15]

The reason for continuing the search for an optimum anesthetic approach that is both efficacious and useful in reducing unwanted physiological effects was presented above. Multiple approaches and medicines have been used in an attempt to obtund these unfavorable responses. The choice of a pharmaceutical adjuvant might be difficult since effectiveness must be balanced with tolerability. These two medications have shown efficiency, but only in higher dosages, and only a few studies with modest doses are reported. As a result, the purpose of the current study was to compare the effectiveness of single-dose Esmolol (1.5 mg/kg) and single-dose lignocaine (1.5 mg/kg) in the prevention of intubation induced tachycardia and hypertension during general anesthesia following laryngoscopy and endotracheal intubation in terms of heart rate (HR), SBP, DBP, and MAP.

Aims and objectives

This study aimed to compare single-dose esmolol with single-dose lignocaine in the prevention of intubation induced tachycardia and hypertension during general anesthesia.

MATERIALS AND METHODS

On 60 patients, a prospective comparative study was done in the department of Anaesthesiology, Nobel Medical College Teaching Hospital from January 2021 to January 2022 after getting ethical clearance from the Institutional Review Committee. Patients ranging in age from 20 to 60 years old were included in the study. Informed written consent was obtained. Individuals with physical status I and II as per the guidelines of the American Society of Anesthesiologists scheduled for elective surgeries with endotracheal intubation were divided into two groups randomly of 30 each, namely, Group L (Lignocaine group) and Group E (esmolol group).

Normotensive patients of either gender, of age 20–60 years were included in the study. Those with an anticipated difficult airway, pregnancy, atrioventricular block, bronchial

asthma, diabetes mellitus, or those using beta-blockers within 24 h of surgery, were excluded from the study. A comprehensive interview, physical assessment, respiratory evaluation, systematic inspection, as well as chest X-ray, routine blood tests, and an electrocardiogram (ECG) were performed on all the selected patients according to our hospital protocol. On arrival to the operation theatre, an 18G cannula was inserted into a prominent vein on the non-dominant hand of the patient. Isotonic fluid was started at the rate of 70 ml/h. Standard monitoring (ECG, HR, NIBP, and SPO2) was carried out. Induction was done with midazolam (0.02 mg/kg), fentanyl (2 mcg/kg), propofol (2.5 mg/kg), and vecuronium (0.1 mg/kg). Two minutes before intubation, 1.5 mg/kg esmolol and 1.5 mg/kg lignocaine bolus doses were given diluted in 10 ml normal saline. Measurement of the mean HR, the mean SBP, and the mean DBP was taken at the base level, during intubation and 1st, 2nd, 3rd, 5th, and 10th min after intubation. Furthermore, MAP was calculated based on these values.

Sample size calculation

The sample size calculation was done with G*power version 3.1.9.4. The study was done to compare hemodynamic instability between the two groups. Assuming the values of HR, SBP and diastolic pressure (mean difference, mean±SD of Group L, and mean±SD of Group E) at 2 min post-intubation as (5, 80±6, 75±6), (10, 120±15, 110±12), and (10, 80±12, 70±12), respectively, the sample size was found to be 24, 30, and 21, respectively, for a power of 80% and α=0.05 (two tailed). The maximum sample size of 30 in each group was chosen as the sample size for our study to achieve adequate power for all the three parameters used for assessing hemodynamic stability. HR and blood pressure were considered in the standard units of per minute and mm of Hg, respectively.

Statistical analysis

IBM SPSS v.26 was used to carry out the analysis. The baseline characteristics of the patients accompanied by the data retrieved from the variables were represented by means and standard deviation. A Chi-square test was used to analyze the demographic data. Comparison between treatment groups of esmolol (E) and lignocaine (L) was performed by independent sample t-test.

RESULTS

The average age of patients in the current study was 38.72 years. There were more total female patients (61.7%) than males (38.3%) with 54.1% females in the lignocaine group and 45.9% in the esmolol group. However, male patients were equally distributed among both the groups as being mentioned in Table 1.

Table 2 demonstrates the recording time of HR showing patients in the lignocaine group having a higher mean HR (79.37±5.461) than the esmolol group (74.63±5.411) after 5 min of intubation. Similarly, after 10 min of the intubation process, a drop in the mean HR of the esmolol treated group was noticed (68.13±4.023) in comparison with the lignocaine group (76.00±5.065). A mean difference was found between both groups with P=0.001 and <0.001 after 5 and 10 min of intubation, respectively.

As shown in Table 3, mean SBP in the lignocaine treated group was found to be much higher than the esmolol treated group at every stage of recording. Thus, acquired results showed differences among both the groups with P<0.05 showing the efficiency of esmolol dosage.

