Original Article

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Anaesthesia Section

Effects of Intravenous Dexmedetomidine and 2% Lignocaine on Perioperative Haemodynamic Stability and Postoperative Recovery Profile in Laparoscopic Cholecystectomy: A Randomised Double-blind Controlled Trial

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ABSTRACT

Introduction: Laryngoscopy and endotracheal intubation induces significant sympathetic stress response, leading to tachycardia and hypertension which can be often deleterious, particularly in laparoscopic surgeries. Several pharmacological agents have been evaluated in order to reduce this response. Dexmedetomidine, a selective alpha 2-adrenergic agonist, and intravenous lignocaine have been shown to have some effects on the haemodynamic responses during surgery.

Aim: To compare the effectiveness of intravenous dexmedetomidine and intravenous lignocaine on perioperative haemodynamic stability and postoperative recovery profile in laparoscopic cholecystectomy.

Materials and Methods: This randomised double-blind controlled trial, was conducted in the Department of Anaesthesia, in SRM Medical College Hospital and Research Centre, Chennai, Tamil Nadu, India, over a period of eight months, from April 2025 to December 2025. which included 90 patients undergoing elective Laparoscopic cholecystectomy under general anaesthesia and randomly allocated into three groups- Group D-Dexmedetomidine, Group L-Lignocaine (2%), Group P-Placebo (0.9% Normal saline) of 30 patients in each group. Haemodynamic parameters were taken at baseline, induction, intubation, intraoperatively and shortly after intubation. The intubation response was assessed as the change from baseline {changes in Heart Rate (ΔHR), Systolic BP (SBP), Diastolic BP (DBP), Mean Arterial Pressure (MAP)}. Factors evaluated after surgery were extubation time, Visual Analogue Scale (VAS) pain scores and Richmond Agitation-Sedation Scale (RASS). Statistical analysis was performed using One-way Analysis of Variance (ANOVA) for intergroup comparison. Non parametric variables were analysed using the Kruskal-Wallis test and categorical variables were compared using Chi-square test. A p-value of <0.05 was considered statistically significant.

Results: Baseline characteristics were similar with mean age was 43.7±4.3 years in dexmedetomidine group, 41.1±3.8 years in lignocaine group and 43.0±5.1 years in placebo group (p=0.06). During laryngoscopy and intubation, the HR was significantly less in the dexmedetomidine group (84.7±4.7 beats/min) than in the lignocaine (90.3±6.2) and placebo groups (91.2±6.1) (p<0.01). MAP also was lower with dexmedetomidine 96.9±5.6 mmHg than lignocaine 99.1±5.2 mmHg and placebo 100.6±6.0 mmHg (p=0.04). Early postoperative sedation (RASS at 0 minutes) was significantly higher in dexmedetomidine group (p=0.02).

Conclusion: Intravenous dexmedetomidine was better than lignocaine and placebo in blunting the haemodynamic response due to laryngoscopy and endotracheal intubation. Dexmedetomidine was also associated with better perioperative haemodynamic stability with acceptable postoperative recovery profile.

Keywords: Adrenergic agonist, Endotracheal intubation, General anaesthesia, Hypertension, Laryngoscopy, Postoperative Pain, Tachycardia, Visual analogue scale

INTRODUCTION

Laparoscopic cholecystectomy has become the preferred method for a numerous abdominal surgeries because of the benefits associated with it, such as reduced rates of morbidity, reduced postoperative pain and hospital stay compared to open surgery techniques [1]. However, the laparoscopy technique requires the use of pneumoperitoneum, which has a significant impact on sympathetic nervous system activation. The introduction of CO2 to the abdomen leads to increase in plasma catecholamine levels and also increased systemic vascular resistance, leading to hypertension and tachycardia [2]. In addition, manipulation of the airway during induction especially the laryngoscopy and endotracheal intubation will cause a rapid sympathoadrenal reflex [3]. This reflex will cause temporary but observable increase of BP and HR due to a catecholamine release [4]. Such autonomic haemodynamic fluctuations can be dangerous especially to patients suffering from cardiovascular and cerebrovascular conditions [4].

