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A clinical study to compare the effectiveness of dexmedetomidine in attenuating sympathoadrenal response induced by laryngoscopy and endotracheal intubation in smokers versus non-smokers

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ABSTRACT

Background: The process of laryngoscopy and tracheal intubation is potent stressful stimuli that cause sympathetic activation, which is transient. Some of the modalities for attenuation of laryngoscopic and intubation response have been found to be less effective in smokers than in non-smokers. In view of this, the present study was performed to compare the effectiveness of dexmedetomidine in male smoker and non-smoker patients. **Aims and Objectives:** The aim of this study was to compare the effectiveness of dexmedetomidine in suppressing sympathoadrenal pressor response due to laryngoscopy and endotracheal intubation between smokers and non-smokers. **Materials and Methods:** This was a hospital-based non-randomized controlled study. Sixty patients were divided into two groups: 30 patients with no history of smoking (Group NS) and 30 patients with a smoking history (Group S). Both groups were received dexmedetomidine 0.75 mcg/kg over 10 min intravenous infusion. Hemodynamic parameters and Ramsay sedation score were measured at perioperative period. **Results:** Group S patients showed a significant rise in heart rate (HR), systolic blood pressure (SBP), diastolic BP (DBP), mean arterial pressure (MAP), and rate pressure product (RRP) during the immediate post-intubation period, but Group NS showed a decrease in HR, SBP, DBP, MAP and RRP throughout the post-intubation period. **Conclusion:** Single dose of 0.75 mcg/kg IV dexmedetomidine given over a period of 10 min before the induction of anesthesia is completely effective in attenuating the hemodynamic responses associated with laryngoscopy and intubation in non-smokers, but it is not efficient in smokers.

Key words: Smoker; Non-smoker; Dexmedetomidine; Laryngoscopy; Sympathoadrenal response

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INTRODUCTION

Laryngoscopy and tracheal intubation are one of the most noxious stimuli that arouse a brief but marked sympathetic response manifesting as an increase in heart rate (HR) and blood pressure (BP). These fluctuations are maximum immediately after intubation and last for 5–10 min, which may be well-endured by American Society of Anesthesiologist (ASA) 1 and 2 physical status patients. This stress response to laryngoscopy is an important concern for anesthesiologist. There is an altered cardiovascular physiological reflex response, which is mediated by vagus and glossopharyngeal cranial nerves, activates vasomotor center to cause the release of adrenaline and noradrenaline by peripheral adrenal sympathetic response.[1] In patients with cardiovascular disease and cerebrovascular disease,

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this change may lead to life-threatening complications including myocardial ischemia, acute heart failure, and cerebrovascular accident.[2] Nicotine which is present in the cigarette smoke has acute and chronic cardiovascular effects, mainly through sympathetic activation.[3] The sympathetic activation induced by smoking depends on an increased release and a decreased clearance of catecholamines at neuroeffector junctions. Smokers, being prime candidates for cardiovascular disease, may have more chances of myocardial infarction after an exaggerated intubation responses.[4-8] Smokers have been shown to exhibit heightened upper airway reflex responses to physical and chemical stimulation. It has been proposed that subepithelial receptors are more exposed in smokers than in non-smokers, this is due to the changes induced by chronic smoking in the upper airway epithelium. These changes in smokers have been found to be responsible for exaggerated sympathoadrenal response to laryngoscopy and intubation. Some of the modalities for attenuation of laryngoscopic and intubation response have been found to be less effective in smokers than in non-smokers.[8] Dexmedetomidine is an imidazole derivative and a highly selective alpha-α2-adrenergic receptor agonist α2-agonists produce decreased systemic noradrenaline release which results in attenuation of sympathoadrenal responses, this leads to hemodynamic stability. With this above knowledge, we have planned to compare the effectiveness of dexmedetomidine in attenuating sympathoadrenal response induced by laryngoscopy and endotracheal intubation in male smoking and non-smoking patients.

Aims and objectives

To compare the effectiveness of dexmedetomidine in suppressing sympathoadrenal pressor response due to laryngoscopy and endotracheal intubation between smokers and non smokers.

