Section: Anesthesiology

Original Research Article

STUDY TO DETERMINE THE EFFECT OF ORAL PREGABALIN AS PREMEDICATION ON HEMODYNAMIC RESPONSE TO LARYNGOSCOPY, ENDO-TRACHEAL INTUBATION, AND CARBO-PERITONEUM DURING ELECTIVE LAPAROSCOPIC SURGERIES UNDER GENERAL ANAESTHESIA

S.Balasubramanian[1], A.Gnanavel Rajan[2], M.Sivasankari[1]

[1]Assistant Professor, Department of Anesthesiology, Government Medical College, Ramanathapuram, Tamilnadu, India
[2]Associate Professor, Department of Anesthesiology, Government Medical College, Ramanathapuram, Tamilnadu, India

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Corresponding Author:
Dr. M.Sivasankari,
Email: dr.siva10.9@gmail.com

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ABSTRACT

Background: Laryngoscopy, intubation, and CO_2 pneumoperitoneum activate the sympathetic nervous system, resulting in increased heart rate (HR) and arterial pressure. Pregabalin premedication may attenuate these hemodynamic changes in laparoscopic surgery. This study evaluated whether oral pregabalin 150 mg reduces pressor responses during laryngoscopy, intubation, and early pneumoperitoneum. **Materials and Methods:** This prospective interventional study included 86 ASA I–II patients aged 19–65 years scheduled for elective laparoscopic surgery at PSG Institute of Medical Sciences and Research, Coimbatore. Patients were randomly allocated to Group A (control) or Group B (pregabalin 150 mg given orally 1–2 hours before laryngoscopy). Hemodynamic parameters were recorded at baseline, after induction, post-intubation, and during early pneumoperitoneum. Group differences were analysed using independent t-test and chi-square test, with $p<0.05$ considered significant. **Result:** Baseline characteristics were comparable between groups. Group B demonstrated significantly lower HR at pre-induction (77.7 vs. 93 bpm; p=0.000), one minute after induction (76.79 vs. 87.84 bpm; p=0.000), and one and five minutes after intubation (80.26 vs. 93.14 bpm; p=0.000; 77.37 vs. 86.28 bpm; p=0.001). Systolic and diastolic pressures were significantly lower in Group B during induction and post-intubation intervals. Mean arterial pressures were significantly reduced at baseline (88.66 vs. 95.10 mmHg; p=0.013), pre-induction (93.40 vs. 101.05 mmHg; p=0.030), and early post-intubation phases, with later pneumoperitoneum values showing no significant differences. **Conclusion:** Pregabalin 150 mg premedication attenuated increases in HR and blood pressure during laryngoscopy, intubation, and early pneumoperitoneum in ASA I–II patients undergoing elective laparoscopic surgery, supporting its role in improving peri-intubation hemodynamic stability.

INTRODUCTION

Laryngoscopy and endotracheal intubation are essential procedures to secure the airway during general anaesthesia, preventing aspiration of gastric contents and secretions. These interventions ensure patient safety throughout the perioperative period. Airway manipulation provokes a well-known sympathetic pressor response, resulting in increased heart rate (HR) and arterial blood pressure. Although usually brief, this response may become significant in individuals with cardiovascular, respiratory, or neurological disease.[1] Carbon dioxide (CO_2) insufflation used to establish pneumoperitoneum in laparoscopic surgery influences circulatory dynamics by raising systemic vascular resistance and mean arterial pressure (MAP). The coexistence of airway instrumentation and pneumoperitoneum during laparoscopy heightens the risk of exaggerated sympathetic stimulation.[2] Laparoscopic surgery now represents a major proportion of elective abdominal procedures worldwide, increasing the importance of optimizing perioperative hemodynamic stability.