Due to this pressor response from laryngoscopy, myocardial ischemia or arrhythmias may sometime occur in susceptible patients. Therefore, a variety of pharmacological interventions have been investigated. Intravenous lignocaine (lidocaine) and α_2 agonists are commonly used to reduce the effect of the airway reflex. Lignocaine has systemic pain relief and appropriately regulates heart rhythm when given intravenously [3]. Additionally, lignocaine also has anti-inflammatory and analgesic effects working at a central level [4]. The administration of intravenous lignocaine at a dose of 1-1.5 mg/kg two to three minutes before intubation stabilises the nerve membranes so there is a lower trigeminocardiac

and sympathetic response [5] . Meta-analyses suggest its efficacy to decrease the post-laryngoscopy Blood Pressure (BP) and HR compared to placebo [6]. Lignocaine is associated with a decrease in MAP following intubation [7]. In addition, it reduces the rate of postoperative opioid use, results in less postoperative pain and nausea and enables faster return to oral intake and bowel function, which is critical in reducing the duration of hospital stay [8].

While there are alternatives such as opioids and beta blockers, they are accompanied by side-effects. The search for an ideal drug continues, one with minimal sedation and airway complications. Dexmedetomidine, a selective alpha 2-adrenergic agonist, like intravenous lignocaine have been shown to have some effects on the haemodynamic responses during surgery, although data for their comparison are still limited [9]. It reduces central sympathetic outflow and catecholamine release, managing stress reactions to surgery and intubation [9]. Dexmedetomidine induces sedation and pain relief by acting on α_2-receptors in the locus coeruleus and spinal cord, allowing patients to stay awake without significant respiratory depression [10]. It also has properties that reduce the need for opioids and lessen the release of neuroendocrine stress hormones [11].

In practice, dexmedetomidine is given as an intravenous (i.v.) infusion either before or during anaesthesia to ease induction and recovery [12]. The primary side-effects are cardiovascular; the medication may slightly decrease HR and BP through sympatholysis.In laparoscopic cholecystectomy, intravenous dexmedetomidine has been shown to achieve a haemodynamic stability. A dose of 0.6 ug/kg before induction decreases HR and BP fluctuations during and after pneumoperitoneum [12]. Patients who are given this dosage also have less coughing after waking up and also have less postoperative pain levels and would require less pain medications [12]. Dexmedetomidine has potent sympatholytic and sedative actions and i.v. lignocaine has antiarrhythmic and analgesic actions which may help improve bowel recovery and reduce the need for postoperative pain management [13,14].

Despite the affordability and established safety of lignocaine, it is not known which agent is superior in terms of haemodynamic stability and postoperative recovery. Hence, it was hypothesised that dexmedetomidine would be superior to lignocaine in attenuating haemodynamic responses to laryngoscopy and intubation. The primary outcome was reduction of the haemodynamic response to laryngoscopy and intubation as measured by HR, SBP, DBP and MAP. Secondary outcomes were time to extubation, postoperative levels of pain measured with the VAS and postoperative sedation levels assessed with the RASS.

MATERIALS AND METHODS

This double-blinded, randomised controlled trial was conducted in the Department of Anaesthesia in SRM Medical College Hospital and Research Centre, located in Chennai, Tamil Nadu, India, over a period of eight months conducted from April 2025 to December 2025. The study was approved by the Institutional Ethics Committee (SRMIEC-ST0825-2785) and informed consent was obtained from all participants. Registration of trial was done in the Clinical Trial Registry of India (CTRI/2025/10/095744). Informed consent was obtained in writing from all participants before they were enrolled and the study was performed in accordance with the principles of the Declaration of Helsinki

Sample size calculation: Estimation was performed using effect size (f) of 0.446, an alpha error probability of 0.05 and power of 95%, a sample size of 81 patients was determined. To compensate for 10%, dropout rate, a total of 90 patients were recruited, consisting of 30 in each group.

