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Original Study

Effect of dexmedetomidine on emergence delirium and recovery parameters with sevoflurane and desflurane anaesthesia in children : a double randomized study

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Abstract

Background: **Emergence delirium in pediatric patients is a significant cause of increased anxiety among parents. The incidence of emergence delirium in children varies mainly according to the anesthetic agents used.**
Methods: **In this prospective, double-blind, randomized trial, 152 children of age group 1-6 years were randomized into two groups after induction of anesthesia: Group S received Sevoflurane, and Group D received Desflurane. Children in the S group were further randomized into subgroup S- Dex (receiving dexmedetomidine 0.3 mcg/kg in 5 ml saline) and subgroup S-Saline (receiving saline 5 ml). Similarly, Group D was also randomized into two subgroups; D-Dex and D-Saline. We compared perioperative hemodynamic variables, postoperative emergence delirium, recovery profile, pain scoring, the requirement of rescue analgesics, and time to discharge.**
Results: **At 5, 15, and 30 minutes, the incidence of emergence delirium was significantly higher in S-Saline and D-Saline groups than S-Dex and D-Dex groups. Both PAED and FLACC scores were significantly higher in the S-Saline group than the S-Dex group and the D-Saline group compared to the D-Dex group ($P<0.05$). Significantly more patients required analgesia in the S-Saline group than in the S-Dex group ($P<0.05$). No significant difference for analgesia was present between D-Saline and D-Dex groups. ($p = 0.153$). Discharge time was significantly longer in S-Dex and D-Dex groups as compared to S-Saline and D-saline groups.**
Conclusions: **Dexmedetomidine effectively reduced the incidence of emergence delirium and postoperative pain in pediatric patients undergoing surgery using Sevoflurane and Desflurane anesthesia.**

Keywords: **Pediatric patients, Emergence Delirium, Dexmedetomidine, Sevoflurane, Desflurane, Systolic, Diastolic Blood Pressure.**

Clinical trial registration: CTRI/2019/03/018293

Introduction

Delirium is defined as a disturbance in a child's awareness and attention to his/her environment with disorientation and perceptual alterations, including hypersensitivity to stimuli and hyperactive motor behavior in the immediate postanaesthesia period[1-3]. The incidence of emergence delirium (ED) in children ranges from 10 to 80% and varies with the anesthetic agent[4]. It is a significant cause of increased anxiety among parents and caregivers. The average duration of occurrence of Emergence Delirium ranges from 10-60 minutes. Patients are often kicking and tilting, holding their head backward, no eye contact with the surrounding person, and are inconsolable. The psychological immaturity of children and their lack of adaptation to the perioperative anxiety might also participate in the genesis of the Emergence Delirium[5,6].

Recent studies have found that many sedatives and analgesics such as Propofol, Ketamine, Midazolam, Clonidine, Dexamethasone, Fentanyl, etc., are administered systemically or regionally were efficient in the prevention of ED[7]. Among them, Dexmedetomidine is superior in its action with lesser adverse effects[8]. Although Dexmedetomidine might increase the duration of the post-anesthesia care unit (PACU) stay of a patient, the combination of analgesic, anesthetic, and postoperative antiemetic properties make it of great interest in the prevention of Emergence Delirium[9].

As per the literature available, most of the studies described the ED either in Sevoflurane or Desflurane and reducing its incidence with either Dexmedetomidine or other drugs[9,10]. So far, no randomized controlled trial (RCT) could be found that compared the effect of Dexmedetomidine on ED between children who received either sevoflurane or desflurane for maintenance of anesthesia. Until now the incidence of ED after Sevoflurane or Desflurane has been compared in underpowered studies that can yield findings that are opposite to any true effect. We therefore aimed to compare the incidence of ED after sevoflurane and desflurane in a well-designed RCT with an adequate sample size.

Methods

The present study was registered with the Clinical Trials Registry of India (CTRI number: CTRI/2019/03/018293) following clearance by the institutional ethical committee. After obtaining written informed consent from parents, 152 children of age group 1-6 yrs & American Society of Anesthesiologists (ASA) grade I & II undergoing elective ophthalmic (vitreoretinal, corneal, glaucoma and cataract) surgeries lasting for 30-90 minutes duration requiring GA with supraglottic devices were included in this prospective, double-blind, randomized study.

