Case Series

Ketodex for MRI sedation in syndromic children with congenital cardiac anomalies – A case series

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ABSTRACT

Safe paediatric sedation in a magnetic resonance imaging (MRI) suite can be challenging. The challenges intensify in uncooperative syndromic children compounded by an accentuated risk of periprocedural cardio-respiratory complications with anaesthetic sedation in this peculiarly predisposed subset. Amidst ardent debates on the ideal sedative agent for paediatric MRI, we report an encouraging application of ketamine-dexmedetomidine combination (ketodex) sedation for MRI in our case-series including syndromic children with coexistent congenital cardiac anomalies.

Key words: Dexmedetomidine, ketamine, magnetic resonance imaging, non-operating room anaesthesia, sedation

INTRODUCTION

The modern-day role of the anaesthesiologist extends beyond the operation theatre. While a resource-limited setting renders the administration of safe non-operating room anaesthesia (NORA) difficult, patient-related factors intensify the challenges.[1,2] Anaesthesia in a magnetic resonance imaging (MRI) suite mandates NORA in diverse age-groups with complex syndromes, entailing periprocedural risks.[1,3]

Management of syndromic children undergoing MRI necessitates a prudent selection of anaesthetic agent class and dose, given the combined risk of sedation induced respiratory depression and haemodynamic compromise owing to detrimental effects on the pathophysiology of the coexistent cardiac lesion.[3]

While MRI (sensitive to motion-artifacts) requires an immobile child, the ideal drug regime remains debatable.[4] We report an encouraging application of ketamine and dexmedetomidine combination (ketodex) sedation for MRI in syndromic children with congenital cardiac anomalies, focusing on the cardio-respiratory consequences, sedation quality, recovery profile and periprocedural complications.

CASE REPORT

This case series includes 10 syndromic children in the 3-5 years age-group requiring sedation for MRI brain for the assessment of neurodevelopmental delay [Table 1]. They presented over a year between

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Table 1:The demographic profile and incidence of periprocedural cardiorespiratory events

Age (years)	Gender	Syndrome	Cardiac anomaly	Duration of MRI (minutes)	Periprocedural cardiorespiratory events			
					B	H	D	A
3	M	Down syndrome	VSD	45	-	-	-	-
3	M	Holt-Oram syndrome	ASD	45	-	-	-	-
5	F	Down syndrome	VSD	50	-	-	-	-
4	F	DiGeorge syndrome	TOF	45	-	-	-	-
4	F	Down syndrome	VSD	45	-	-	+	+
3	M	TAR syndrome	TOF	45	-	-	-	-
3	F	Ellis-Van Creveld syndrome	ASD	50	-	-	-	-
3	M	Down syndrome	AVSD	50	-	-	-	-
4	M	Down syndrome	ASD+VSD	50	-	-	-	-
5	F	CHARGE association	TOF	55	-	-	-	-

(A: Airway manipulation/adjunct; ASD: Atrial septal defect; AVSD: Atrioventricularseptal defect; B: Bradycardia; CHARGE: Coloboma, heart defects, atresia choanae, growth retardation, genital abnormalities, and ear abnormalities; D: Desaturation; F: Female; H: Hypotension; M: Male; MRI: Magnetic resonance imaging; TOF: Tetralogy of Fallot; VSD: Ventricular septal defect; (-): Negative; (+): Positive)

March 2020 and February 2021 at our tertiary care centre. All patients were classified as American Society of Anesthesiologists (ASA) physical status III and underwent pre-anaesthetic evaluation.

The patients were nil per oral as per ASA guidelines. A written-informed consent was obtained from the parents after explaining the procedure and permission to use the data for research publication. The baseline vitals (heart-rate (HR), blood pressure (BP), oxygen saturation (SpO_2)) were recorded. Apnoea monitoring was done using end-tidal carbon dioxide ($EtCO_2$). Eutectic mixture of local anaesthetics (EMLA) cream was applied over dorsum of the hand. Intravenous (IV) access was secured and ketamine 1 mg/kg + glycopyrrolate 5 μg/kg was administered intravenously. Thereafter, the child was positioned and oxygen was administered via facemask at 6 L/min via the auxiliary oxygen port of the anaesthesia workstation (Dräger Fabius® MRI).