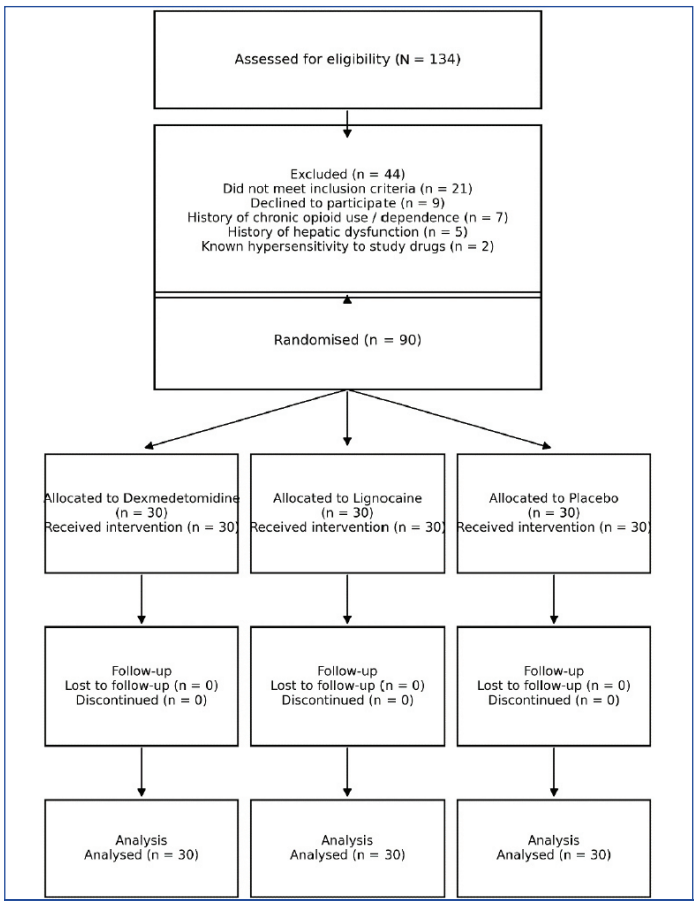
Inclusion and Exclusion criteria: Eligible participants were adults aged 18-65 years with American Society of Anaesthesiologists (ASA) physical status I-II, scheduled for elective laparoscopic cholecystectomy lasting < 2 hours. Patients with baseline bradycardia (HR <60 beats/min) or MAP <65 mmHg were excluded from the study. In addition, those who were known to have hypersensitivity to dexmedetomidine or lignocaine, had a history of chronic opioid use or opioid dependency, or had significant hepatic dysfunction which might affect drug metabolism, patients who refused to participate or were not able to provide an informed consent were also excluded from the study. A total of 134 patients were studied for eligibility, of which 44 patients were excluded. Of those excluded, 21 of the patients failed to meet the inclusion criteria; 9 patients declined to participate; 7 patients were opioid dependence: 5 were excluded due to hepatic dysfunction and 2 patient previous history of hypersensitivity reaction to study drugs.

Remaining 90 patients were randomised in three groups where each group consisted of 30 patients-

- Dexmedetomidine group (n=30),
- Lignocaine group (n=30) and
- Placebo group (n=30)

All allocated participants received the intervention allocated to them. During the follow-up period, no patients were lost to follow-up and no patients stopped the intervention during the follow-up period in any group. Therefore, all the randomised patients (n=90) were considered in the final analysis with 30 patients studied in each group.

Ninety patients who met the inclusion criteria were randomly assigned into three groups of 30 patients each (1:1:1). Randomisation was done using a computer-generated random allocation sequence [Table/Fig-1]. A senior anaesthetist not involved in the study performed allocation and enrolled the participants.The concealment was ensured by use of sealed opaque envelopes. The study was performed in a double blind manner in which no one, including the patients and investigator recording outcomes, knew to which group the patients were assigned.



[Table/Fig-1]: CONSORT (Consolidated Standards of Reporting Trials) flowchart of recruited participants.

1. Group D (Dexmedetomidine): Patients were treated with i.v. dexmedetomidine with a loading dose of 1.0 µg/kg over 10 minutes followed by a continuous infusion of 0.5 ug/kg/hr until surgery was completed [15].
2. Group L (Lignocaine): Patients were given i.v. lignocaine (2% preservative-free) induction 1.5 mg/kg and infusion 1.5 mg/kg/hr during surgery [15].
3. Group P (Placebo): Patients were given placebo infusion of 0.9% saline 1 mL/kg/hr. All infusions were coded to keep the study blinded.