Hemodynamic effects of CO_2 insufflation have been demonstrated in small clinical studies. In one evaluation involving seven patients, increases in HR, MAP, and central venous pressure corresponded with

rising $PaCO_2$ levels and reduced pH following insufflation.[3] A study observed that laryngoscopy alone caused measurable increases in plasma catecholamines, and this reaction intensified when tracheal intubation followed.[4]

Several pharmacological agents have been assessed to attenuate this sympathetic response. Dexmedetomidine lowered HR and blood pressure during intubation and surgery while reducing anaesthetic and opioid requirements.[5] Gabapentin premedication decreased the stress response to laryngoscopy without significantly affecting HR.[6] Pregabalin, a Gamma-Amino Butyric Acid analogue, acts as an α2-δ ligand with analgesic, anxiolytic, and anticonvulsant properties. By binding to the α2-δ subunit of voltage-gated calcium channels, it reduces the release of excitatory neurotransmitters such as noradrenaline, glutamate, and substance P. It does not completely block calcium channels, even at higher doses. These pharmacologic characteristics indicate potential to blunt the sympathetic response associated with airway manipulation and pneumoperitoneum.[7] Evidence from clinical research supports this possibility. Oral Pregabalin 150 mg limited the rise in blood pressure and HR during the first attempt at intubation and produced higher sedation scores than lower doses.[8] Pregabalin was also more effective than Gabapentin in reducing pressor responses.[9] Another study reported that Pregabalin 150 mg given one hour before induction safely reduced hemodynamic variability during laryngoscopy.[10] However, current literature remains limited regarding its influence during laparoscopic procedures that involve concurrent CO_2 insufflation. We hypothesised that preoperative oral pregabalin would attenuate the increases in HR and arterial blood pressure associated with laryngoscopy, endotracheal intubation, and CO_2 insufflation. Therefore, this study aimed to determine whether preoperative oral Pregabalin can attenuate hemodynamic responses to laryngoscopy, endotracheal intubation, and CO_2 insufflation in patients undergoing elective laparoscopic surgery under general anaesthesia.

MATERIALS AND METHODS

This prospective interventional study was conducted on 86 patients undergoing elective laparoscopic surgeries under general anaesthesia in the Department of Anaesthesiology, PSG Institute of Medical Sciences and Research, Coimbatore, over two years from December 2019 to December 2021. Written informed consent was obtained from all participants, and ethical approval was granted by the Institutional Human Ethics Committee prior to study initiation.

Sample size calculation: The sample size was calculated using the MAP at one minute after induction, with 80% power and 5% significance. Using Zβ = 0.84, Zα = 1.96, and SD = 16.73, the study required 86 patients, with 43 subjects allocated to each group, where formula $n = 2 \times (Z\alpha + Z\beta)^2 \times SD^2 / (mt - mc)^2$.[11]

Inclusion and exclusion criteria

Adults aged 19–65 years, ASA I–II, scheduled for elective laparoscopic surgery under general anaesthesia, who provided consent and had no expected difficult airway, were included.

Patients <19 and >65 years age, classified as ASA III or higher, had major organ disease, an anticipated difficult airway, prolonged or repeated intubation, allergies to pregabalin or anaesthetic drugs, used sedatives or anticonvulsants, had cognitive issues or dizziness, were pregnant, required emergency surgery, or declined consent were excluded.