Following this, when mean DBP was being measured, there was not a great difference between both groups at the start of intubation. Although, there was a mean difference found after 1, 2, 3, 5, and 10 min after intubation as elaborated in Table 4.

In addition, at the baseline level, the MAP was the same in both groups. However, just like mean DBP a significant mean difference was noticed in the MAP showing that the esmolol treated group was more effective than the lignocaine group. These details are summarized in Table 5.

DISCUSSION

A short but considerable autonomic and sympathetic-adrenal activation is induced by laryngoscopy and endotracheal intubation, resulting in hypertension and tachycardia.[1] In normotensive patients, these potentially harmful alterations

Table 1: Characteristics of the patients

Parameters	Groups		Total	P-value
	Group L	Group E		
	Mean±S.D			
Age (years)	39.63±8.323	37.80±8.240	38.72±8.263	0.395
Gender		Frequency (percentage)		0.213
Female	17 (45.9)	20 (54.1)	37 (61.7)	
Male	13 (50)	10 (50)	23 (38.3)	

Group L: Lignocaine group, Group E: Esmolol group, S.D: Standard Deviation

Table 2: Comparison of the mean heart rate (HR) between the groups

Recording time of heart rate	Group L	Group E	P-value (Group E vs. Group L)
	Mean±S.D		
BaseValue	83.77±8.645	84.73±8.081	0.656
During intubation	80.00±8.910	80.83±7.461	0.696
1 min AI	77.80±8.134	79.07±7.100	0.523
2 min AI	86.20±6.435	88.17±6.899	0.258
3 min AI	82.97±5.398	81.97±6.729	0.528
5 min AI	79.37±5.461	74.63±5.411	0.001
10 min AI	76.00±5.065	68.13±4.023	<0.001

Group L: Lignocaine group, Group E: Esmolol group, AI: After Intubation, S.D: Standard Deviation

Table 3: Comparison of mean systolic blood pressure between the groups

Recording time of systolic blood pressure	Group L	Group E	P-value (Group E vs. Group L)
	Mean±S.D		
Base value	132.57±7.342	130.80±8.984	0.408
During intubation	128.83±7.354	124.53±8.349	0.039
1 min AI	124.90±7.434	119.70±8.163	0.012
2 min AI	138.13±7.123	125.37±6.785	<0.001
3 min AI	133.13±6.621	118.97±6.636	<0.001
5 min AI	127.87±5.387	114.50±6.377	<0.001
10 min AI	125.27±6.142	109.77±7.745	<0.001

Group L: Lignocaine group, Group E: Esmolol group, AI: After Intubation, S.D: Standard Deviation

Table 4: Comparison of mean diastolic blood pressure between the groups

Recording time of diastolic blood pressure	Group L	Group E	P-value (Group E vs. Group L)
	Mean±S.D		
BaseValue	82.10±3.754	80.43±4.083	0.105
During intubation	77.87±3.608	76.33±4.046	0.127
1 min AI	74.80±3.595	72.87±3.721	0.045
2 min AI	92.67±4.188	77.73±3.140	<0.001
3 min AI	87.73±3.151	69.70±1.765	<0.001
5 min AI	83.07±3.028	65.10±2.771	<0.001
10 min AI	79.43±2.909	61.33±2.171	<0.001

Group L: Lignocaine group, Group E: Esmolol group, AI: After Intubation, S.D: Standard Deviation

resolve after 3 min of laryngoscopy.[1] High blood pressure individuals, on the other hand, require considerable time to recover from alterations in cardiovascular measures, making them more vulnerable to consequences such as pulmonary edema, left ventricular dysfunction, ventricular dysrhythmias, myocardial infarction, and brain hemorrhage. In hypertensive individuals, this has been the most common reason for a decreased cardiovascular reaction following laryngoscopy and tracheal intubation.[1] Overall timeframe of laryngoscopy and intubation, the type of instruments used, the anesthetic medication employed, and the degree

Table 5: Comparison of mean arterial pressure between the groups

Recording time of mean arterial pressure	Group L	Group E	P-value (Group E vs. Group L)
	Mean±S.D		
Base Value	98.97±4.382	97.17±5.032	0.145
During intubation	95.07±4.402	92.70±4.956	0.055
1 min AI	91.57±4.207	88.70±4.879	0.018
2 min AI	108.10±4.566	94.10±4.130	<0.001
3 min AI	102.90±3.346	86.20±2.709	<0.001
5 min AI	98.03±2.883	81.57±2.812	<0.001
10 min AI	94.60±2.931	77.47±3.170	<0.001