All patients were premedicated with glycopyrrolate i.v. (0.2 mg) and midazolam (1-2 mg) and given their assigned study drug. After a period of 10 minutes of equilibration, they were preoxygenated for three minutes. Anaesthesia induction contained thiopentone (5 mg/kg) and succinylcholine (1.5 mg/kg) for intubation. Maintenance with sevoflurane (end-tidal 1.5-2%), nitrous oxide/oxygen (50:50) mix and vecuronium (0.1 mg/kg) for neuromuscular blockage and monitored for haemodynamics and recovery. The primary outcome was reduction of the haemodynamic response to laryngoscopy and intubation as measured by HR, SBP, DBP and MAP. Secondary outcomes were time to extubation, postoperative levels of pain measured with the VAS and postoperative sedation levels assessed with the RASS recorded at 0 and 20 minutes after tracheal extubation by observers blinded to group allocation [16].

STATISTICAL ANALYSIS

Data were analysed with the use of the Statistical Package for Social Sciences (SPSS) v 20.0 IBM Corp., Armonk, NY, USA. Continuous variables were given either as Mean±SD or median with IQR. Intergroup comparisons between normally distributed continuous variables were conducted by means of One-way ANOVA with post-hoc tests of Tukey's for pairwise comparisons. Non parametric continuous variables (VAS and RASS scores) were compared with Kruskal-Wallis test. Significance for categorical variables were measured using Chi-square test. A p-value less than 0.05 was taken as statistically significant.

Variables	Dexmedetomidine n (%)	Lignocaine n (%)	Placebo n (%)	p-value
Age (years) (Mean±SD)	43.7±4.3	41.1±3.8	43.0±5.1	0.06
Gender				
Male	15 (50.0%)	12 (40%)	13 (43.3%)	0.73
Female	15 (50.0%)	18 (60%)	17 (56.7%)	
(Mean±SD)	24.54±2.1	23.96±3.4	24.37±2.4	0.69
ASA (I/II)				
I	13 (43.3%)	17 (56.7%)	12 (40%)	0.39
II	17 (56.7%)	13 (43.3%)	18 (60%)	
Duration of surgery (min) (Mean±SD)	83.0±4.3	82.3±4.7	84.4±5.1	0.21

[Table/Fig-2]: Baseline demographic and clinical characteristics.
Intergroup comparisons between normally distributed continuous variables were conducted by means of One-way (ANOVA) and Significance for categorical variables were measured using Chi-square test.

RESULTS

A total of 90 patients were included in the study and they were equally divided into three groups including dexmedetomidine, lignocaine and placebo. The demographic and clinical data characteristics of the three groups were similar, as presented in [Table/Fig-2]. There were no statistically significant differences in age, gender distribution, Body Mass Index (BMI) or ASA physical status among the three groups. Also, the duration of surgery was comparable between the groups of dexmedetomidine, lignocaine and placebo (p=0.21), suggesting similar baselines.

The HR was significantly higher in lignocaine and placebo group in all the times compared to dexmedetomidine group [Table/Fig-3].

Baseline SBP and DBP values were similar among the three groups (SBP: p=0.62; DBP: p=0.12), indicating no preinduction differences [Table/Fig-4]. Significant SBP and DBP values in lignocaine and placebo group compared to dexmedetomidine group in all the times.

Baseline MAP values were similar across the dexmedetomidine, lignocaine and placebo groups. During induction, MAP increased in all groups, however, the rise was significantly lower in the dexmedetomidine group compared to lignocaine and placebo group [Table/Fig-5].

The haemodynamic response to laryngoscopy and endotracheal intubation, shown in [Table/Fig-6], reveals that the increase in HR (ΔHR) was significantly lower in the dexmedetomidine group compared to lignocaine and placebo.

Similarly, SBP increased less with dexmedetomidine than with lignocaine and placebo DBP and Mean Arterial Pressure MAP also showed lesser increases in the dexmedetomidine group compared to lignocaine and placebo group [Table/Fig-6].

Postoperative pain levels, measured with the VAS and RASS at 0 and 20 minutes, were no different between the three groups [Table/Fig-7] . RASS was significant at zero minute in dexmedetomidine group compared to lignocaine and placebo.

No adverse events was seen in all three groups.