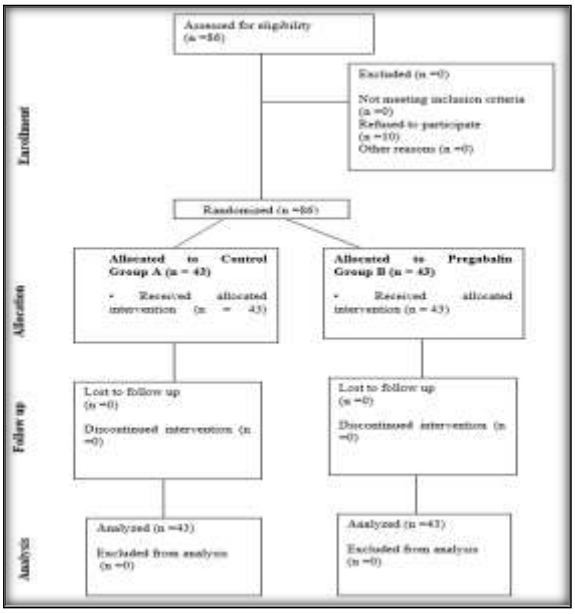

Figure 1: Consort diagram

Methods: Participants were randomly allocated into two equal groups of 43 using computer-generated randomisation. Group A served as the control, while Group B received a single oral dose of Pregabalin 150 mg as premedication. The anaesthesiologist performing laryngoscopy and intubation was blinded to group assignment. HR, systolic, diastolic and MAP, along with intubation time, were recorded at baseline, before induction, one minute after induction, one and five minutes after intubation, and one, five and ten minutes after CO_2 insufflation.

All patients underwent routine pre-anaesthetic evaluation and adhered to fasting guidelines (8 hours for solids, 6 hours for milk/liquids and 2 hours for clear fluids). Premedication included Pantoprazole 40 mg the night prior and Pantoprazole 40 mg plus Metoclopramide 10 mg two hours before induction. In the operating theatre, ECG, non-invasive blood pressure and pulse oximetry were applied, and an intravenous line was secured.

Pre-oxygenation was given for three minutes with 100% oxygen. Induction was achieved with Fentanyl 2 µg/kg and Propofol 2 mg/kg IV, followed by Atracurium 0.5 mg/kg to facilitate intubation. After three minutes, direct laryngoscopy and endotracheal

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intubation were performed by the blinded anaesthesiologist. Anaesthesia was maintained with Sevoflurane and ventilation was adjusted to maintain $EtCO_2$ between 30–40 mmHg. Muscle relaxation was maintained with intermittent Atracurium boluses. At the completion of surgery, neuromuscular blockade was reversed using Neostigmine 0.05 mg/kg and Glycopyrrolate 0.01 mg/kg, and extubation was performed when adequate spontaneous ventilation returned.

The primary outcome was the MAP response to laryngoscopy, intubation and CO_2 insufflation. Secondary outcomes included changes in HR, systolic and diastolic blood pressures at predefined measurement intervals.

Statistical analysis: All data were entered into Microsoft Excel 2019 and analysed using SPSS version 22.0. Continuous variables were presented as mean ± standard deviation, and categorical variables as frequency and percentage. Group comparisons were performed using the independent t-test for continuous variables and Pearson's chi-square test for categorical variables. A p-value < 0.05 was considered statistically significant.

RESULTS

A total of 86 participants were included, with 43 patients in each group. Baseline characteristics such as age (38.88 ± 12.85 vs. 40.79 ± 13.16 years; p = 0.499) and weight (63.98 ± 9.51 vs. 66.98 ± 11.31 kg; p = 0.187) were comparable. Females predominated in both groups (60.5% vs. 69.8%; p = 0.365). Mallampati Class II was more frequent in Group B (81.4% vs. 51.2%; p = 0.003), while Cormack–Lehane Grade I views were similarly distributed across groups (62.8% vs. 53.5%; p = 0.382). ASA II patients were slightly more common in Group B (58.1% vs. 44.2%; p = 0.196). Intubation times were comparable (17.47 ± 2.12 vs. 17.26 ± 2.61 sec; p = 0.684) [Table 1].

Table 1: Baseline demographic and clinical characteristics

Variable		Group A (n = 43)	Group B (n = 43)	P value
Age (years)		38.88 ± 12.85	40.79 ± 13.16	0.499
Gender	Male	17 (39.5%)	13 (30.2%)	0.365
	Female	26 (60.5%)	30 (69.8%)	
Weight (kg)		63.98 ± 9.51	66.98 ± 11.31	0.187
Mallampati Class	I	21 (48.8%)	8 (18.6%)	0.003
	II	22 (51.2%)	35 (81.4%)	
Cormack–Lehane Grade	I	27 (62.8%)	23 (53.5%)	0.382
	II	16 (37.2%)	20 (46.5%)	
ASA Physical Status	I	24 (55.8%)	18 (41.9%)	0.196
	II	19 (44.2%)	25 (58.1%)	
Intubation time (sec)		17.47 ± 2.12	17.26 ± 2.61	0.684