Patients with a history of allergy to anesthetic drugs, previous episodes of ED, seizures, endocrine disorders, liver, renal dysfunction, psychiatric illness, emergency surgeries, and craniofacial abnormalities, including difficult airway, were excluded.

All children were kept fasting according to the standard American Society of Anesthesiologists nil per os guidelines and pre-medicated with oral midazolam 0.5 mg/kg (mixed in 5-10 ml of 5% dextrose solution) 30 minutes before surgery. Intravenous (IV) access was obtained after applying EMLA cream 30 minutes before surgery. Routine anesthetic monitors included electrocardiogram (ECG), non-invasive blood pressure (NIBP), pulse oximetry (SPO_2), end-tidal carbon dioxide ($ETCO_2$). All children were pre – oxygenated with 100% oxygen (5-6 liters).

All children were induced by standard anesthesia practice with intravenous Propofol 2-2.5 mg/kg, fentanyl 0.5-1 mcg/kg, atracurium 0.5 mg/kg, and ventilated with 100% oxygen for 3 minutes. A supraglottic device (Ambu LMA) of appropriate size (based on the child's weight and manufacture's guidelines) was inserted to maintain the airway. In all children, ventilation was maintained by pressure control mode keeping end-tidal carbon dioxide in the range of 33 – 35 mmHg.

After induction, all children were randomized into two groups (Group S and Group D) based on a computer-generated random number table. In Group S, children received Sevoflurane (2-4%, keeping minimum alveolar concentration (MAC) of 1-1.5); In Group D, children received Desflurane (6-10%, keeping MAC of 1-1.5). As per the designated group, anesthesia was maintained on Sevoflurane / Desflurane in a mixture of oxygen and air (50:50), keeping MAC of 1-1.5 in both the groups. Intraoperative analgesia was maintained by boluses of fentanyl (0.3 to 0.5 mcg/kg) when heart rate (HR) or blood pressure (BP) was more than 20% of baseline. If HR and NIBP were still more than 20% of baseline, IV paracetamol (10 mg/kg) was administered.

Intraoperative muscle relaxation was maintained using atracurium 0.2-0.3 mg·kg-1 boluses as required, to keep the train-of-four ratio (Kinemyography (KMG), NMT-Mechanosensor, Datex Instrumentations Inc., Madison, WI, USA) between 0.1 and 0.4. Intra-operative hypotension, i.e., mean arterial pressure (MAP) of less than 20% of baseline, was treated by boluses of intravenous ephedrine (1.5-3 mg) and /or i. .v fluids (2-3 ml/kg). Intra-operative bradycardia, i.e., HR less than 20% of baseline, was treated with intravenous atropine (0.3-0.6 mg).

Children in group S (Sevoflurane) were again randomized into two subgroups, S-Dex (received IV Dexmedetomidine 0.3 mcg/kg in 5 ml) and subgroup S-Saline (received Saline 5 ml) 10 minutes before the end of the surgery, based on a computer-generated random number table. Similarly, children in group D (Desflurane) were also randomized into two subgroups, subgroup D-Dex (received IV Dexmedetomidine 0.3 mcg/kg in 5 ml) and subgroup D-Saline (received Saline 5 ml) 10 minutes before the end of the surgery, based on computer-generated random

number table. An independent observer picked the randomization slip and prepared the study drugs, ensuring blinding (labeling syringe A, B, C, and D) and did not have any further involvement with the study individual. After completing the surgery, anesthesia in all children was reversed with neostigmine (0.7 mcg/kg) and glycopyrrolate (0.02 mg/kg), and AMBU LMA was removed when children were fully awake and generate adequate tidal volume. Data collection was done by personnel blinded to the allocated patient group.