Time point (in minutes)	HR Dexmedetomidine (beats/min)	HR Lignocaine (beats/min)	HR Placebo (beats/min)	p-value
Baseline (pre-induction)	76.4±4.6	76.5±5.7	76.3±5.5	0.989
During induction (post-induction drugs)	76.3±4.8	80.5±5.7	80.8±5.3	0.002
During intubation (laryngoscopy)	84.7±4.7	90.3±6.2	91.2±6.1	<0.001
3 min	80.3±5.0	88.5±5.7	89.8±5.3	0.001
9 min	78.6±5.9	87.5±6.1	88.3±5.0	<0.001
12 min	77.8±5.1	86.9±5.1	88.1±5.4	<0.001
15 min	77.2±5.3	86.7±3.5	87.6±4.5	<0.001
30 min	76.3±4.8	85.8±5.4	87.2±3.3	0.001
45 min	76.2±4.9	85.6±4.6	87.0±5.1	<0.001
60 min	76.1±4.4	84.0±5.2	86.1±5.6	<0.001
75 min	75.8±4.6	83.6±6.4	85.8±4.3	<0.001
90 min	77.0±4.7	85.5±4.8	86.9±5.9	<0.001
Post-intubation (early)	80.2±4.4	90.3±5.3	93.6±4.6	0.002

[Table/Fig-3]: Comparison of Heart Rate (HR) from baseline to post-intubation.
Intergroup comparisons between normally distributed continuous variables were conducted by means of ANOVA. Values are expressed in (Mean±SD).

Time point (in minutes)	SBP Dex (mm Hg)	SBP Lig (mm Hg)	SBP Plb (mm Hg)	p-value	DBP Dex (mm Hg)	DBP Lig (mm Hg)	DBP Plb (mm Hg)	p-value
Baseline (pre-induction)	114.4±4.6	115.6±5.4	115.4±5.1	0.62	78.5±4.9	77.8±5.9	75.7±5.4	0.12
During induction (post-induction drugs)	118.4±3.8	120.5±5.6	121.5±4.8	0.04*	76.3±5.5	78.9±4.6	79.7±5.0	<0.001
During intubation (laryngoscopy)	127.2±6.1	131.7±5.8	131.8±5.4	0.03*	82.2±4.8	89.5±4.2	90.6±5.1	<0.001
3 min	118.0±3.8	120.7±4.9	120.9±5.6	0.03*	75.3±5.5	79.6±4.6	80.3±5.0	<0.001
9 min	117.1±5.0	119.9±5.2	120.4±5.4	0.03*	74.9±4.6	78.2±5.8	79.1±4.6	0.001
12 min	116.3±5.4	119.4±4.6	120.4±3.9	<0.01*	74.6±4.7	78.0±5.4	78.9±4.6	0.001
15 min	115.6± 4.6	119.0±5.1	120.1±5.0	<0.01*	74.5±4.9	78.0±4.8	78.7±4.7	<0.001
30 min	115.1±5.1	119.0±5.0	119.8±5.7	<0.01*	73.7±4.8	77.2±5.0	78.3±5.1	<0.001
45 min	114.7±4.9	118.5±4.7	119.6±4.9	<0.01*	72.5±5.5	76.9±5.1	77.8±5.6	<0.001
60 min	114.4±5.0	117.5±5.0	119.4±5.6	<0.01*	72.1±4.2	76.3±4.8	77.2±4.3	<0.001
75 min	115.3±5.1	118.7±5.5	119.5±4.8	<0.01*	71.7±4.0	77.6±4.8	79.0±4.3	0.002
90 min	116.9±3.7	118.9±5.4	120.5±4.3	0.01*	72.0±5.4	79.5±4.6	80.7±5.8	<0.001
Post-intubation (early)	119.9±5.6	122.0±5.2	124.0±4.2	<0.01*	81.2±5.0	85.5±4.2	90.2±5.8	0.002

[Table/Fig-4]: Comparison of Systolic (SBP) and Diastolic BP (DBP) from baseline to post-intubation.
Intergroup comparisons between normally distributed continuous variables were conducted by means of ANOVA