Group A showed higher HR than Group B at several points: pre-induction (93 vs. 77.70; p = 0.000), one minute after induction (87.84 vs. 76.79; p = 0.000), one minute after intubation (93.14 vs. 80.26; p = 0.000), and five mins after intubation (86.28 vs. 77.37; p = 0.001). Following CO_2 insufflation, HR at one minute (78.00 ± 11.11 vs. 75.93 ± 12.49; p = 0.419), five mins (75.86 ± 12.79 vs. 77.95 ± 14.43; p = 0.479) and ten mins (76.81 ± 12.60 vs. 76.00 ± 12.17; p = 0.761) were comparable between the two groups [Figure 1].

Figure 2: Comparison of HR between groups at different time points

Group A showed higher systolic pressures than Group B at key points: one minute after induction (109.4 vs. 99.35 mmHg, p = 0.007), one minute after intubation (127.35 vs. 106.65 mmHg, p = 0.001), five mins after intubation (111.42 vs. 98.37 mmHg, p = 0.001), and one minute after CO_2 insufflation (119.37 vs. 110.16 mmHg, p = 0.015) [Figure 2].

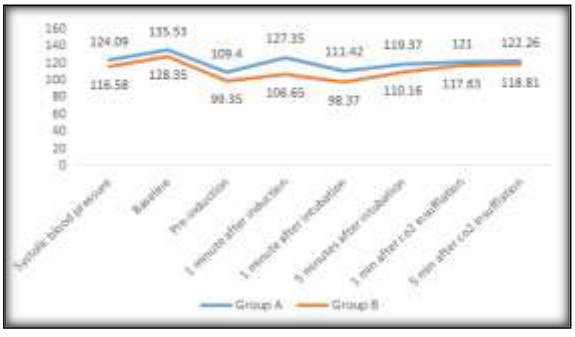

Figure 3: Comparison of systolic blood pressure (SBP) between groups at different time points

Group A showed higher diastolic pressures than Group B at the significant time points: baseline (80.60 vs. 74.70 mmHg; p = 0.004), pre-induction (83.81 vs. 75.93 mmHg; p = 0.003), one minute after induction (67.63 vs. 62.44 mmHg; p = 0.032), one minute after intubation (80.84 vs. 68.63 mmHg; p =

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0.001), and five mins after intubation (70.07 vs. 62.09 mmHg; p = 0.007). Other time points showed no significant differences between the groups [Figure 3].

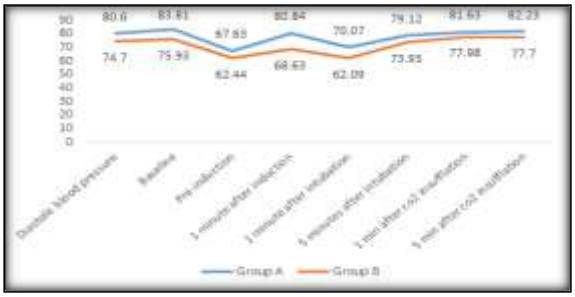

Figure 4: Comparison of diastolic blood pressure (DBP) between groups at different time points

Group A had higher MAP than Group B at all significant points: baseline (95.10 vs. 88.66 mmHg; p = 0.013), pre-induction (101.05 vs. 93.40 mmHg; p = 0.030), one minute after induction (81.55 vs. 74.74 mmHg; p = 0.008), one minute after intubation (96.34 vs. 81.30 mmHg; p = 0.001), five mins after induction (83.85 vs. 74.19 mmHg; p = 0.002), and one minute after CO_2 insufflation (92.53 vs. 86.01 mmHg; p = 0.030) [Figure 4].