MATERIALS AND METHODS

After approval by the Institutional Ethics Committee, this non-randomized controlled study was conducted in 60 normotensives, the ASA physical status Class 1 and 2 male patients in the age group of 18–50 years. A written informed consent was taken from 30 male patients with a history of smoking and 30 male patients with no history of smoking, who required general endotracheal anesthesia for elective surgeries.

Patients with the following conditions were excluded from the study: Hypertension, diabetes, hypovolemia, body mass index of >30 $kg.m^{-2}$, anticipated difficult airway, physically dependent on narcotics, and known drug allergy to any drug, those who were having cerebrovascular, neurologic, respiratory, hepatic, or renal disease, patients who were on drugs (beta blockers, antidepressants, anxiolytics, anticonvulsant, or antipsychotics), and in those patients in whom laryngoscopy time exceeded 15 s.

All patients were evaluated 1 day before the surgery. Patients in both groups were instructed nil per oral 8 h before the surgery and received Capsule Omeprazole 20 mg orally and Tablet Alprazolam 0.5 mg orally, as a premedication at night before surgery. Patient groups were divided into two, Group S (n=30) – Patients who were smokers and Group NS (n=30) – Patients who were non-smokers. Smokers were defined as those patients who have smoked at least 2 years, and consumed a minimum of 10 cigarettes or 7–8 beedies per day.

On the day of elective surgery, anesthesia machine and circuits were checked. Before induction, working laryngoscopes with appropriate-size endotracheal tubes and working suction apparatus were kept ready. All anesthetic and emergency drugs were loaded into labeled syringes and kept ready for use. 30 min before the surgery, an 18-gauge intravenous (IV) cannula was secured to gain vascular access in the pre-operative room. In the operating room, they were connected to standard monitors like non-invasive BP, Pulse oximetry, electrocardiogram, and baseline readings were recorded before administration of drug. IV fluid normal saline was started to administer through the secured IV access. Then, all the patients were administered with 0.75 μg/kg dexmedetomidine over 10 min as slow IV infusion, after diluting the dose (10 μg/ml in 10 mL syringe).

HR, systolic BP (SBP), diastolic BP (DBP), mean arterial pressure (MAP), rate pressure product (RPP), oxygen saturation, and sedation score (using Ramsay sedation score) were measured at 1 min, 2 min, 5 min, and 10 min after administration of dexmedetomidine. All the patients were pre-oxygenated for 3 min with 100% oxygen. General anesthesia technique was standardized for both groups. Then, patients were induced intravenously with propofol 2 mg/kg until loss of the verbal contact and fentanyl l 2 μg/kg. After assessing the ease of mask ventilation, 0.1 mg/kg IV vecuronium was administered. Endotracheal intubation was performed by trained anesthesiologist with a minimum 3 years of experience in the field of anesthesiology, using Macintosh laryngoscope blade 4 and high volume low pressure cuffed disposable endotracheal tube size according to the patient. After confirmation of tracheal intubation by checking bilateral air entry, the tube was secured and anesthesia was maintained with O_2 and N_2O in a ratio of 50% each and 2% inhaled sevoflurane. Ventilator parameters were adjusted to maintain end-tidal carbon dioxide concentration between 35 and 40 mm of Hg.

Hemodynamic parameters such as HR, SBP, DBP, MAP, RPP, and sedation score (by using Ramsay sedation score) were measured at pre-induction (post-dexmedetomidine) at 1, 2, 5, and 10 min and at 0, 1, 2, 3, and 4 min after post-induction, and 1, 2, 5, and 10 min after post-intubation, in the rest of intraoperative period at 10 min interval and in the post-operative period 1, 2, 5, 10 min and at 20 min interval. At the end of the surgery, patients were reversed with injection neostigmine 0.05 mg/kg and glycopyrrolate 0.01 mg/kg intravenously. The patients were extubated when awake and breathing adequately and shifted to recovery.

Any hypotension (SBP fall >30% from baseline) was treated with a titrated dose of IV mephentermine 6 mg, and the incidence of bradycardia (HR <50 beats/min) was treated with atropine 0.6 mg. Time of administration of rescue analgesic in the post-operative period was noted.

Ethics

The study was approved by the Institutional Ethics Committee of Mandya Institute of Medical Sciences, Mandya with IEC No: MIMS/IEC/2018/272.