Time point (in minutes)	MAP Dexmedetomidine (mm Hg)	MAP Lignocaine (mm Hg)	MAP Placebo (mm Hg)	p-value
Baseline (pre-induction)	85.1±5.5	85.6±5.8	86.6±6.2	0.591
During induction (post-induction drugs)	87.5±5.4	90.0±5.1	90.9±5.6	0.04*
During intubation (laryngoscopy)	96.9±5.6	99.1±5.2	100.6±6.0	0.04*
3 min	89.7±5.4	92.4±4.8	92.8±5.2	0.04*
9 min	89.0±5.4	91.9±5.1	92.2±5.6	0.04*
12 min	88.0±5.1	90.6±5.5	91.5±5.8	0.04*
15 min	87.5±4.8	90.5±5.8	91.0±4.0	0.02*
30 min	87.2±5.2	90.4±5.1	90.7±4.9	0.02*
45 min	86.8±3.6	89.5±5.1	89.8±4.7	0.02*
60 min	85.2±5.8	88.3±5.8	88.6±4.0	0.02*
75 min	85.0±5.9	88.1±5.4	89.1±4.3	<0.001
90 min	84.9±6.3	88.4±6.9	88.7±6.0	0.04*
Post-intubation (early)	99.1±5.2	100.1±5.2	102.6±6.0	0.04*

[Table/Fig-5]: Comparison of Mean Arterial Pressure (MAP) from baseline to post-intubation.
Intergroup comparisons between normally distributed continuous variables were conducted by means of One-way ANOVA

Variables	Dexmedetomidine	Lignocaine	Placebo	p-value
ΔHR	8.3±4.7	13.83±6.2	14.90±6.1	<0.001
ΔSBP	12.8±6.1	16.1±5.8	16.4±5.4	<0.05*
ΔDBP	10.7±4.8	16.7±4.2	18.9±5.1	0.001
ΔMAP	11.8±5.6	13.5±5.2	14.0±6.0	<0.05*

[Table/Fig-6]: Intubation response (change from baseline).
Intergroup comparisons between normally distributed continuous variables were conducted by means of One-way ANOVA.

Variables	Dexmedetomidine	Lignocaine	Placebo	p-value
Time to extubation (min)	8.91±1.60	9.09±1.73	8.85±1.65	0.85
VAS at 0 min	2.03±0.64	2.37±0.72	2.30±0.76	0.08
VAS at 20 min	2.18±0.70	2.12±0.65	2.25±0.82	0.40
RASS at 0 min	-0.90±0.62	-0.20±0.52	0.05±0.58	0.02
RASS at 20 min	-0.05±0.35	0.0±0.30	0.05±0.42	0.42

[Table/Fig-7]: Recovery and postoperative outcomes.
Time to extubation interpreted in mean and SD; VAS and RASS are interpreted in median and interquartile ranges. Intergroup comparisons between normally distributed continuous variables were conducted by means of One-way ANOVA. VAS and RASS scores) were compared with Kruskal-Wallis test.

DISCUSSION

The present study evaluated the effectiveness of dexmedetomidine, intravenous lignocaine and a placebo in reducing the haemodynamic response of laryngoscopy and endotracheal intubation, as well as determining the effect of these agents on postoperative recovery parameters. The baseline demographic characteristics such as age, gender distribution, BMI, ASA physical status, duration of surgery were similar among the three groups of patients and therefore homogeneity was maintained and variables that could affect the outcome were kept low. Similar demographics comparability has also been observed in studies conducted by Keniya V et al., and Aho M et al., which showed no statistically significant differences in baseline patient characteristics between dexmedetomidine and control groups [17,18]. This similarity in baseline variables justifies the premise that the differences in haemodynamic parameters seen in the present study are probably caused by the pharmacologic effects of the administered drugs, not by demographic differences. Additionally, Yildiz M et al., also found similar demographic profiles while testing dexmedetomidine for attenuation of intubation responses, which also strengthens the validity of the present study design [19].

In the current study, a significant reduction in the increase of HR occurring during induction, laryngoscopy and intubation was found with dexmedetomidine, compared with both lignocaine and placebo. The increase in HR during intubation was least seen in the dexmedetomidine group (84.7±4.7 bpm) as compared with the

lignocaine (90.3±6.2 bpm) and placebo group (91.2±6.1 bpm). These findings support the sympatholytic effects of dexmedetomidine which are mediated through central a2-adrenergic receptor activation. Similar findings were reported by Yildiz M et al., who showed that dexmedetomidine had a significant effect on reducing the tachycardic response to laryngoscopy compared to placebo [19]. Keniya V et al., also found a significant decrease in the HR responses during intubation in patients receiving dexmedetomidine infusion [17]. In contrast, lignocaine acts mainly by local anaesthetic action and suppression of airway sensory pathways to blunt airway reflexes rather than by central sympatholysis. Studies by Hamaya Y and Dohi S have established that lignocaine causes only moderate reduction of HR responses in line with the results reported in the current study [20].

