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Relative Evaluation of IV Propofol and IV Dexmedetomidine For Small Surgical Procedures

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ABSTRACT

Short surgical procedures under total intravenous anesthetic have been found to respond favorably to propofol. The "star" drug in the anesthetic therapy toolbox is dex-medetomidine a sedative and analgesic selective alpha-2 agonist. To determine if dexmedetomidine can be used as the only anesthetic drug to maintain depth of anesthesia. Dexmedetomidine was infused continuously at a rate of 0.2-0.6 mg kg⁻¹ hrs after this solution was injected at a rate of 10 mL min for a total dose of 1 g kg⁻¹. The mean ages in both groups were similar, with group 1 (Dexmedetomidine) recording a mean age of 36.07 years and group 2 (Propofol) recording a mean age of 33.8 years. Dexmedetomidine was related with identical hemodynamic effects, maintaining appropriate respiratory function and achieving an Aldrete recovery score of 10 at similar sedation levels of ramsay sedation score with propofol. Dexmedetomidine, surgical procedures, propofol.

INTRODUCTION

In these situations, whole intravenous anesthetic is beneficial. A speedy and "street fit" recovery after childcare surgery has become crucial in modern anesthetic practice. Short surgical procedures under total intravenous anesthetic have been found to respond favorably to propofol. The "star" drug in the anesthetic therapy toolbox is dex-medetomidine a sedative and analgesic selective alpha-2 agonist. Dextmedetomidine is an imidazole derivative. It has an 8-fold higher specificity for alpha-2 adrenoreceptors (1620:1 for dexmedetomidine vs. 220:1 for clonidine) than clonidine [1]. The sedative, analgesic, sympatholytic and anxiolytic effects of dexmedetomidine lessen several of the cardiovascular reactions that happen right after surgery. Without appreciably impairing respiration, it lessens the need for volatile anesthetics, sedatives and analgesics^[2,3]. Dexm-edetomidine has been explored with success for conscious sedation during endoscopies, for intravenous sedation during dental surgery and for ICU situations. It has also been attempted intrathecally for lower abdominal operations and vaginal hysterectomy as well as for reducing the dosage and enhancing the analgesic impact of local anesthetics [4-6]. A survey was conducted by Wong et al. [7] to identify the issues that impact the usage of TIVA, including the unavailability of TCI pumps (which could be due to cost) additional costs that drive up the cost, the inability to anticipate wakefulness and the rise in awareness incidence. There is still disagreement over whether depth of anesthetic and intraoperative recall with TIVA are appropriate, despite the availability of comprehensive literature and established benefits. Because of these worries, the current study was designed to compare how well TIVA versus traditional inhalational mode maintained acceptable depths of anesthesia in patients undergoing modified radical mastectomy (MRM) as determined by the bispectral index (BIS) recovery profiles and cost analysis. Instead of using nitrous gas and sevoflurane for inhalational anesthesia in this trial, injectable propofol and dexmedetomidine for TIVA were administered using conventional syringe infusion pumps. Inhaled agents have been employed to impede intraoperative awareness. One typical intravenous drug used for intraoperative sedation is propofol. The food and drug administration has authorized the alpha-2 receptor agonist dexmedetomidine for use as a sedative in the intensive care unit (ICU)[8]. At $the rapeut ic\,dos ages,\,dex medeto midine\,does\,not\,cause$ respiratory depression and has analgesic sparing properties^[9,10]. It has not been the only anesthetic, rather, it has been used in addition to anesthesia. Eleven We suggested examining the effect of dexmedetomidine and propofol on depth of anesthesia using hemodynamic variables and BIS values in order

to ascertain if dexmedetomidine may be used as the sole anesthetic medication to maintain depth of anesthesia.

MATERIALS AND METHODS

Sixty patients were included in the study. This study was conducted in Department of General Surgery, National Institute of Medical Sciences and Research Center.