Statistics

Sample size was calculated from the past medical records of elective surgical patients who required general endotracheal anesthesia in Mandya Institute of Medical Science for the past 1 year, we found that around 2–3 patients/month with a history of smoking and 4–5 patients with no history of smoking satisfied our study inclusion criteria.

Purposive sampling was used for the sampling method, Group S (n=30) – Patients who were smokers, and Group NS (n=30) – Patients who were non-smokers included in our study.

All the collected data were entered in the Microsoft Excel software and analyzed using the Statistical Package for the Social Sciences (SPSS) version 17.0 software (SPSS, Inc., Chicago, IL). Categorical data were presented as frequencies and percentages, and quantitative data were presented as mean and standard deviation. Quantitative data were analyzed by student's t-test (independent sample's t-test) to know the difference between means. P<0.05 was considered statistically significant.

RESULTS

Demographic data were similar in both groups were shown in Table 1. None of the patients allocated were excluded from the study. No adverse events occurred in either of the group.

Table 1: Demographic details among smokers and non-smokers of the study subjects

Demography	Smokers mean±SD	Non-smokers mean±SD	P-value
Age	38.23±8.88	34.43±10.57	0.137
BMI	22.32±2.20	22.91±2.63	0.353

*P<0.05 (statistically significant), BMI: Body mass index, SD: Standard deviation

In smokers, the basal mean HR was 80.43±7.62 bpm, and persistent increases in HR were observed post-intubation 0, 1, and 2 min, that was 89.00±6.46 bpm, 86.47±5.53 bpm and 84.7±6.37 bpm, respectively, representing an increase in HR 9 bpm (11%), 6.04 bpm (7.5%), 4.27 bpm (5.3%), respectively, from the basal mean HR and all are statistically significant.

In non-smokers, the basal mean HR was 86.47±7.38 bpm, Post-intubation 0, 1 and 2 min, that was 80.40±7.9 bpm, 79.17±5.87 bpm and 77.00±5.72 bpm, respectively, representing a decrease in HR 6.07 bpm (7%), 7.3 (8.4%), 9.47 (10.9%) respectively from the basal mean HR values and all were statistically significant (Figure 1).

Statistical evaluation between the smoker and non-smoker groups showed that maximum HR changes were observed 0, 1, and 2 min post-intubation, those values are statistically significant (P<0.001).

In smokers, the basal mean SBP was 135.10±10.07 mm of Hg, an increase in SBP was observed post-intubation 0 min and 1 min, that was 145.03±7.70 mm of Hg, 136.90±10.95 mm of Hg, representing an increase in mean SBP 10 mm of Hg (7.4%), 1.8 mm of Hg (1.3%) from baseline mean SBP and post-intubation 5 min, 10 min values were 130.63±15.01 mm of Hg, 125.60±15.83 mm of Hg, and 123.27±15.68 mm of Hg, respectively, showing a decrease in mean SBP 5 mm of Hg 5 (3.7%), 9.4 mm of Hg (6.9%), 11.73 mm of Hg (8.6%), respectively, from the basal mean SBP and all are statistically significant. In non-smokers, the basal mean SBP was 132.73±9.27 mm of Hg, post-intubation at 0, 1, 2,5, and 10 min, that is 122.43±11.58 mm of Hg, 120.53±9.58 mm of Hg, 116.30±11.33 mm of Hg, 114.00±11.45 mm of Hg, 113.27±12.51 mm of Hg, respectively, representing a decrease in SBP 10.3 mm of Hg (7.7%), 12.23 mm of Hg (9.2%), 16.4 mm of Hg (12.3%), 18.73 mm of Hg (14%),19.46 mm of Hg (14.6%), respectively, from the basal mean SBP values and all are statistically significant (Figure 2).

Statistical evaluation between the smoker and non-smoker groups showed that Maximum mean SBP changes were observed 0, 1, 2, 5, and 10 min post-intubation, those values are statistically significant (P<0.001).