The results of the present study show that the SBP, DBP and MAP were higher during laryngoscopy and intubation in all groups, but the increase in this group was significantly lesser in the dexmedetomidine group. The MAP during intubation was 96.9±5.6 mmHg among dexmedetomidine cohort as compared to 99.1±5.2 mmHg and 100.6±6.0 mmHg among lignocaine and placebo groups respectively. These results are consistent with the known sympatholytic effects of dexmedetomidine which reduce catecholamine release and reduce systemic vascular resistance. Comparable haemodynamic stability with dexmedetomidine has been reported by Menda F et al., who found significantly less arterial pressure responses during intubation in cardiac surgery patients receiving dexmedetomidine [21]. Moreover, Bajwa SS et al., showed improved haemodynamic stability with dexmedetomidine when compared to clonidine during laryngoscopy [22]. In comparison intravenous lignocaine has been shown to bring a small, but inconsistent reduction in BP responses. Singh V et al., suggested that lignocaine acts mainly to reduce the effect on the pressor response by suppression of the airway reflexes rather than on reduction of central sympathetic outflow, which explains the relative lesser effects seen in the present study [15].

The evaluation of the haemodynamic changes from baseline confirmed further the superior efficacy of dexmedetomidine in attenuation of intubation responses. The changes in Heart Rate (ΔHR) and BP parameters (Δ SBP, Δ DBP, Δ MAP) were significantly less in the group with dexmedetomidine as compared to both lignocaine and placebo. This suggests good inhibition of the sympathetic response evoked by laryngoscopy and tracheal intubation. Similar decreases in haemodynamic fluctuations have been reported by Lawrence CJ and De Lange S who showed that dexmedetomidine is effective for blunting the catecholamine-mediated cardiovascular responses during airway manipulation [23]. The small reductions seen with lignocaine in this study are in line with that of Joshi A et al., who found lignocaine reduces airway reflex-mediated haemodynamic responses but may not completely attenuate sympathetic activation [24].

The parameters of postoperative recovery assessed in the present study revealed no significant differences in the extubation time and postoperative pain scores between the three groups. However, early postoperative sedation (assessed using the RASS) was significantly greater in the dexmedetomidine group immediately after extubation. This effect can be explained by the sedative and anxiolytic action of dexmedetomidine, which work through activation of the alpha two receptors in the locus coeruleus. Previous studies by Hall JE et al., agree with the findings, showing that dexmedetomidine promotes a co-operative sedation without significant respiratory depression [25]. Similarly, Gerlach AT et al., reported improved sedation profiles and patient comfort in a postoperative setting when dexmedetomidine was used [26]. Also, sedation levels between the groups converged after 20 minutes suggesting a rapid recovery and no evidence of prolonged sedation. These outcomes are consistent with observations made by Bhana N et al., who also described predictable recovery profiles related to the use of dexmedetomidine [27].

Limitation(s)

First, the study was carried out in one centre with a relatively small sample size which may limit the generalisation of the results. Second, long-term postoperative results were not evaluated. Future multicentric studies with larger sample size may have stronger evidence regarding the comparative efficacy of dexmedetomidine and lignocaine.

CONCLUSION(S)

Dexmedetomidine was superior to intravenous lignocaine for reducing the haemodynamic response of laryngoscopy and endotracheal intubation, thus consistent with the hypothesis. Patients who were receiving dexmedetomidine showed improved perioperative haemodynamic stability with less HR and BP variation. Although postoperative pain scores were similar in the groups, dexmedetomidine was associated with increased early postoperative sedation scores. Lignocaine had a moderate effect on the intubation response but was inferior to dexmedetomidine. The drugs were well tolerated with no major adverse effects observed with both drugs. Dexmedetomidine therefore may be considered a useful agent for maintaining the haemodynamic stability of patients undergoing laparoscopic surgeries.

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