In the PACU, all children were monitored for HR, NIBP, O_2 saturation every 15 minutes. An independent observer who was unaware of group allocation recorded the degree of agitation using Pediatric Emergence Delirium (PAED) scale and pain by Face, Legs, Activity, Cry and Consolability (FLACC) scale on shifting to recovery, at 5 minutes, at 15 minutes, at 30 minutes and 60 minutes after completion of surgery. A PAED score of more than 10/20 was considered as delirium. If agitation was present, the first measure taken was to console the child by parents. If this technique failed, fentanyl 0.3 mcg/kg was administered intravenously. If the FLACC score was more than 5 (0-10), fentanyl (0.3-0.5 mcg/kg) was administered intravenously. Postoperative nausea and vomiting were assessed by VAS score in all children, and if any child had either nausea lasting for 10 minutes or any episode of vomiting, he/she was treated by injection ondansetron (0.1 mg/kg) intravenously. The discharge criteria from PACU were based on Modified Aldrete Scoring, and all patients were discharged from PACU when the score is more than 9.

The primary outcome of the present study was to find out the effect of Dexmedetomidine on the incidence of postoperative ED after Sevoflurane and Desflurane anesthesia in children undergoing elective ophthalmological surgeries. The secondary outcome was to evaluate the effect of Dexmedetomidine on perioperative hemodynamic variables (HR, BP) and recovery profile of the patients, pain scoring, the requirement of rescue analgesics, and time to discharge.

Statistical analysis

Taking the incidence of ED with and without Dexmedetomidine in children induced with Sevoflurane 10 % vs. 35 % in a two-sided test with type I error or α probability level 5% and 90% power, we required 73 subjects each in Dexmedetomidine and Saline[11]. Accordingly, about 152 patients were proposed for the study. Sample size calculation was done to find a difference of 25% using the following formula[12].

$$N_1 = \left\{ z_{1-\alpha/2} * \sqrt{\bar{p} * \bar{q} * (1 + \frac{1}{k})} + z_{1-\beta} * \sqrt{p_1 * q_1 + (\frac{p_2 * q_2}{k})} \right\}^2 / \Delta^2$$

$$q_1 = 1 - p_1$$

$$q_2 = 1 - p_2$$

$$\bar{p} = \frac{p_1 + kp_2}{1 + K}$$

$$\bar{q} = 1 - \bar{p}$$

$$N_1 = \left\{ 1.96 * \sqrt{0.22 * 0.78 * (1 + \frac{1}{1})} + 1.64 * \sqrt{0.1 * 0.9 + (\frac{0.35 * 0.65}{1})} \right\}^2 / 0.25^2$$

$$N_1 = 73$$

Data collected during the study was compiled using a Microsoft Excel spreadsheet. Continuous data were presented as mean ± standard deviation (SD) and analyzed using unpaired t-test; categorical data were presented as frequency and percentage and analyzed by Pearson's chi-squared test ($\chi 2$) or Fisher's Exact test. The incidence of ED was compared between Dexmedetomidine and Saline group using a Chi-Square test. A $P < 0.05$ value was considered statistically significant.

Results

One hundred and fifty-two children were assessed for eligibility and randomized into four groups; group S-Dex (n=38), group S-Saline (n=38), group D-Dex (n=38), and group D-Saline (n=38) (Fig.1). No significant difference was seen in the age, gender, and weight between S-saline and S-Dex and D-saline and D-Dex (p-value >0.05) (Table 1).

There was a significant difference in the PAED and FLACC scores between S-saline/D-Saline and S-Dex/D-Dex on shifting to recovery at 5 minutes, 15 minutes, 30 minutes, and 60 minutes (p-value < 0.05). PAED and FLACC scores were significantly higher in the S-Saline/D-Saline group than the S-Dex/D-Dex group (Table 2). Taking the cut off of 10/20 of the PAED score for ED, the presence of ED was significantly less with the use of Dexmedetomidine at 5,15 and 30 minutes with no patients showing ED at 60 minutes in any group[13]. There was no significant difference between the PAED scale and FLACC scores at different time intervals between age < 1 year or > 1 year.

There was a significant difference in the incidence of ED between S-Saline/S-Dex and D-Saline//D-Dex at 5, 15, and 30 minutes (p-value <0.05). At 60 minutes, none of the patients had ED in all the groups (Table 3, Figure 2). No significant difference was seen in the incidence of ED between females and males at all-time intervals.