In smokers, the basal mean DBP was 83.23±5.89 mm of Hg, An increase in DBP was observed post-intubation 0, 1, 2, 5, and 10 min, that was 87.53±10.66 mm of Hg, 83.17±9.67 mm of Hg, 81.33±6.85 mm of Hg, 77.77±9.75 mm of Hg and 75.47±10.90 mm of Hg, respectively, representing an increase in Mean DBP 4.3 mm of Hg (5.1%), initially, followed by a decrease in mean DBP 0.06 mm of Hg (0.07%), 1.9 mm of Hg (2.2%), 5.46 mm of Hg (6.5%), and 7.76 (9.3%), respectively, from the basal mean DBP and all are statistically significant. In non-smokers, the basal mean DBP was 82.47±8.50 mm of Hg, decrease in DBP were observed post-intubation 0, 1, 2,5, and 10 min, which were 77.03±9.16 mm of Hg, 75.87±8.36 mm of Hg, 72.20±10.54 mm of Hg, 69.97 10.14 mm of Hg, 68.70±11.58 mm of Hg, respectively, representing a decrease in DBP 5.4 mm of Hg (6.5%), 6.6 mm of Hg (8%), 10.27 mm of Hg (12.4%), 12.5 mm of Hg (15%), 13.7 mm of Hg (16.6%), respectively, from the basal mean DBP values and all are statistically significant (Figure 3).

Statistical evaluation between the study groups showed that maximum means DBP changes were observed 0-, 1-, 2-, 5-, and 10-min post-intubation; those values are statistically significant (P<0.001).

In smokers, the basal mean MAP was 90.27±9.62 mm of Hg, An increase in MAP was observed post-intubation 0, 1, 2, 5, and 10 min, that was 95.40±4.12 mm of Hg, 95.20±3.08 mm of Hg, 87.63±1.22 mm of Hg, 85.03±1.96 mm of Hg and 77.03±1.22 mm of Hg, respectively, representing an increase in mean MAP 5.13 mm of Hg (5.6%), 4.93 mm of Hg (5.4%) at 0 and 1 min, followed by a decrease in mean MAP 2.64 (2.9%), 5.23 mm of Hg (5.7%), and 13.24 mm of Hg (14.6%), respectively, from the basal mean MAP at 2, 5 and 10 min and all are statistically significant. In non-smokers, the basal mean MAP was 88.23±11.07 mm of Hg, decrease in MAP were observed post-intubation 0, 1, 2, 5, and 10 min, that was 87.6±3.03 mm of Hg, 86.63±1.71 mm of Hg, 81.30±2.53 mm of Hg, 77.07±1.23 mm of Hg and 73.83±1.94 mm of Hg, respectively, representing a decrease in Mean MAP 0.6 mm of Hg (0.06%), 1.6 mm of Hg (1.8 %), 6.93 mm of Hg (7.8%), 11.16 mm of Hg (12.6%), and 14.4 mm of Hg (16.3%), respectively, from the basal mean MAP with P<0.01, all are statistically significant (Figure 4).

Statistical evaluation between the study groups showed that maximum mean MAP changes were observed 0-, 1-, 2-, 5-, and 10-min post-intubation and postoperatively (post-extubation) 1, 2, 5, 10, and 20 min, respectively, those values are statistically significant (P<0.05).

In smokers, the basal mean RPP was 10848.13±1372, an increase in RPP were observed post-intubation 0, 1, and 2 min, which were 12959.37±1082, 11846.07±1314, and 11063.27±1673, respectively, representing an increase in mean RPP 2111 (19.4%), 998 (9.1%), 215 (1.5%), followed by a decrease in mean RPP from the baseline, which was 9953.93±1893 and 9820.80±1934, respectively, representing a decrease in mean RPP 895 (8.2%) and 1028 (9.4%), respectively, from the basal mean RPP. Intraoperative 20 min, 40 min, 60 min, 1 h 20 min, values were 9780.27±1582, 9552.13±1444, 9609.93±1547, 9676.31±1638, 8899.25±692, respectively, with P<0.05 and all are statistically significant. In non-smokers, the basal mean RPP was 11318.00±1362, decrease in mean RPP was observed post-intubation 0, 1, 2, 5, and 10 min, that was 9782.27±1231, 9562.57±1093, 8965.40±1164, 8679.43±1323, and 8485.33±1419, respectively, representing a decrease in mean RPP 1536 (13.5%), 1756 (15.5%), 2353 (20.7%), 2639 (23.3%), and 2833 (25%), respectively, from the basal mean RPP with P<0.05, all are statistically significant (Figure 5).