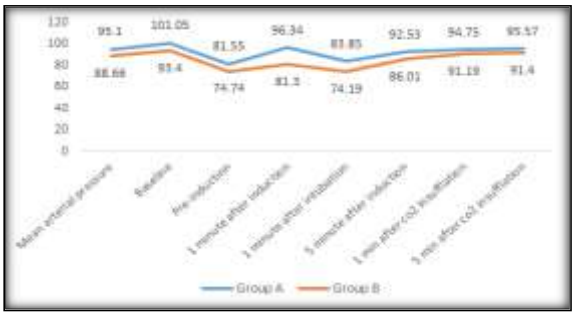

Figure 5: Comparison of MAP between groups at different time points

DISCUSSION

This study observed whether oral Pregabalin given as premedication can reduce the hemodynamic stress response to laryngoscopy, endotracheal intubation, and CO_2 pneumoperitoneum during elective laparoscopic surgery under general anaesthesia. Airway manipulation and pneumoperitoneum normally activate the sympathetic system, increasing HR and blood pressure, which may be risky in vulnerable patients. In our study, Pregabalin significantly limited the rise in HR, systolic and DBP, and MAP during induction and intubation. This stabilising effect continued into the early pneumoperitoneum phase, though later values were similar between groups. Overall, Pregabalin appears to be a useful premedication for maintaining peri-intubation hemodynamic stability.

Baseline characteristics were similar between groups, including demographics and airway assessments, with no differences in intubation conditions. Similarly, Rastogi et al. reported similar baseline profiles across placebo, pregabalin 75 mg, and 150 mg groups, with mean ages 36.82, 39.79, and 37.42 years; weights 59.75, 60.23, and 57.93 kg; and higher ASA II proportions in the pregabalin groups.[12] These findings show similar baseline profiles support that later hemodynamic changes were due to pregabalin and not patient differences.

Pregabalin reduced HR increases during airway manipulation, but HR were similar between groups once pneumoperitoneum was recognised. Similarly, Reddy and Murari demonstrated that pregabalin reduced HR over time, but clonidine achieved a greater reduction in early post-intubation periods, with lower HR at baseline (79.54 ± 6.34 vs. 82.52 ± 6.30 bpm) and at 1, 3, and 5 mins after intubation (87.78 ± 6.04 vs. 92.06 ± 6.32 bpm; 86.68 ± 6.53 vs. 88.88 ± 6.83 bpm; 79.20 ± 6.49 vs. 82.06 ± 5.47 bpm; all p < 0.05).10 Sangeetha et al. pregabalin reduced HR more effectively, with values falling below baseline by 10 mins (83.60 ± 9.25 bpm), while the control group remained elevated even at 15 mins (89.75 ± 9.94 bpm).[13] Sandill et al. reported that pregabalin consistently produced lower HR after intubation compared with placebo, most particularly at 1 minute (78.4 vs. 92.74 bpm), and continued to show lower values at 3, 5, 7, and 9 mins (77.83 vs. 75.33 bpm; 75.63 vs. 73.0 bpm; 71.73 vs. 70.68 bpm; 70.2 vs. 69.9 bpm).[14] These studies show that Pregabalin limits HR rises during airway manipulation, helping control sympathetic activation and maintain stable hemodynamics.

Pregabalin reduced systolic and diastolic pressure rises during airway manipulation, while pressures became comparable between groups after CO_2 insufflation. Similarly, Bhandari et al. pregabalin maintained lower systolic pressures (137.30–139.23 mmHg) and diastolic pressures (86.57–88.93 mmHg) from laryngoscopy to 10 mins after intubation than placebo (SBP 148.57–154.20 mmHg; DBP 94.43–97.00 mmHg), with comparable baseline values.[15] In contrast to our study, Agrawal et al. in Group A (oral pregabalin 150mg), SBP rose to 122.30 ± 5.76 mmHg at two mins after intubation and returned toward baseline by four mins (117.23 ± 6.07 mmHg).[16] These studies show Pregabalin lowers blood pressure after intubation, supporting our findings, though one study reported only a brief SBP rise before quick stabilisation.