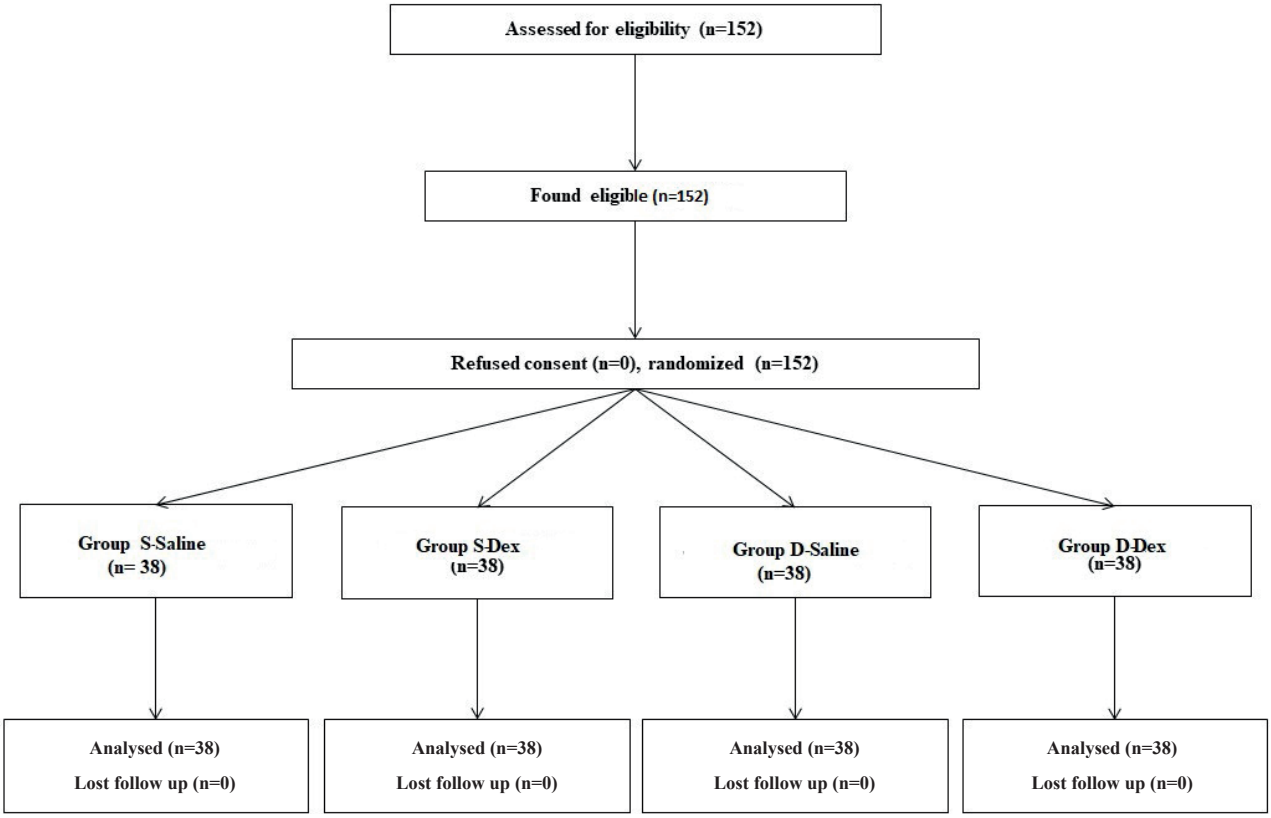

Fig. 1 — Consort flow diagram.

Table I. — Demographic Variable.

Variables	Group		P-value	Group		P-value
	S-Saline (n=38)	S-Dex (n=38)		D-Saline (n=38)	D-Dex (n=38)	
Age in years (Mean ± SD)	3.63 ± 1.60	3.68 ± 1.77	0.882	3.4 ± 1.64	3.60 ± 1.48	0.540
Male/Female	18/20	19/19	0.818	16/22	19/19	0.490
Weight in kg (Mean ± SD)	13.05 ± 4.53	13.74 ± 4.53	0.456	12.34 ± 4.43	12.90 ± 3.97	0.492

Table II. — Comparison of PAED & FLACC scale between S-Saline & S-Dex And D-Saline & D-Dex groups.

