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Original Research Article

Comparative Study of Effect of Intravenous Dexmedetomidine and Intravenous Lignocaine on Hemodynamic Response to Tracheal Extubation in Laparoscopic Surgeries

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Abstract

Background: Patients go through a period of extreme physiological stress during their recovery from general anesthesia and extubation. The most common post-Laparoscopic surgical consequences are myocardial ischaemia, arrhythmias, increased intracranial pressure, cerebral edema and respiratory problems leading to postoperative morbidity. Thus, maintaining normal respiratory and cardiovascular parameters is an important part of anesthetic emergence.

The aim of present study is to comparatively study the effect of Intravenous Dexmedetomidine and Intravenous Lignocaine on hemodynamic response to tracheal extubation in laparoscopic surgeries.

Methods: 60 Patients in ASA Grade I and II, ages 18 to 60, who were scheduled for Laparoscopic surgery were split into two groups of 30 at random. The general anesthesia was balanced to all patients. At the end of surgery patients in Group I got intravenous (IV) injection of dexmedetomidine (0.5 µg/kg) and Group II received 2% preservative free IV injection of lignocaine (1.5 mg/kg). After extubation, measurements were made of the heart rate (HR) and mean arterial pressure (MAP) at intervals of 1, 3, 5, 10, and 15 minutes. Emergence and extubation times were recorded, and a cough grading system was used to assess the quality of the extubation.

Results: MAP and HR were significantly lower in Group I than in Group II at all-time points following extubation ($p<0.05$). Each of the groups experienced emergence and extubation at around the same length of time.

Conclusion: Compared to 1.5 mg/kg of lignocaine, a dose of 0.5 µg/kg IV dexmedetomidine administered before tracheal extubation effectively attenuates the hemodynamic response to extubation.

Keywords: Dexmedetomidine, Laparoscopic surgery, Lignocaine, postoperative analgesia, Tracheal extubation.

Introduction

During the use of general anesthesia in Laparoscopic surgeries, one of the most unpleasant conditions is extubation. It is nearly invariably linked to a number of undesirable side effects, such as hemodynamic and respiratory abnormalities [1]. Extubation during lighter plane of anesthesia elicits reflexes by irritating the trachea and larynx. Hemodynamic alterations are brought on by a reflex increase in sympathetic activity linked to this laryngotracheal stimulation. Numerous theories, including a surge in catecholamines, airway irritation from suction, and excruciating pain from surgical wounds and emergence, have been proposed as explanations for the abrupt rise in heart rate and blood pressure following extubation [2,3]. Typically, these hemodynamic alterations are erratic, fleeting, and variable [3-6]. Nonetheless, patients with cerebrovascular diseases, cerebral aneurysms, heart disease, and systemic hypertension are at higher risk. Acute consequences include cerebral haemorrhage, arrhythmias, myocardial ischemia, left ventricular failure, pulmonary edema, and rupture of intracranial aneurysms can arise from even brief variations in arterial blood pressure and heart rate [7].

Intra-tracheal local anesthetic instillation, intravenous lignocaine, intra endotracheal cuff lignocaine, short acting opioids such as fentanyl and remifentanil, β blocker and calcium channel blockers are used to attenuate these responses during tracheal extubation.[8]

Lignocaine is an amide local anesthetic. Due to its ability to impede neuronal conduction, it may also be used as an adjuvant medication to relieve pain following surgery. Intravenous lignocaine exhibits anti-inflammatory, antihyperalgesic, and analgesic effects. It works centrally by altering neural responses in the spinal dorsal horn and peripherally by reducing the release of inflammatory mediators. Numerous mechanisms, including sodium channel blocking, G-protein coupled receptor inhibition, and N-methyl-D-aspartate receptor inhibition are likely to be involved in mediating these effects [8]. One of the earliest, least expensive, and easiest to obtain medications for reducing airway and circulatory reflexes during the awakening from general anesthesia is intravenous lignocaine [9].

Strong α2 adrenoreceptor agonist Dexmedetomidine is a valuable adjuvant to help with smooth tracheal extubation and offers good sedation with little respiratory depression or cardiovascular instability [10]. Additionally, because of a decrease in the plasma catecholamine reactions to extubation, it has been found to lower arterial blood pressure, heart rate, and hemodynamic response [11,12].

There are very few studies in the literature comparing intravenous dexmedetomidine and lignocaine on the basis of hemodynamic response to tracheal extubation in small invasive surgeries. Hence the present study was done to comparatively study the effect of Intravenous Dexmedetomidine and Intravenous Lignocaine on hemodynamic response to tracheal extubation in laparoscopic surgeries.

Material & Methods

The present prospective, interventional study was conducted among 60 patients who were admitted to department of surgery and were scheduled for Laparoscopic surgeries during the study period of 1 year. The ethical permission was taken from institutional review board of allied medical college and hospital before commencement of study.