Pregabalin lowered MAP during airway manipulation and early pneumoperitoneum, while MAP became similar between groups later in insufflation. Similarly, Chaudhary found that MAP in the pregabalin group remained below baseline throughout anaesthesia (81.8–88.1 mmHg) with only small differences from clonidine, and both groups showed similar MAP recovery after extubation (96.10 vs 94.98 mmHg).[17] Gupta et al. in the pregabalin group, MAP remained closer to baseline and significantly lower after laryngoscopy and pneumoperitoneum, such as at intubation (109.5 ± 6.7 vs. 116.9 ± 6.3 mmHg) and at 5 mins' post-insufflation (107.5 ± 6.6 vs. 119.5 ± 7.0 mmHg),

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showing faster stabilisation and reduced sympathetic response compared with controls.[18] These studies show that pregabalin controls MAP during airway manipulation and early pneumoperitoneum, supporting its role in reducing sympathetic stress, though effects lessen as surgery progresses. The reduction in group differences during the later pneumoperitoneum phase may reflect a delayed autonomic adjustment to increased intra-abdominal pressure and steady-state CO_2 absorption, leading to more uniform cardiovascular responses once ventilation stabilises.

This study used computer-generated randomisation, blinded intubation assessment, and a standardised anaesthetic protocol, which reduced measurement bias and ensured that hemodynamic differences reflected the pharmacological effect of pregabalin rather than procedural variation.

Limitations: This study included only ASA I–II patients at a single centre, used a single pregabalin dose, and monitored hemodynamics for a limited duration without assessing sedation or stress markers. The prospective randomized design and standardized anaesthetic protocol support the validity of observed peri-intubation responses. Pregabalin 150 mg appears clinically useful for attenuating HR and blood pressure surges during laryngoscopy and early pneumoperitoneum. Future studies should evaluate higher-risk populations, additional dosing strategies, extended monitoring periods, and biochemical stress responses to further define its perioperative role.

CONCLUSION

Pregabalin 150 mg given preoperatively reduced rises in HR and blood pressure during laryngoscopy, intubation, and the early period of pneumoperitoneum in patients undergoing elective laparoscopic surgery. Intubation conditions were unaffected, and no adverse hemodynamic effects were observed. These findings support the role of pregabalin as a useful premedication to improve peri-intubation stability in ASA I–II patients. Further research should include higher-risk groups, varied dosing, longer intraoperative monitoring, and biomarkers to clarify its broader perioperative value.