Variables	Group		P-value	Group		P-value
	S-Saline (n=38)	S-Dex (n=38)		D-Saline (n=38)	D-Dex (n=38)	
PAEDS scale On shifting to recovery	13.21 ± 3.63	7.08 ± 2.71	<.0001	12.60 ± 3.88	7.10 ± 2.66	<.0001
PAEDS scale at 5 minutes	11.92 ± 3.96	5.74 ± 2.99	<.0001	11.18 ± 4.09	5.66 ± 2.72	<.0001
PAEDS scale at 15 minutes	9.60 ± 3.14	4.11 ± 2.40	<.0001	9.13 ± 3.23	4.37 ± 2.12	<.0001
PAEDS scale at 30 minutes	6.76 ± 3.14	2.32 ± 1.44	<.0001	6.40 ± 3.12	2.71 ± 1.75	<.0001
PAEDS scale at 60 minutes	2.76 ± 1.32	1.00 ± 0.87	<.0001	2.97 ± 1.57	1.63 ± 1.15	0.0002
FLACC scale On shifting to recovery	4.29 ± 2.36	0.66 ± 1.91	<.0001	3.60 ± 2.43	0.66 ± 1.76	<.0001
FLACC scale at 5 minutes	3.90 ± 2.30	0.68 ± 1.80	<.0001	3.29 ± 2.18	0.63 ± 1.68	<.0001
FLACC scale at 15 minutes	2.66 ± 1.68	0.32 ± 0.96	<.0001	1.97 ± 1.48	0.24 ± 0.79	<.0001
FLACC scale at 30 minutes	1.50 ± 1.11	0.05 ± 0.23	<.0001	0.87 ± 0.88	0.08 ± 0.36	<.0001
FLACC scale at 60 minutes	0.71 ± 0.61	0.00 ± 0.00	<.0001	0.42 ± 0.55	0.00 ± 0.00	<.0001

Table III. — Comparison of Emergence Delirium between S-Saline & S-Dex And D-Saline & D-Dex groups.

Variables	Group		Total	P-value	Group		Total	P-value
	S-Saline (n=38)	S-Dex (n=38)			D-Saline (n=38)	D-Dex (n=38)		
At 5 minutes	30 (78.95%)	4 (10.53%)	34 (44.74%)	<.0001	28 (73.68%)	5 (13.16%)	33 (43.42%)	<.0001
At 15 minutes	26 (68.42%)	3 (7.89%)	29 (38.16%)	<.0001	23 (60.53%)	2 (5.26%)	25 (32.89%)	<.0001
At 30 minutes	6 (15.79%)	0 (0.00%)	6 (7.89%)	0.025	8 (21.05%)	0 (0.00%)	8 (10.53%)	0.005
At 60 minutes	0 (0.00%)	0 (0.00%)	0 (0.00%)	-	0 (0.00%)	0 (0.00%)	0 (0.00%)	-