Group L: Lignocaine group, Group E: Esmolol group, AI: After Intubation, S.D: Standard Deviation, The baseline characteristics of all parameters, HR (83.77±8.645 in Group L and 84.73±8.081 in Group E), SBP (132.57±7.342 in Group L and 130.80 ±8.984 in Group E), DBP (82.10±3.754 in Group L and 80.43±4.083 in Group E), and MAP (98.97±4.382 in Group L and 97.17±5.032 in Group E) were similar in both groups. HR: Heart rate, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, MAP: Mean arterial pressure

of anesthesia are all variables that impact the severity of cardiovascular alterations.[1]

The current study investigated both esmolol and lignocaine efficacy in preventing intubation-induced tachycardia and hypertension during general anesthesia by evaluating the mean HR, the mean SBP, the mean DBP, and the mean MAP. This is a standard dose of lignocaine generally used in these indications.[8,20] At the starting level, there was not much difference found between both the esmolol and lignocaine groups but during the process of intubation, there was a rise in the mean SBP and the MAP within the lignocaine group while maintenance of the overall blood pressure was seen in the esmolol group. After 1 min into intubation, the overall blood pressure and MAP gradually rose and with each passing minute, it kept rising in the lignocaine group. Statistically, a significant mean difference was observed in the systolic, diastolic, and arterial blood pressure in response to which HR level increased after the 5th and 10th min of intubation process in lignocaine treated group while esmolol treated group maintained the blood pressure by lowering it.

The present study's result was corresponding with research done in 2019 showing esmolol being more effective than lignocaine in preventing life-threatening hemodynamic response.[14] They found an increase in mean HR in the lignocaine group which stayed increased till the 5th min after intubation.[14] Just like in our study, the esmolol group had reduced mean HR throughout intubation.[14] Similarly, the MAP was also decreased in the esmolol group during and 1 and 2 min after intubation in the above-mentioned study. Another comparative study conducted in Gwalior assessed the efficacy of esmolol with lignocaine and labetalol, which conveyed that esmolol dosage reduced the hemodynamic

stress response better than lignocaine.[15] The effectiveness of esmolol was notified in multiple pieces of literature. A Brazilian research study concluded that esmolol-treated patients had a lower rate of tachycardia (2.2%) than placebo-treated patients (48.9%) and P=0.002.[19] Esmolol group also had a lower rate of hypertension (4.4% vs. 31.1%, with a P=0.004) [19]. Patients who took esmolol had a better extubating quality than patients who got a placebo (P=0.001).[21] In a Turkish study, esmolol infusion helps to the idea of generalized controlled anesthesia among elective patients planned for surgical intervention, both in young (age 18–35) and older (aged 65+) patients. Among both the groups, the esmolol treated group provided perioperative hemodynamic consistency, satisfactory anesthetic restoration, and a low risk of adverse reactions.[22] In a clinical trial, 1.5 mg/kg esmolol and 0.25 mg/kg labetalol bolus doses were given 2 min before extubating and it was concluded that at extubating and immediately post-extubation, esmolol was more proficient than labetalol.[23] Talwar et al., found that after the laryngoscopy and the endotracheal intubation, 1.5 mg/kg esmolol besides the esmolol plus diltiazem bolus doses both were found to be successful in decreasing mean HR, mean SBP and mean DBP, and MAP.[24] Sharma et al.,[25] found that in comparison with the control group, 1.5 mg/kg dose of esmolol and dexmedetomidine both were reducing cardiovascular stress

Table 6: Different studies comparing esmolol and lignocaine in attenuation of the hemodynamic response to intubation

