Original Article

Effect of dexmeditomidine on postoperative junctional ectopic tachycardia after complete surgical repair of tetralogy of Fallot: A prospective randomized controlled study

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ABSTRACT

Introduction: Incidence of junctional ectopic tachycardia (JET) after repair of tetralogy of Fallot (TOF) is 5.6–14%. Dexmeditomidine is a α-2 adrenoceptor agonist modulates the release of catecholamine, resulting in bradycardia and hypotension. These effects are being explored as a therapeutic option for the prevention of perioperative tachyarrhythmia. We undertook this study to examine possible preventive effects of dexmedetomidine on postoperative JET and its impact on the duration of ventilation time and length of Intensive Care Unit stay. **Methods:** After obtaining approval from the hospitals ethics committee and written informed consent from parents, this quasi-randomized trial was initiated. Of 94 patients, 47 patients received dexmedetomidine (dexmedetomidine group) and 47 patients did not receive the drug (control group). **Results:** Dexmedetomidine group had more number of complex variants like TOF with an absent pulmonary valve or pulmonary atresia ($P = 0.041$). Hematocrit on cardiopulmonary bypass (CPB), heart rate while coming off from CPB and inotrope score was significantly low in the dexmedetomidine group compared to control group. The incidence of JET was significantly low in dexmedetomidine group ($P = 0.040$) compared to control group. **Conclusions:** Dexmedetomidine may have a potential benefit of preventing perioperative JET.

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INTRODUCTION

The incidence of junctional ectopic tachycardia (JET) is around 5.6–14% after operations for congenital heart diseases (CHDs) especially TOF repairs and the reasons are multifactorial.[1,6,7] Various centers have their protocols to reduce the incidence of JET after repair of CHDs; like the use of beta-blockers, cooling of the patient to a core temperature of around 35°C and use of magnesium sulphate.[8-10] Dexmedetomidine is used as a sedative and analgesic pediatric Intensive Care Units (ICUs). It is a selective α-2 adrenoceptor agonist that has sedative, analgesic and anxiolytic properties with associated opioid and anesthetic sparing effects.[11-14] The agonistic action on the α-2 adrenoceptors modulates the release of catecholamines, resulting in a sympatholysis with negative chronotropic and dromotropic effects.[15,16] These sympatholytic effects are being studied as a therapeutic option for the prevention and treatment of various perioperative tachyarrhythmia's associated with CHD.[2,15,16] These rhythm disturbances

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can cause significant hemodynamic instability and increases morbidity, particularly after cardiopulmonary bypass (CPB) period.[17] There are some reports and few studies of usage of dexmedetomidine to prevent JET after congenital heart surgery.[2-4] The aim of this study was to examine the possible preventive effect of dexmedetomidine on junctional ectopic tachycardia after TOF surgery and to study its impact on various morbidity indicators like duration of ventilation time (VT) and length of ICU stay.

METHODS

The study was initiated after obtaining permission from the institutional ethics committee. Informed parental consent was taken for anesthesia, surgical procedure and use of dexmedetomidine. This was a prospective, quasi-randomized controlled study, done in the period between June 2010 and June 2013. Of the 94 patients included in this study, 47 patients received dexmedetomidine (dexmedetomidine group) and 47 did not receive dexmedetomidine (control group). All the patients diagnosed to have tetralogy of Fallot (TOF) and needed complete intracardiac repair were included in the study. Induction of anesthesia was done in the operating room (OR).

Exclusion criteria

- Patients with preoperative arrhythmias were excluded from this study
- Patients coming in cyanotic spell and taken for emergency surgery were excluded from our study
- Patients who developed complete heart block postrepair were excluded from study.

After insertion of arterial blood pressure (ABP) and central venous pressure (CVP) monitoring lines, every second patient was started on dexmedetomidine infusion at 0.75 mcg/kg/h after a loading dose of 1 mcg/kg over 15 min. This infusion was continued during CPB and then in the postoperative period for ~48 h for analgesia and sedation as per the need. Top up bolus doses of fentanyl and midazolam were given according to need in study group. Control group was started on 2 mcg/kg/h of fentanyl infusion during same period along with bolus doses of midazolam and fentanyl as per need. Sedation was monitored with Richmond Agitation-Sedation Scale. Data collection was done in the OR and in pediatric cardiac Intensive Care Unit (PCICU) with the help of Drager - Infinity Delta XL, which provide facility to record the events.