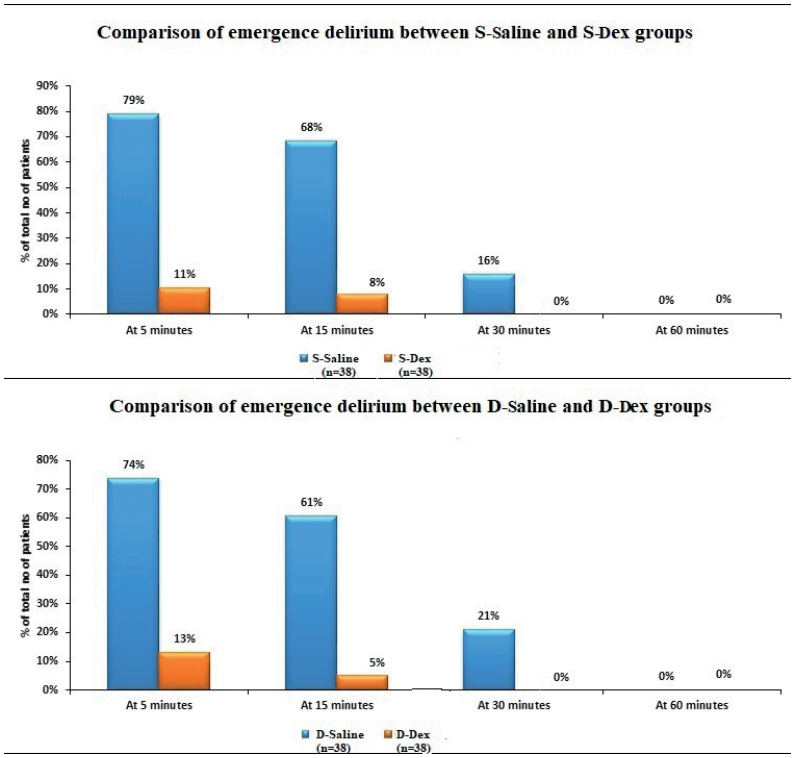

Fig. 2 — Comparison of emergence delirium between S-saline & S-dex And D-saline & D-dex groups.

In our study, a significant difference was seen in the requirement of analgesia between the S-Saline and S-Dex group (p-value = 0.005). No significant difference was seen in the analgesic requirement between D-Saline and D-Dex group (p-value = 0.153). No significant difference was seen in the PONV between S-Saline/ S-Dex group (p-value = 0.674) and D-Saline and D- Dex group. (p-value = 0.493) (Table 4).

Table IV. — Comparison of analgesic requirement AND Postoperative Nausea Vomiting between S-Saline & S-Dex And D-Saline & D-Dex groups.

Variables	Group		Total	P-value	Group		Total	P-value
	S-Saline (n=38)	S-Dex (n=38)			D-Saline (n=38)	D-Dex (n=38)		
No Analgesic requirement	24 (63.16%)	35 (92.11%)	59 (77.63%)	0.005	31 (81.58%)	36 (94.74%)	67 (88.16%)	0.153
Analgesic requirement	14 (36.84%)	3 (7.89%)	17 (22.37%)		7 (18.42%)	2 (5.26%)	9 (11.84%)	
PONV Absent	34 (89.47%)	36 (94.74%)	70 (92.11%)	0.674	36 (94.74%)	38 (100.00%)	74 (97.37%)	0.493
PONV Present	4 (10.53%)	2 (5.26%)	6 (7.89%)		2 (5.26%)	0 (0.00%)	2 (2.63%)	

The mean value of time to discharge from PACU in the S-Saline group was 60.4 ± 2.43 minutes and in the S-Dex group was 75.26 ± 11.02 minutes. The mean value of time to discharge from PACU in the D-Saline group was 60.4 ± 2.43 minutes and in the D-Dex group was 74.6 ± 12.54 minutes. So, PACU's discharge time was significantly longer in S-Dex and D-Dex groups than in the S-Saline and/D-Saline group. (p-value <.0001)

There was no significant difference in the mean value of the heart rate between S-Saline and S-Dex at all time intervals (p-value >0.05). Similarly, there was no significant difference seen in the mean heart rate between D-saline and D-dex at all the time intervals except at extubation (p-value >0.05). At extubation, HR was significantly higher in the D-Saline group as compared to the D-Dex group (101.5 ± 13.27 bpm vs 88.74 ± 12.35 bpm, p =0.0001).

There was no significant difference seen in the mean systolic BP (SBP) between S-Saline and S-Dex at baseline, at induction, after 1 hour, and at extubation (p-value >0.05). At intubation, after 15 and 30 minutes from baseline systolic BP was significantly lower in the S-Dex group than the S-Saline group (p-value <0.05). No significant difference was observed in the mean SBP between D-Saline and D-Dex at all the time intervals. (p-value >0.05)

There was no significant difference seen in the mean diastolic BP (DBP) between S-Saline and S-Dex group at baseline and at extubation (p-value >0.05). At induction, at intubation, at 15 minutes, 30 minutes, and 60 minutes diastolic BP was significantly lower in the S-Dex group as compared to the S-Saline group (p-value <0.05). No significant difference was seen in the mean DBP between D-Saline and D-Dex at baseline, induction, at intubation, at 15 minutes from baseline, and at extubation (p-value >0.05). At 30 minutes and 60 minutes from baseline, the mean DBP was significantly lower in the D-Dex group than the D-Saline group(p value<0.05).