Inclusion criteria: Age groups 20-50 years both males and females, belonging to ASA I and II undergoing short surgical procedures were included in the study. Use of any opioid or sedative medication in the week prior to surgery, alcohol or drug abuse, known allergy to either dexmedetomidine or propofol and cardiovascular, respiratory, neurological, psychological, hepatic or renal disease.

Every patient was assessed properly and in detail one day prior to surgery. Routine investigations were performed in each case and whenever required, specific tests like X-ray ECG, LFT etc were asked for. Patients were interviewed for drug history and past history of anesthesia are related complications. Patients were instructed to undergo overnight fasting before surgery. Using a computerized random generation table, the patients were randomly divided in to two groups of 30 patients each. On arrival in operating room, standard monitoring such as NIBP, pulse oximeter and ECG leads were attached to the patients. Supplemental Oxygen was given throughout the procedure at 4 L^{-1} min with Hudson"s mask. Intravenous access was established using an 18G cannula and Ringer Lactate 10 mL kg was infused. Preoperative pulse rate, systolic and diastolic blood pressure, respiratory rate and oxygen saturation were recorded. In Group-1 received Inj. fentanyl 1 μg kg⁻¹ was given 5 min before surgery and Inj. Dexmedetomidine 100 µg was added to 100 mL of normal saline and made to a concentration of 1 µg kg. This solution was administered at a rate of 10 mL min to a total dose of 1 $\mu\text{g}~\text{kg}^{-1}$ of dexmedetomidine followed by continuous infusion of Dexmedetomidine 0.2-0.6 μg kg⁻¹ hrs. In Group-2 received Inj. fentanyl 1 μg kg⁻¹ was given 5 min before surgery. Inj. Propofol 0.7 mg kg⁻¹ Body weight initially over a period of 10 min and followed by maintenance infusion of 0.5-2 mg kg⁻¹ hrs. Following Parameters were notedas Heart rate (HR) Systolic Blood Pressure (SBP) Diastolic Blood Pressure (DBP) Mean blood pressure (MAP) Oxygen saturation (spo2) and Respiratory Rate (RR). They were recorded before premedication and for every 2 mins upto 20 min and there after every 5 min till the end of the surgery. Vasopressor requirements will be noted as Hypotension (defined by a decrease In MAP below 20% of baseline or systolic pressure <90 mm Hg) was treated with intravenous fluids and intravenous ephedrine 5 mg increments.

RESULTS

The current study was conducted at our hospital during a one-year period and the findings are examined. Both group's mean ages were comparable group 1 (Dexmedetomidine) recorded a mean age of 36.07 years, while group 2 (Propofol) recorded a mean age of 33.8 years. In group 1, the mean body weight was 58.62 kg, while in group 2, it was 60.11 kg. The mean weight student's t-test was used to see if the mean age and mean weight were equal and the results were not significant Table 1. Table 2 shows the gender distribution for the two groups in the study population. Given that the two groups did not differ statistically (p>0.05) the distribution of the sex groups included in this investigation was comparable. The mean arterial pressure decreased significantly in both groups when compared to the base line values (p>0.05). Significant is defined as p<0.05.

DISCUSSION

comparable sedative dosages, dexmedetomidine and propofol markedly reduced heart rate and mean arterial pressure as compared to baseline values. The similar results were found by Kaygusuz et al. [12]. Propofol has been demonstrated in earlier studies to have a strong inhibitory effect on sympathetic outflow^[13]. It is also known that dexmedetomidine decreases sympathetic outflow and circulating catecholamine levels; hence, it is expected to have a comparable effect on mean arterial pressure to that of propofol^[14]. The heart rate reduction may be caused by the sympatholytic actions and, to a lesser extent, a vagomimitic action^[15]. Another interesting conclusion of the study was that, in contrast to Propofol sedation, Dexmedetomodine sedation preserved a normal respiratory function. The respiratory SpO2 values of the propofol group were