The patients were randomly divided into two groups of 30 each i.e. group I (received intravenous Dexmedetomidine 0.5 μg/kg) and group II (received 2% preservative free IV injection lignocaine 1.5 mg/kg). The patients of ASA I and II, aged 18-60 years undergoing laparoscopic surgery were included for the study. Patients with history of ischemia, congestive cardiac disease, renal, hepatic, and respiratory dysfunction, patients taking beta blocker, digoxin, patients with heart rate (HR)<60 bpm, and blood pressure<100/60, history of allergic reactions to study drugs, history of sleep apnea, anticipated difficult airway and history of seizures were excluded from study.

Prior to surgery, each patient had their anesthetic suitability assessed and was instructed to fast for six to eight hours. A wide bore intravenous (IV) line was placed in the preoperative area followed by application of monitors in the form of a five lead ECG, Pulse oximetry, and non-invasive blood pressure in the operating room. Midazolam 1-2.5mg IV, Glycopyrrolate (0.2mg), ondansetron 0.8mg/kg, fentanyl 3μg/kg as premedication, IV Propofol 2-2.5 mg/kg for induction and Vecuronium 0.1mg/kg for intubation were given. All the patients were intubated with appropriate size endotracheal tube in 1or 2 attempts. End-tidal carbon dioxide (EtCO2) was maintained in all patients by mechanical ventilation at a fresh gas flow rate of 2 L/min. Sevoflurane 0.8–1.0 minimum alveolar concentration (MAC) in combination with nitrous oxide and oxygen (60:40) MAC and Vecuronium intermittent boluses were used to maintain anesthesia. After induction, no further opioid was administered. The baseline was the measurements of HR, systolic arterial pressure (SAP), diastolic arterial pressure (DAP), and mean arterial pressure (MAP) taken shortly before the study medication was administered.

Patients in Group I were given 0.5 μg/kg of Dexmedetomidine (diluted with 10 ml of normal saline), and patients in Group II were given 1.5 mg/kg of lignocaine (diluted with 10 ml of normal saline) over 10min at the start of skin closure. Inhalation anesthetic and nitrous oxide were discontinued at the end of the surgery.

Neostigmine 0.05 mg/kg and glycopyrrolate 0.01 mg/kg were used to reverse the neuromuscular block throughout a 60-second period. When the patient obeyed orders, endotracheal tube was removed. Notable during tracheal extubation were HR and MAP. Prior to giving the study medication, 1min and 3min after study medication, right after extubation, and one, three, five, ten, and fifteen minutes later, hemodynamic variables were recorded. Time taken for emergence and extubation were noted.

Statistical Analysis

The statistical analysis was carried out with SPSS 25.0. The data are shown as a number (%) or as the mean (standard deviation). The Chi-square test was used to compare the qualitative data. The ANOVA test was used to examine the values for HR and blood pressure with a normal distribution. The Chi-square test was used to assess non-parametric data. Accepted statistical significance was $p<0.05$.

Results

The patients in the two groups were comparable for age, weight and male: female ratio, ASA physical status, duration of surgery, duration of anesthesia, and the difference between the two groups was not statistically significant with p >0.05 as shown in table 1.

Table 1: Showing demographic data of patients

Variable	Group I	Group II	P value
Age (in years)	38.5±14.2	39.2 ±12.2	0.102
Sex (M/F)	19:11	16:14	0.221
Duration of surgery (in minutes)	160±57.02	163±55.8	0.134
Duration of anesthesia (in minutes)	175.63±55.2	176±65.1	0.147

There was a statistically significant (p<0.05) difference in HR between the two groups. A statistically significant difference (p<0.05) in the mean arterial pressure (MAP) was seen between the two groups commencing five minutes after the agent was administered and continuing until the time observations were made, as indicated in Table 2.

Table 2: Showing hemodynamic data

Duration	Heart rate		MAP	
	Group I	Group II	Group I	Group II
Baseline	77.27±10.8	78.7±10.43	93.63±12.3	102.2±4.5
After drug administration (1 min)	78.23±11.4	79.23±10.2	98.23±7.41	104.3±10.6
After drug administration (3 min)	78.13±8.4	79.63±10.7	95.67±8.4	103.2±9.4
Immediate after extubation	75.16±13.5	86±10.5	96.02±11.3	102.1±5.3
1 min	73.03±12.3	86.3±10.5	91.7±10.5	101.2±9.1
3 mim	71.28±10.4	85.2±10.2	90.3±10.7	101.1±8.2
5 min	71.42±11.2	80.2±8.6	88.2±9.3	100.3±10.2
10 min	71.21±10.2	76.3±9.6	85.2±10.2	99.4±10.3
15 min	71.1±10.1	73.2±8.8	84.23±10.0	98.7±10.2

The two groups' emergence and extubation timings did not differ significantly (p>0.05). Table 3 indicates that there was a delay (p=0.001) in the time to first analgesic necessity in Group I.