Discussion

In our study, the reduction in ED incidence after adding Dexmedetomidine was more in both S-Dex and D-Dex groups, i.e., 68 % and 60 %, respectively. This was even more than the study published by B Ghai et al.[11]., which showed a reduction in the incidence of ED by 40% with the use of Dexmedetomidine. Similarly, Di M et al.[14] showed a decrease in the incidence of ED by 44% with the use of Dexmedetomidine. In a similar study by Shi et al.[9], the incidence of ED (PAED ≥ 10) in the Dexmedetomidine group was significantly lower than that in the control group. Lin Y et al. also reported decreased PAED score in the Dexmedetomidine group compared to the normal saline (NS) group[15].In our present study, we had administered a bolus of Dexmedetomidine to evaluate the reduction in the incidence of Emergence Delirium in Sevoflurane and Desflurane anesthesia. This is similar to the study by Begam et al.[8], who compared intravenous Dexmedetomidine bolus 0.4 μg/kg over 10 minutes or its continuous infusion of 0.4 μg/kg/h to prevent ED in children after Sevoflurane anesthesia. They found that the PAED score was significantly lower in the bolus group than the infusion group. The PAED scale, developed in 2004 for children > 2 yr (2-6 years) of age, has been psychometrically evaluated and is used most commonly. This scale has limitations, including the subjective nature of some assessments, inter-rater variability, a high false-positive rate, and qualifying behaviors that overlap with other negative postoperative diagnoses, i.e., pain[7].

The analgesic effect of Dexmedetomidine is mediated by binding the drug to α2-receptors of the CNS, possibly those of the Locus Coeruleus and spinal cord. It thus blocks pain signal propagation and induces analgesic effects. We found that postoperative pain was significantly reduced using Dexmedetomidine among the S-Dex and D-Dex groups (P<0.0001) at 5, 15, 30, and 60 minutes. Taking the cutoff of 5 on the FLACC scale, the analgesic requirement was significantly reduced in group S (Sevo) but was comparable in Group D (Des). Many such studies also support this finding of our research. A similar study by Shi et al.[9] reported that the FLACC score was significantly lower in the Dexmedetomidine group than the FLACC score in the control group. In a study by Jain et al.[10], there were substantially lower FLACC scores in those who received Dexmedetomidine @ 0.3 mcg/kg than in those who received Dexmedetomidine @ 0.15 mcg/kg. Similarly, Kim et al.[16] indicated that continuous intraoperative infusion of low-dose Dexmedetomidine @ 0.2 mcg/kg/h could reduce the postoperative pain of ED following Desflurane anesthesia in pediatric patients undergoing strabismus surgery.

However, in contrast to our study's finding, Ghai B et al.[11] reported that no differences were found between the Dexmedetomidine group and Control groups with respect to pain, and no patient in any of these groups attained a pain score >5 requiring rescue medication. This difference may be attributed to the fact that the surgeon administered 0.1 mL/kg of 0.5 percent bupivacaine in the sub-Tenon block in their study.

Postoperative nausea and vomiting is a frequent complication in the immediate postoperative period. Possible explanations for the lower incidence of PONV with the use of Dexmedetomidine may be because it reduces noradrenergic activity with binding to α2 presynaptic inhibitory adrenergic receptors in the Locus Coeruleus; this can lead to an antiemetic effect. It may also be associated with the overall reduction in sympathetic outflow and catecholamine release caused by Dexmedetomidine. A high sympathetic tone and release of catecholamine may trigger PONV[17]. We evaluated PONV with a VAS score and found no significant difference with the use of Dexmedetomidine. In our study, it may be related to the timing of Dexmedetomidine dose, type of surgery, and most importantly, because we observed PONV only in the recovery room. In studies by Jain et al.[10] and Shi et al.[9], postoperative complications such as nausea and vomiting were comparable. However, Begum et al.[8] reported that Dexmedetomidine caused a significant decrease in the incidence of the occurrence of PONV in children with Sevoflurane anesthesia.