significantly lower during the sedation phase than those of the dexmedetomidine group. Hsu et al. [16] reported similar effects on respiratory processes during dexmedetomidine sedation. They explained the relationship between the rise in minute breathing and the arousal phenomenon. During spontaneous sleep, this kind of subsequent arousal to hypercapnia stimulus has been observed. The typical sleep route is where dexmedetomidine converges to provide its sedative effects. Moreover, De Sarro et al. [17] showed that the central nervous system contains many sites for-2 receptors. Hypercapnia activates the locus ceruleus, which is associated with heightened anxiety and activates the respiratory centers. Ebert et al. [13] also reported similar results when dexmedetomidine was used as a sedative. Arain and Ebert^[18] reported similar respiratory end points between the dexmedetomidine and propofol groups, despite Kaygusuz et al. [12] reporting significantly lower respiratory rate values and significantly higher SpO2 values in the dexmedetomidine group compared with the propofol group. The disparity in the results could be due to various drug combinations or rates of infusion. Because the severe side effects appeared as soon as the loading dosage was given, the loading dose in this experiment has been lowered. Reducing the loading dose causes the goal BIS to be delayed, nevertheless, in this experiment, the addition of an induction agent made it possible to attain the goal BIS (40-65). Nineteen Even at lower loading doses, dexm edetomidine produced more amnesia for procedural sedation than remifentanil did^[19]. Ramsay and colleagues reported utilizing a higher dose of dexmedetomidine for intubation in difficult airway conditions without the use of an induction medication or a neuromuscular blocker and finding hypotension, bradycardia and upper airway blockage^[20]. Compared to the cases reported by Ramsay, the current inquiry required far less of the study drug, along with an induction agent and neuromuscular blockers, to facilitate endotracheal intubation. It has been

Table 1: Comparison of age and weight between the two study groups.

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Parameters	Group	Mean	±SD	T-value	p-value
Age (years)	Dexmedetomidine	37.08	11.84	-0.923	p>0.05
	Propofol	34.81	9.821		
	Dexmedetomidine	58.62	10.573	-0.682	p>0.05
	Propofol	60.11	8.804		

Table 2: Distribution of gender in two study groups

Gender	Group	Total	
	Dexmedetomidine	Propofol	
Male	18	19	37
	45%	47%	46.2%
Female	22	21	43
	55%	52%	53.7%
Total	40	40	80

Chi Square: 0.067; p>0.05

Table 3: Showing mean time to achieve Ramsay Sedation score of 4

Group	Mean	±SD	p-value
Dedetomidine	27.2	7.89	<0.01*
Propofol	17.11	7.88	

proposed that BIS a continuous, noninvasive electroencephalographic approach, be used to track hypnotic state during sedation anesthesia [21,13]. Verified studies have connected its use to a decreased risk of intraoperative awareness^[23] By using dexmedetomidine and propofol, In a study involving volunteers, Yusuke and colleagues compared the BIS score to a clinical grading system known as the Observer Assessment of Alertness Sedation Score (OAA/S). They found that at equivalent doses, dexmedetomidine reduced BIS values and that BIS values of 46 for dexmedetomidine and 67 for propofol both produced OAA/S scores of 2, which indicate adequate sedation. In the current investigation, BIS values of patients receiving dexmedeto-midine infusions were statistically substantially lower than those getting propofol (BIS values of 50-55 for dexmedetomidine versus 60-65 for propofol). The modified Brice questionnaire showed that, with both drugs, similar depth was reached with no recollection in any patient, despite the difference in BIS. This could be explained by the different sedative effects of the two study drugs. Unlike propofol, which causes GABA agonism, dexmedeto-midine causes noradrenergic locus ceruleus neurons to hyperpolarize in order to produce sleep.

CONCLUSION

Dexmedetomidine was related with identical hemodynamic effects, maintaining appropriate respiratory function and achieving an Aldrete recovery score of 10 at similar sedation levels of Ramsay sedation score with propofol. Short surgical procedures under total intravenous anesthetic have been found to respond favorably to propofol.

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