S. No.	Year of study	Author et al.	Sample size	Drugs with doses	Primary outcome	Mean differences of the primary outcome	P-value
1.	2011	Begum et al.[1]	120	1.5 mg/kg Esmolol & 1.5 mg/kg Lignocaine	Heart rate changes 4 min post intubation ,it was significantly lower in Esmolol group	4.33 (1.55-7.10)	0.002
					Mean arterial pressure changes 4 min post intubation, it was significantly lower in Esmolol group	4.61(2.25-6.10)	0.000
					Rate pressure product changes 4 min post intubation, it was significantly lower in Esmolol group	789.22(434.25-1144.20	<0.0001
2.	2013	Singh et al.[2]	120	1.5 mg/kg lidocaine (L), Esmolol (E) 2 mg/kg	Heart rate changes at 1 min post intubation, it was significantly lower in Esmolol group	17.80 (21.24–14.37)	<0.000
					Mean arterial pressure changes at 1 min post intubation, it was significantly lower in Esmolol group	6.50 (8.59–4.40)	<0.0001
					Rate pressure product changes at 1 min post intubation , it was significantly lower in Esmolol group	2778 (3386.25–2169.75)	<0.0001
3.	2014	Shrestha et al.[3]	60	1.5 mg/kg lidocaine, Esmolol 1.5 mg/kg	Systolic blood pressure changes after intubation, it was significantly lower in Esmolol group	3.45 (15.77–8.87)	0.574
					Diastolic blood pressure changes after intubation, it was significantly lower in Esmolol group	1.20 (9.09–11.49)	0.814
					Mean arterial pressure changes after intubation, it was significantly lower in Esmolol group	0.050 (11.14–11.24)	0.992
					Heart rate changes after intubation, it was significantly lower in Esmolol group	24 (35.32–12.68)	0.000
4.	2021	Muralidharan et al.[4]	52	2 mg/kg lidocaine, Esmolol 2 mg/kg	Heart rate changes at 3 min after intubation, it was significantly lower in Esmolol group	26.26(22.58–30)	<0.000
					Mean arterial pressure changes at 3 min after intubation, it was significantly lower in Esmolol group	20 (17.75–22.25)	<0.001
5.	2022	Mulimani et al.[5]	60	1.5 mg/kg Esmolol and 1.5 mg/kg Lignocaine	Heart rate changes 5 min after intubation, it was lower in Esmolol group	29.37 (25.10–32.80)	<0.000
					Mean arterial pressure changes 5 min after intubation, it was lower in Esmolol group	14.07 (11.17–16.10)	<0.000
					Rate pressure product changes 5 min after intubation, it was lower in Esmolol group	4251 (4215.55–4286.45)	<0.000

in response to intubation. Direct cardiac depression and peripheral vasodilation are mechanisms by which lignocaine reduces the physiological responses during endotracheal intubation.[14] It possesses analgesic and antiarrhythmic effects.[14] In the Marulasiddappa and Nethra[26] research, 1.5 mg/kg bolus dose of lignocaine was found to be incapable of maintaining blood pressure and HR in neurological patients when compared to clonidine. In our study, we chose a dose of 1.5 mg/kg esmolol over a dose of 1.5 mg/kg of lignocaine, and we observed substantial outcomes when compared to the previous studies.

According to the literature, phlebitis was the sole negative consequence of esmolol, which could be prevented with proper dilution.[26-29] In the current research, we found no adverse effects. Following laryngoscopy and endotracheal intubation, we noticed a mean difference in HR and MAP between esmolol and lignocaine treated groups, as seen in the previous studies. However, no complications have been discovered during the research.

Similar studies have been conducted in the past (Table 6); however, no consensus has been reached regarding the drug, the dose and timing of its delivery. This type of study also has not been done in any institute in our region and areas around it. Despite lack of conclusion on the best agent, lidocaine is still the most commonly used agent in our hospital set up. Hence, in this study, we have taken single dose esmolol (1.5 mg/kg) and single dose lidocaine (1.5 mg/kg) given 2 min before intubation for comparison on their efficacy in prevention of laryngoscopy and intubation induced tachycardia and hypertension during general anesthesia in elective surgical cases.

Limitations of the study

The current research has some limitations. During this prospective comparative study, authors were unable to measure catecholamine levels in the blood. We only had looked at variations in normotensive individuals, not those with concomitant conditions. As a result, more research is needed to determine the medication's efficiency with precise plasma levels, to minimize vasodilatory responses during the laryngoscopy and the intubation in high-risk cases.

CONCLUSION

Esmolol, a beta-adrenergic antagonist, ultra-short acting, cardio-selective drug, when given intravenously as 1.5 mg/kg bolus dose, is proven to be efficient in dampening the vasopressor response to laryngoscopy and endotracheal intubation in comparison with lignocaine given intravenously as 1.5 mg/kg bolus dose during general anesthesia without inducing unexpected hypotension and bradycardia.

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Authors Contribution:
BP- Concept and design of the study, prepared first draft of manuscript, reviewed the literature; **SG-** Interpreted the results, statistical analysis and interpretation, preparation of manuscript, coordination; **SP-** Concept, reviewed the literature, preparation of manuscript and revision of the manuscript

Work attributed to:
Nobel Medical College Teaching Hospital, Biratnagar, Nepal.

Orcid ID:
Dr. Bandana Paudel - https://orcid.org/0000-0001-8225-0983
Dr. Samir Ghimire - https://orcid.org/0000-0002-0519-6191
Dr. Sumitra Paudel - https://orcid.org/0000-0003-2017-3278

Source of Support: None, **Conflicts of Interest:** None.