Anesthesia, cardiopulmonary bypass and surgical management

After premedicating with injection midazolam 0.1 mg/kg and injection ketamine 1 mg/kg intravenously in the preoperative waiting area, all children were shifted to the OR. Endotracheal intubation was done after giving injection fentanyl and injection vecuronium. Dexmeditomidine and fentanyl infusions were started in standard doses as mentioned above. All the surgeries were conducted with similar techniques by same team of surgeons, anesthesiologist and perfusionist during the study period under standard CPB techniques. Cardiac arrest was achieved with modified del-Nido cardioplegia in all patients. Weaning from CPB was done with the inotropic support of adrenaline 0.04 mcg/kg/min and milrinone 0.5 mcg/kg/min in all cases as a protocol. Rescue inotropes were added depending on the requirement.

Standard 12-lead electrocardiogram (ECG) was recorded in all patients preoperatively and then immediately after surgery in the ICU. Continuous monitoring of ECG, ABP, CVP, end-tidal carbon dioxide, and oxygen saturation of pulsatile tissue was done with Drager monitors.

Diagnostic criteria for JET included the following: (1) Tachycardia with QRS similar to sinus rhythm QRS, (2) a ventricular rate more than 170 beats/min, (3) atrioventricular dissociation with or without hemodynamic compromise, and (4) a ventricular rate faster than the atrial rate.[8]

When JET was suspected on the monitor, it was confirmed with rhythm strip in OR and 12 lead ECG and or atrial-wire ECG if the diagnosis was not confirmed in the PCICU.

Patient demographics, intraoperative variables like total aortic cross-clamp (AXC) time, CPB time, lowest temperature during CPB, occurrence of JET after coming off from CPB and postoperative data like maximum vasoactive inotrope score (VIS), VT and ICU stay were recorded in the OR and PCICU.

Maximum VIS as described by Davidson *et al*. is calculated as,

Inotropic score= (dopamine × 1) + (dobutamine × 1) + (adrenaline × 100) + (noradrenaline × 100) + (milrinone × 10). Dosages of above drugs were in mcg/kg/min.[18]

Statistical analysis used

All data are expressed as mean and standard deviation. Categorical data was compared with Pearson Chi-square *t*-test and other data compared with independent two-sample *t*-test. The difference was considered statistically significant when $P < 0.05$.

RESULTS

From preoperative variables, gender distribution, mean age, mean weight, right ventricular outflow tract (RVOT) gradient, preoperative heart rate (HR) and preoperative use of propranolol were recorded and they found out comparable in two groups ($P > 0.05$) [Table 1]. There were four patients with absent pulmonary valve and four patients with pulmonary atresia in the dexmedetomidine group while one each in the control group. These cases with absent pulmonary valve and pulmonary atresia require prolonged CPB and AXC time and thus making them more complex within TOF group. This difference between two groups with a higher incidence of complicated patients in dexmedetomidine group was statistically significant ($P = 0.044$) [Table 1]. Mean pulmonary artery annulus size was comparable in two groups ($P = 0.800$) [Table 1]. Twenty-eight (59.57%) patients in dexmedetomidine group and 26 (55.31%) patients in control group underwent transannular patch (TAP) as a part of the complete surgical repair. This difference in performance of TAP in two groups was insignificant [Table 2]. Fourteen patients underwent RVOT patch, 2 pulmonary valve replacement, 2 conduit repair and one underwent TAP with the monocuspid valve in dexmedetomidine group. So, total 19 (40.42%) patients in dexmedetomidine group underwent a procedure which minimizes pulmonary valve regurgitation compared to 21 (44.68%) patients in the control group. Though more patients in the control group had valve preserving repairs or regurgitation minimizing repairs, difference was not significant [Table 2]. Tricuspid valve detachment (TVD) was done in 8 (17%) patients in dexmedetomidine group compared to 9 (19.14%) in control group. It is done for ease of ventricular septal defect (VSD) closure and it was comparable in two groups ($P = 0.788$) [Table 2]. Lowest hematocrit during CPB was low in dexmeditomidine group compared to control group and difference was statistically significant ($P = 0.005$) [Table 2]. Reasons for this difference in hematocrit are not clear. CPB time and AXC time was longer in control group compared to dexmedetomidine group. HR while coming off from CPB was significantly lower in dexmedetomedine group compared to control group ($P = 0.009$). VIS was lower in dexmedetomidine group compared to control group ($P = 0.024$). Cooling of these patients on CPB that is lowest temperature on was almost same in two groups [Table 2]. A total of four patients in the dexmedetomidine group had JET compared to the 11 patients in the control group in the postoperative period. Overall incidence of JET was significantly low in dexmedetomidine group ($P = 0.040$) [Table 3]. Mean VT was 26.2 and 27.08 h in dexmedetomidine and control group respectively ($P = 0.867$) [Table 3]. Mean duration of ICU stay (104.5 h) was more in dexmedetomidine group compared to control group (99.23 h), but the difference was statistically not significant ($P = 0.659$) [Table 3]. There was one mortality each in both the groups in the perioperative period ($P = 0.963$) [Table 3].