REFERENCES

1. Lakhe G, Pradhan S, Dhakal S. Hemodynamic response to laryngoscopy and intubation using McCoy laryngoscope: A descriptive cross-sectional study. JNMA J Nepal Med Assoc 2021;59:554–7. https://doi.org/10.31729/jnma.6752.
2. Jiang R, Sun Y, Wang H, Liang M, Xie X. Effect of different carbon dioxide (CO2) insufflation for laparoscopic colorectal surgery in elderly patients: A randomized controlled trial: randomised controlled trial. Medicine (Baltimore) 2019;98:e17520. https://doi.org/10.1097/MD.0000000000017520.
3. Umar A, Mehta KS, Mehta N. Evaluation of hemodynamic changes using different intra-abdominal pressures for laparoscopic cholecystectomy. Indian J Surg 2013;75:284–9. https://doi.org/10.1007/s12262-012-0484-x.
4. Kumar BSM, Prameela VS, Chhabria S, Brijesh K, Murthy TNS. A randomised double-blind comparative study of intravenous magnesium sulphate and intravenous lignocaine for attenuation of haemodynamic response to laryngoscopy and tracheal intubation. Medplus Res Publ. https://medpulse.in/Anesthsiology/html_17_2_8.
5. Bajwa SJS, Kaur J, Singh A, Parmar S, Singh G, Kulshrestha A, et al. Attenuation of pressor response and dose sparing of opioids and anaesthetics with pre-operative dexmedetomidine. Indian J Anaesth 2012;56:123–8. https://doi.org/10.4103/0019-5049.96303.
6. Yousef Abd-Allah MY, Abd-Allah MYY, Ghareeb NAEG. Efficacy of preoperative oral gabapentin for patients undergoing intracranial surgery: Effect on laryngoscopic stress response. Egypt J Anaesth 2020;36:90–6. https://doi.org/10.1080/11101849.2020.1777518.
7. Li Z, Taylor CP, Weber M, Piechan J, Prior F, Bian F, et al. Pregabalin is a potent and selective ligand for α(2)δ-1 and α(2)δ-2 calcium channel subunits. Eur J Pharmacol 2011;667:80–90. https://doi.org/10.1016/j.ejphar.2011.05.054.
8. Sundar AS, Kodali R, Sulaiman S, Ravullapalli H, Karthekeyan R, Vakamudi M. The effects of preemptive pregabalin on attenuation of stress response to endotracheal intubation and opioid-sparing effect in patients undergoing off-pump coronary artery bypass grafting. Ann Card Anaesth 2012;15:18–25. https://doi.org/10.4103/0971-9784.91473.
9. Waikar C, Singh J, Gupta D, Agrawal A. Comparative study of oral gabapentin, pregabalin, and clonidine as premedication for anxiolysis, sedation, and attenuation of pressor response to endotracheal intubation. Anesth Essays Res 2017;11:558–60. https://doi.org/10.4103/aer.AER_34_17.
10. Reddy MS, Murari N. Efficacy of pregabalin premedication before surgery in attenuating the adverse hemodynamic response to laryngoscopy and tracheal intubation. Int J Adv Med 2019;6:769. https://doi.org/10.18203/2349-3933.ijam20191447.
11. Gupta K, Bansal P, Gupta PK, Singh YP. Pregabalin premedication - A new treatment option for hemodynamic stability during general anaesthesia: A prospective study. Anesth Essays Res 2011;5:57–62. https://doi.org/10.4103/0259-1162.84192.
12. Rastogi B, Gupta K, Gupta PK, Agarwal S, Jain M, Chauhan H. Oral pregabalin premedication for attenuation of haemodynamic pressor response of airway instrumentation during general anaesthesia: A dose response study. Indian J Anaesth 2012;56:49–54. https://doi.org/10.4103/0019-5049.93344.
13. Sangeetha RP, Ramachandraiah R. Evaluation of the effect of preemptive oral pregabalin in the attenuation of cardiovascular response to direct laryngoscopy and endotracheal intubation during general anaesthesia: A randomised placebo-controlled trial. Karnataka Anaesth J 2018;16:58–65. https://doi.org/10.4103/kaj/2018/v16i3-4/157768.
14. Sandill S, Khuvung M, Singh R, Baruah U. Evaluation of oral pregabalin premedication for attenuation of haemodynamic response during tracheal intubation in adult patients with controlled hypertension for elective surgery. Arch Anesthesiol Crit Care 2024;10(Suppl 2):582–9. https://aacc.tums.ac.ir/index.php/aacc/article/view/949/876.
15. Bhandari G, Mitra S, Shahi KN, Rani A, Chauhan A. Pre-emptive oral pregabalin attenuates pressor response to laryngoscopy and endotracheal intubation: A double-blind randomised placebo-controlled trial. Ann Int Med Dent Res 2016;2:110–4. https://www.aimdrjournal.com/wp-content/uploads/2021/11/28.
16. Agrawal S, Goyal S, Audichya PC, Kumar V, Jangir KG, Chouhan RS, et al. Evaluation of the effect of oral pregabalin (150 mg) premedication in attenuation of hemodynamic responses to laryngoscopy and intubation in patients undergoing general anaesthesia. JCDR 2024;15:4270–6. https://doi.org/10.48047/.
17. Chaudhary A. Oral premedication with pregabalin and clonidine for hemodynamic stability during laryngoscopy: a comparative study. Int J Basic Clin Pharmacol 2015:1. https://doi.org/10.5455/2319-2003.ijbcp20150423.
18. Gupta K, Sharma D, Gupta PK. Oral premedication with pregabalin or clonidine for hemodynamic stability during laryngoscopy and laparoscopic cholecystectomy: A comparative evaluation. Saudi J Anaesth 2011;5:179–84. https://doi.org/10.4103/1658-354X.82791.

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