Table 3: Showing intraoperative and postoperative data

Duration	Group I	Group II	P value
Extubation time (minutes)	10.3±1.2	9.2±2.0	0.119
Emergence time (minutes)	8.6±2.3	7.5±6.9	0.345
Time of first analgesic (minutes)	23±6.5	13.2±5.2	0.001

Comparison of extubation quality based on cough score on 4-point scale was done and it was found that group 1 shows less cough score than group 2 as shown in table 4

Table 4: Showing comparison of extubation quality on 4-point scale

Cough score	Group I	Group II
0	20 (66.6)	18 (60)
1	10 (33.4)	8 (26.6)
2	0	4 (13.4)
3	0	0

Discussion

Numerous problems, including coughing, respiratory issues (laryngospasm, blockage of the airway, desaturation), and hemodynamic alterations, can arise after extubation. [13] Various strategies have been employed to avert hemodynamic reactions resulting from the emergence from anesthesia, such as deep anesthesia extubation, the injection of local anesthetics, vasodilators, and short-acting opioids [14].Vasodilators including nitroglycerin, hydralazine, and sodium nitroprusside may cause side effects such reflex tachycardia and elevated plasma renin activity when administered [15].

We utilized dexmedetomidine, a highly selective α-2 agonist with sedative, analgesic, and anesthetic sparing effects, during extubation in our study. It results in a dose-dependent drop in HR and arterial blood pressure that is accompanied by a drop in plasma catecholamine levels. [16] Following the termination of assisted breathing, there was no discernible clinical respiratory depression. In postsurgical ventilated patients, dexmedetomidine caused fast and consistent sedation while preserving a high level of patient arousability and anxiety reduction [17].

In the present study, dexmedetomidine 0.5 μg/kg IV given before tracheal extubation was associated

with significantly less coughing and better quality of extubation than lignocaine.

In a study conducted by Ghimire et al., the opioid-sparing properties of intravenous lignocaine were verified in patients undergoing laparoscopic procedures. In comparison to the placebo group the overall opiod requirement was less in lignocaine group.[18] Staikou et al found that intraoperative intravenous lidocaine did not provide any advantage over placebo in terms of postoperative analgesia. Anis et al. found that there was no significant difference in postoperative pain scores in two groups but the dexmedetomidine group experienced more sedation.[9] We credit the elimination half-lives of intravenous lignocaine and dexmedetomidine for the larger Extubation-first rescue analgesic time (EFRD) interval in dexmedetomidine and the comparable EFRD interval in lignocaine. While dexmedetomidine has elimination half-life of 2.1–3.1 hours in healthy adults, lignocaine has a shorter half-life, ranging from 6 minutes following a bolus to up to 90–120 minutes following infusions lasting less than 12 hours. The control group's very identical EFRD length may have resulted from a greater (almost double) intraoperative dose of fentanyl. [19,20]

We saw immediate hypertension in 20% of the individuals after dexmedetomidine.[21] α-2 agonists have a biphasic effect on hemodynamics: first, they stimulate the peripheral α-2B receptor to increase systemic artery pressure, and then they stimulate the central nervous system's α-2 adrenoceptor to generate a longer-lasting decrease in pressure.[22,23] After administering dexmedetomidine, Sharma et al [24] noted that MAPs rose for the first two minutes and then recovered to normal after three minutes. We discovered comparable findings as well. It is known that α-2 agonists have analgesic properties. We also observed in our trial that Group I had a substantially longer wait time ($p<0.0001$) for the first analgesic following extubation; this finding is consistent with Sharma et al. [24]

In our study the emergence time for group I (8.6±2.3) was higher than group II (7.5±6.9). Despite the patients in the dexmedetomidine group showing a prolonged time to awaken, this cannot be referred to as delayed awakening according to the established definition of delayed awakening. It is proposed that dexmedetomidine-induced sedation, also known as cooperative sedation, resembles healthy, arousable sleep. But according to the authors, this can be risky for patients who are poor candidates for postoperative sedation and in situations where postoperative monitoring facilities are subpar, particularly in settings with low resources. Menshawi and Fahim observed similar results.[25] According to Tufanogullari et al and Kang et al, the dexmedetomidine group emerged sooner and spent less time in the PACU. At 30 minutes after extubation, Patel et al noted considerable drowsiness. [26, 27, 28]

The small sample size, single-centric design, and inability to determine the plasma levels of specific medicines were the study's drawbacks. Moreover, the majority of the patients in our study were ASA physical status classes I and II. In order to truly and universally evaluate the two study medications, more research with patients in higher ASA classes is required.

Conclusion

Opioids may benefit from the addition of dexmedetomidine or lignocaine in patients having laparoscopic procedures. IV Dexmedetomidine HCl 0.5 µg/kg given before tracheal extubation effectively attenuate hemodynamic response to extubation as compared to 1.5 mg/kg lignocaine. More so than lignocaine, dexmedetomidine prolongs the opioid-sparing effect during the postoperative phase in terms of delayed first rescue analgesic consumption and overall analgesic consumption. Nonetheless, the anesthesiologist must exercise caution when administering dexmedetomidine during laparoscopic procedures due to the potential for intraoperative bradycardia, particularly in patients who are not good candidates for postoperative sedation or in settings with limited resources and inadequate monitoring facilities. An alternative for these patients may be to use lignocaine during the postoperative period and dexmedetomidine during the intraoperative phase.

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