In our study, the discharge time was significantly increased using Dexmedetomidine in both the S-Dex and D-Dex groups. This could be due to the excessive sedation associated with Dexmedetomidine and because it increases emergence time and extubation time. It has several useful properties, but its half-life of 2-3 hours can be a disadvantage in ambulatory procedures as it can prolong sedation and thus delay discharge from the hospital[18]. This finding of our study is also supported by similar research by Zhu et al.[19], who observed that compared to the placebo group, time to discharge from PACU in the Dexmedetomidine group was also significantly increased. This contrasts with the study by Shi et al.[9], who observed no difference between the two groups in PACU length of stay after extubation. Also, in the study by Ghai et al.[11], the discharge time was significantly longer in the control group compared to the Dexmedetomidine group. It may be attributed to a larger number of children receiving rescue medication (intravenous fentanyl) postoperatively in the control group.

Administration of Dexmedetomidine can be accompanied by a decrease in heart rate and blood pressure[19].In our study, HR was comparable with and without Dexmedetomidine in both S (Sevo) and D(Des) groups except during extubation, where the patients with D-Dex had significantly lower HR than Desflurane alone group. Like in our study, the comparable equivocal effect on HR was seen in other studies also. Ghai et al.[11] reported that there was no significant reduction of HR was noted in the three groups (Dexmedetomidine: 0.15 mcg/kg, 0.3 mcg/kg, and normal saline)[18]. In a study by Shi et al.[9], when compared with Control, the Dexmedetomidine group displayed a significant decrease in HR for 15 minutes after study drug administration and at the time of extubation. In another study by Begum et al.[8], HR was similar in both groups.

Our study found that the use of Dexmedetomidine caused a significant decrease in the SBP with Sevoflurane; and a considerable reduction in the DBP with both Sevo and Des at various time intervals. This may be because Dexmedetomidine evokes a biphasic blood pressure response, i.e., a short, hypertensive phase and subsequent hypotension. Two different α2-AR subtypes mediate the two phases: the α-2B AR is responsible for the initial hypertensive phase, whereas the α2A-AR mediates hypotension[20]. Similar to our study, Shi et al.[9] also reported that SBP was lower in the Dexmedetomidine group than in the control group at the same two-time points (for 15 minutes after study drug administration and at the time of extubation). There was no significant difference between the two groups in DBP at any time. In contrast to our study's finding, Ghai et al.[11] reported no substantial reduction in blood pressure in the Dexmedetomidine and Control group.

Our study results suggest that the addition of IV Dexmedetomidine to either Sevoflurane / Desflurane significantly decreased ED occurrence among children. Apart from this, even the addition of Dexmedetomidine to Sevoflurane/ Desflurane reduced postoperative pain. Other side effects like PONV showed no significant change, but the time to discharge from PACU was increased considerably with the addition of Dexmedetomidine in both groups.

There were many limitations to the present study. We studied relatively healthy (ASA I-II) children (1–6 years) and excluded patients with a history of airway problems as Dexmedetomidine may subject these children to unacceptably higher risks for postoperative airway complications. In this study, PONV was assessed using the VAS score. However, the use of other scores may show some varied results in terms of nausea and vomiting. We also did not assess the residual sedation during the recovery period. But we compared the discharge time in different groups. In the present study, pain was measured using FLACC scale. This scale may be subject to observational bias, and that behavior may not represent only pain, but can be confounded by

anxiety or other causes of distress. We used PAED scale to assess agitation in this study. This scale has some limitations, including the subjective nature of certain evaluations, inter-rater variability, a high false-positive rate and qualifying behaviors that overlap with other postoperative diagnoses such as pain.

Conclusions

Dexmedetomidine can significantly decrease emergence delirium, postoperative pain with minimal changes in nausea and vomiting when added to Sevoflurane and Desflurane anesthesia. The hemodynamic side effects like hypotension can be increased, subsequently increasing the discharge time of the patients. The findings of this trial support the beneficial effect of IV Dexmedetomidine administered after the induction of anesthesia for the prevention of ED in pediatric patients anesthetized with Sevoflurane and Desflurane. Thus, Dexmedetomidine might be used safely to prevent postoperative behavioral changes in children.

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