Statistical evaluation between the study groups showed that maximum means RPP changes were observed 0-, 1-, 2-, 5-, and 10-min post-intubation, those values are statistically significant (P<0.01).

DISCUSSION

In patients with underlying cardiovascular disease and cerebrovascular disease, this sympathoadrenal response due to laryngoscopy and tracheal intubation can cause serious complications such as myocardial ischemia, acute heart failure and cerebrovascular accident. This sympathoadrenal response has been found to be more profound in smokers than nonsmokers. This exaggerated sympathoadrenal response in smokers has been found to be due to nicotine and carbon monoxide. Nicotine will stimulate adrenal medulla to release adrenaline which in turn leads to increase in sympathetic tone & increase in heart rate, increase in blood pressure. Increase in heart rate and myocardial contractility leading to increase in oxygen demand of cardiac muscles. Myocardial oxygen supply decreases due to increase in coronary vascular resistance, further cut offs the myocardial oxygen supply:demand ratio.

There are various studies, shown that α2-Adrenergic agonists mainly clonidine and dexmedetomidine have been found to have better effects than other pharmacological modalities of attenuation of intubation responses. Gulsoy k et al,[7] found that Intravenous dexmedetomidine, better suppressed the hemodynamic changes in chronic male smokers compared to fentanyl. Gulsoy k et al,[7] Sung et al,[9]

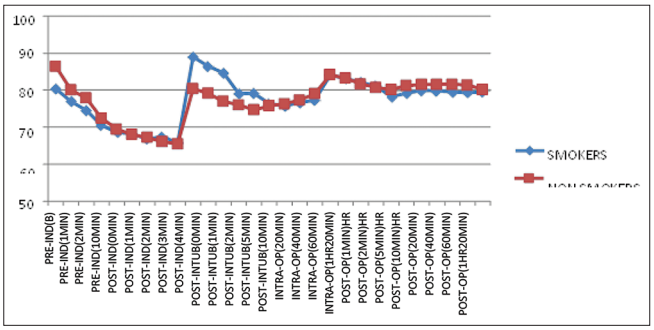

Figure 1: Diagram depicting distribution according to mean heart rate among smokers and non-smokers of the study subjects

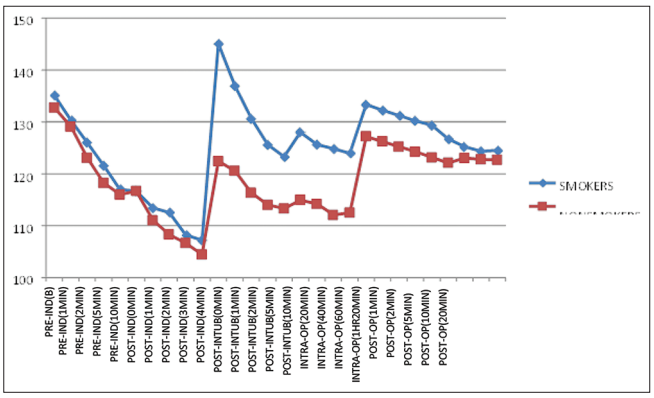

Figure 2: Diagram depicting distribution according to mean systolic blood pressure among smokers and non-smokers of the study subjects

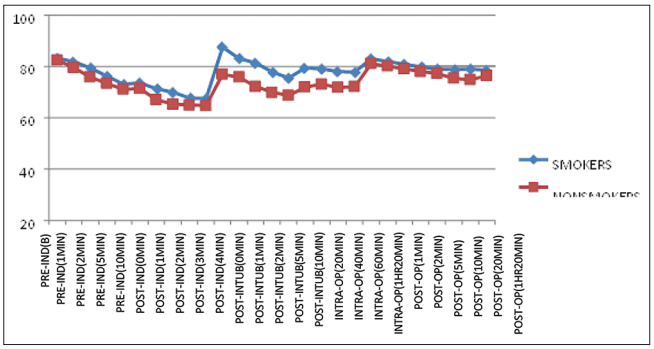

Figure 3: Diagram depicting distribution according to mean diastolic blood pressure among smokers and non-smokers of the study subjects

Cuvas et al[10] and Selvaraj.V & Manoharan K R,[11] concluded that premedication with dexmedetomidine at a dosage of 1 µg.kg-1 attenuated the sympathoadrenal responses of laryngoscopy and intubation adequately. Gulabani et al,[12] Kumari et al,[13] Lee CW and Kim M,[14] were found that pre-anesthetic dose of 0.5µg.kg-1 dexmedetomidine effectively suppressed the hemodynamic responses to endotracheal intubation.