Table 1: Preoperative variables

	Dexmedetomidine group (*n*=47)		Control group (*n*=47)		*P*
	Mean	SD	Mean	SD	
Gender distribution (male:female)	29:18		27:20		0.600*
Age (weeks)	152.27	249.655	120.98	161.348	0.469**
Weight (kg)	11.69	10.962	10.19	6.744	0.422**
RVOT gradient (mmHg)	70.59	20.250	63.89	10.741	0.075**
PA annulus (Z score)	−2.1164	1.88317	−2.0122	2.08343	0.800**
Preoperative HR	118.56	17.937	126.53	13.385	0.066**
Preoperative propranolol (%)	34		49.0		0.078*
Diagnosis (%)					
TOF	82.97		95.77		0.044*
TOF with APV	8.51		2.12		
TOF with PA	8.51		2.12		

*Pearson Chi-square, **Independent samples test. SD: Standard deviation, RVOT: Right ventricular outflow tract, PA: Pulmonary artery, TOF: Tetralogy of Fallot, APV: Absent pulmonary valve, HR: Heart rate

Table 2: Intraoperative variables

	Dexmeditomidine group (*n*=47)		Control group (*n*=47)		*P*
	Mean	SD	Mean	SD	
Trans annular patch (%)	59.57		55.31		0.242*
RVOT patch and other valve sparing procedures (%)	36.17		44.68		0.087*
Tricuspid valve detachment (%)	17		19.14		0.941*
Lowest temperature	25.529	1.4194	24.980	1.6265	0.086**
Lowest hematocrit	27.87	2.841	31.12	3.655	0.005**
Aortic cross clamp time	97.36	33.519	109.49	33.304	0.082**
CPB time	164.84	44.245	173.10	52.529	0.414**
HR while coming off CPB	137.78	19.113	148.06	18.196	0.009**

*Pearson Chi-square, **Independent samples test. SD: Standard deviation, RVOT: Right ventricular outflow tract, CPB: Cardiopulmonary bypass, HR: Heart rate

Table 3: Postoperative variables

	Dexmedetomidine group (*n*=47)		Control group (*n*=47)		*P*
	Mean	SD	Mean	SD	
Inotrope score (maximum)	11.07	5.043	13.39	4.738	0.024**
JET incidence (%)	8.51		23.40		0.040*
Ventilation time	26.27	18.678	27.08	27.071	0.867**
ICU stay	104.55	64.224	99.23	50.629	0.659**
Mortality (%)	2.2		2.1		0.963*

*Pearson Chi-square, **Independent samples test. JET: Junctional ectopic tachycardia, ICU: Intensive Care Unit, SD: Standard deviation

DISCUSSION

Perioperative atrial and junctional tachyarrhythmias are difficult to manage. Currently, available antiarrhythmic drugs are poorly tolerated during the post-CPB period. The incidence of arrhythmias especially JET is high after TOF repairs and is difficult to control.[1,7,17] This high incidence is correlated to various factors like small age at operation, long CPB and AXC time, low hematocrit during CPB, TVD, ventriculotomy, handling of tissue during VSD closure and use of inotropes.[9,19-22] Recently people have started using dexmedetomidine in postcardiac surgery scenarios for sedation and analgesia. In our prospective quasi-randomized trial, HR while coming off from CPB was significantly low in dexmedetomidine group along with this incidence of JET was also significantly low in dexmedetomidine group ($P = 0.04$). Prevention of tachyarrhythmias especially JET is the key to preventing liquid crystal on silicon, and it should be the priority in managing these patients in the postoperative period. There are various methods of reducing occurrence of JET after CHD surgeries like minimization of CPB and AXC time, use of MgSO4 on CPB, avoidance of electrolyte disturbances, optimization of inotropic support, avoiding hyperthermia and use of adequate sedatives and analgesics, etc.[8-10,23] Use of sedatives and analgesics is a necessity in managing ventilated patient in the postoperative period. There are many options, but they have their own problems like respiratory depression, hypotension, bradycardia, development of tolerance, dependence, etc. We are yet to have an ideal sedative and analgesic for ICU in pediatric cardiac patients. Dexmedetomidine is recently being used in many ICUs because of its many near ideal properties like lack of respiratory depression, good sedative, modest analgesic, and anxiolytic properties.[11-14] Various trials demonstrated that it can be used with minimal adverse effects after pediatric cardiac surgeries.[2,3,5] Its main side-effect is bradycardia; however this side-effect is being studied for prevention and treatment of various supraventricular arrhythmias like JET after surgeries for CHDs like TOF. Earlier, a meta-analysis by Wijeysundera *et al*. showed that the use of α-2 agonists, in general, had no effect on the incidence of supraventricular arrhythmias,[4] but another study by Kamibayashi *et al*. shows that dexmedetomidine can prevent epinephrine/halothane induced ventricular tachycardia.[24] Kamibayashi *et al*. suggested that the antiarrythmic property of dexmedetomidine is related to activation of the α-2 adrenoreceptors. However, later studies found evidence that it is produced through its action on cerebral imidazoline receptors and an effect on the vagal nerve.[24] Two pediatric studies have found that

infants and children older than 1-year of age required an average dose of 0.29 ± 0.17 mcg/kg/h (0.1–0.75), whereas children ≤1-year of age needed higher dose, 0.66 ± 0.26 mcg/kg/h (0.1–1.5) to achieve targeted sedation and analgesia.[3,5] During the postoperative period, JET can one of the resistant and life-threatening arrhythmias. Current treatment algorithms include removal of exacerbating factors such as pressors, use of beta-blocker, cooling of patient to maintain core temperature around 35°C and use of amiodarone[8] but ideal is to prevent it. None of the current medications are useful in preventing or reducing incidence of JET in postcongenital heart surgery situations. Hammer *et al*.,[25] found out that dexmedetomidine depressed both sinus and AV nodal function responsible for decreased incidence of JET in these patients. HR while coming off from CPB was significantly lower in the dexmedetomedine group compared to control group ($P = 0.009$) and this reduced HR while coming off from CPB is extended to reduced incidence of JET. VIS was 11.07 ± 5.043 in dexmedetomidine group and 13.39 ± 4.738 in the control group, this difference in VIS was significant with P value of 0.02. So in our study, dexmedetomidine group required less inotropes compared to control group. This reduced inotropic requirement in dexmedetomidine group can be because reduced incidence of JET in that group. Or in other words, high incidence of JET in control group required more inotropes. This can be contributed by prolonged CPB time and AXC time, but this difference was not significant. Lowest hematocrit on CPB in dexmedetomidine group was 27.87 ± 2.841 and 31.12 ± 3.655 in the control group. This difference in hematocrit in two groups was statistically significant with $P = 0.005$. Low hematocrit (<25) is associated with increased risk myocardial dysfunction leading to arrhythmias, bleeding, acute renal failure and prolonged hospital stay.[21] However, in our study mean hematocrit was more than 25 in both the groups. Though dexmedetomidine group had lower hematocrit on CPB, VT and ICU stay was the same in both groups. Thirty-four percent in dexmedetomidine group and 49% patients in the control group were on propranolol in the preoperative period. Mahmoud *et al*. showed in his recent study that preoperative use of propranolol is associated with reduced incidence of postoperative JET after surgical repair of TOF.[26] Though more patients in the control group were on propranolol compared to control group in the preoperative period, the difference was not statistically significant. The incidence of JET was high in a control group which is not corresponding with more patients on preoperative propranolol in that group. So, dexmedetomidine has clearly reduced JET in test group in-spite of lesser patients on propranolol in that group. TVD increases handling of tissue, CPB and AXC time leading to increased risk of arrhythmias.[19] TVD was comparable in this study. Valve preserving procedures like RVOT outflow patch reduce the risk of right ventricular dysfunction and arrhythmias in the immediate postoperative period.[20] The difference in the incidence of JET in valve preserving procedures was not significant in the two groups. Prolonged CPB time and AXC time are associated with increased risk of arrhythmias, VT, ICU stay and hospital stay. It increases morbidity and mortality after any cardiac surgery. CPB and AXC time was shorter in dexmedetomidine group but the difference was not significant. There was one death in each group during the perioperative period. Though there was no significant difference in VT and ICU stay in the two groups, a larger number of patients in this study could have changed our results related to VT and ICU stay.

Study limitations

- The study involves a relatively small number of patients
- Most of our patients were infants and toddlers, so the findings may not be applicable to older age groups
- We should have studied duration of JET along with incidence rather than just incidence
- Duration of hospital stay should have been included into this study.

CONCLUSIONS

This prospective randomized controlled study suggests that dexmedetomidine may have a potential benefit of preventing perioperative JET after surgical correction of TOF.

However, another large prospective study is needed to confirm its effect on VT and ICU stay.

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