Sebastian B, Talikoti AT, Krishnamurthy D,[15] suggested that dexmedetomidine in a dose of 0.75 µg.kg-1 intravenous is the optimal dose to attenuate stress response to laryngoscopy and endotracheal intubation. N Sadanandam and K Vijay Kumar,[16] were concluded that dexmedetomidine at 0.75 µg.kg-1 dose was found superior to 0.5 µ.kg-1 dose with respect to hemodynamic stability so this dose should be used if it is permissible with respect to sedation.

Laxton et al,[4] concluded that heart rate of smokers immediately after intubation was significantly greater than that of nonsmokers similar to our study response. Sebastian B, Talikoti AT, Krishnamurthy D,[15] proved that dexmedetomidine 0.75 µg.kg-1 attenuated the hemodynamic stress response to laryngoscopy and endotracheal intubation completely compared to 0.5 µg. kg-1 in his study and the HR recorded was below the baseline values ,which is concurred with decline in the heart rate seen in our nonsmoker study results.

Gulsoy k et al,[7] observed that increase in SBP in the immediate post intubation time in smokers, even after administration of dexmedetomidine (1 µg.kg-1) which was above the baseline value and followed by a decremental response in SBP which is similar to our smoker group results. Kumari et al,[13] observed that in the dexmedetomidine group, a slight increase was observed in SBP after intubation when compared to pre intubation values; however, when compared to baseline, there was a statistically significant decrease in blood pressure post intubation, which persisted for 5 minutes ($P < 0.001$) similar to non smokers group in our study. Smoking elevates the blood pressure and the heart rate due to its strong adrenergic agonist effect, but abstinence from smoking during a period of preparation for surgery leads to reduction in the plasma catecholamine level, various hemodynamic responses. Kumari et al,[13] showed that significant decrease in DBP in the dexmedetomidine group until 5 minutes post intubation compared to baseline values as seen in our nonsmoker group. Gulsoy k et al,[7] observed that an increase in DBP in the immediate post intubation period in smokers followed by decrease in mean DBP in the remaining post intubation time intervals in the dexmedetomidine which is similar to our smoker group study results. Sung et al,[9] observed that in male smokers, the MAP just after endotracheal intubation was significantly increased as compared with the baseline MAP and at 1 minutes after intubation, similar to our study results in smokers.

Rate pressure product is an indicator of myocardial oxygen requirement, it's a multiplication product of heart rate and systolic blood pressure. It's a measure of cardiac work load. A total value more than 10000 indicates an increased risk of heart disease. Coronary artery disease are more in chronic smokers than nonsmokers. Increases in rate-pressure product can even further increase the myocardial

oxygen requirement in smokers. Cuvas et al.,[10] suggested that smoking history should be assessed for patients as a part of preoperative evaluation, so that we can identify high risk patients. Gulsoy k et al,[7] observed that an increase in RPP in the immediate post intubation period in smokers followed by decremental trend in the rest of the post intubation period which is similar to our.

Limitations of the study

We included only male patients with ASA physical status I and II, study cannot be applied for patients with higher ASA physical status and difficult airways and female patients. Invasive blood pressure monitoring was not used which would have provided us a better comprehension giving us beat-to-beat recording of the parameters. This was not performed due to cost constraints.

CONCLUSION

Single dose of 0.75 mcg/kg IV dexmedetomidine given over a period of 10 minutes prior to the induction of anaesthesia is completely effective in attenuating the hemodynamic responses associated with laryngoscopy and intubation in nonsmokers, but it is not efficient in smokers.

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Authors' Contributions:
G; Topic selection, Literature survey, Prepared first draft of manuscript, implementation of study protocol, **KM**; data collection, data analysis, manuscript preparation and submission of article, **MCB**; Concept, design, clinical protocol, editing, and manuscript revision, Design of study, statistical Analysis and Interpretation, **NP**; Review Manuscript, Coordination and Manuscript revision.

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