Subsequently, dexmedetomidine was administered as a bolus dose of 0.5 μg/kg IV over 10 min followed by a continuous IV infusion of 0.5 μg/kg/hour. Sedation was assessed using the Paediatric Sedation State Scale (PSSS) and MRI was commenced on achieving a target score of 2.[5] The anaesthesiologist stationed at the head end of the MRI gantry looked for facial expressions or vocalisation for scoring PSSS during the MRI. In the background of PSSS <2, the dexmedetomidine infusion was down-titrated by 20% and if PSSS >2, rescue sedation (ketamine 0.2 mg/kg IV) was administered and dexmedetomidine infusion was stepped up by 20%. HR, BP, SpO_2, $EtCO_2$ were continuously monitored and recorded (MIPM TeslaM3 monitor) every 5-min during and after the procedure.

On the completion of imaging, dexmedetomidine infusion was stopped and the time from infusion termination till first response to verbal command was categorised as time to recovery. The time from infusion termination till attainment of modified Aldrete score ≥9 was computed as time to discharge or shifting to ward. Quality of sedation was quantified by the anaesthesiologist as: optimal (rescue sedation not required), suboptimal (rescue sedation required but MRI not interrupted) and failed (MRI interrupted due to inadequate sedation and/or requiring >one dose of rescue sedation). Quality of scan was assessed by the radiologist based on imaging, as: excellent (no motion artifacts); good (minor motion artifacts not requiring repeat scan sequence) and poor (major motion artifacts or MRI interruptions, requiring ≥1 repeat scan sequence).[2]

The mean recovery time was 17.5 min and the mean time to discharge was 41.5 min [Table 2]. One patient demonstrated suboptimal sedation necessitating a rescue analgesic dose albeit image quality continued to be good [Table 2]. Peri-procedurally, the patients were observed for bradycardia, hypotension and desaturation (SpO_2 <95%[2] except in tetralogy of Fallot (TOF) where fall below baseline value or occurrence of cyanotic spells was monitored), need for airway-manipulation/adjunct (jaw-thrust, oropharyngeal or supraglottic airway device, endotracheal intubation), shivering, postoperative nausea and vomiting (PONV). Bradycardia and hypotension were defined and managed according to the Paediatric Advanced Life Support (PALS) guidelines, though none of the patients required any intervention.[2] An episode of desaturation (lowest value 92%) was reported in one patient with PSSS <2 which was managed by jaw-thrust and down-titrating

Table 2: The quality of sedation, scan and postprocedural recovery profile

Quality of sedation	Quality of scan	Time to recovery (minutes)	Time to discharge or shifting to ward (minutes)	PONV	Shivering
Optimal	Excellent	10	30	-	-
Optimal	Excellent	15	35	-	-
Optimal	Excellent	20	45	-	-
Optimal	Excellent	25	50	-	-
Optimal	Poor	25	55	-	-
Optimal	Excellent	10	35	-	-
Optimal	Excellent	15	35	-	-
Suboptimal	Good	20	50	-	-
Optimal	Good	15	35	-	-
Optimal	Excellent	20	45	-	-

PONV: Postoperative nausea and vomiting; (-):Negative

the drug infusion [Table 1]. The MRI had to be interrupted in this patient due to desaturation, and classifying it as a poor scan quality rating [Table 2]. None manifested with PONV or shivering [Table 2].

DISCUSSION

MRI is a day-care procedure which requires inculcating the principles of fast-track anaesthesia for successful patient management. The most ideal anaesthetic drug for MRI sedation is still an area of research.[4] Despite options like propofol, ketamine and dexmedetomidine, none classifies as an ideal anaesthetic agent, with majority of the attributes such as a quick-onset, ease of titration, haemodynamic stability, maintenance of spontaneous respiration alongside the provision of immobility and short duration of action with minimal side-effects.[2,3,6-8]

With the ongoing search for an ideal agent for MRI sedation, the advent of ketodex has gathered significant attention.[3,8] Luscri *et al*.[3] successfully used ketodex for MRI sedation in three patients of trisomy-21 suffering from obstructive sleep apnoea. However, the present case-series extends the application of ketodex sedation for MRI to syndromic children with underlying cardiac anomalies.

The safe use of ketodex can be heralded by the peculiar characteristics of the two anaesthetic drugs. On one end, dexmedetomidine acts on postsynaptic alpha-2 adrenoceptors and inhibits the sympathetic activity causing hypotension and bradycardia. On the other end, ketamine has cardio-stimulatory effects such as tachycardia and hypertension and maintains the systemic vascular resistance (SVR).[6,8] Therefore, the additive effect of the drugs helps to decrease the minimum effective dose, better haemodynamics and minimal side-effects. In this context, a recent meta-analysis by Kim *et al*.[4] outlined a delayed onset of MRI sedation with dexmedetomidine as a sole agent, which can be overcome by the combination with faster acting agents like ketamine.

Notably, dexmedetomidine has been found to have a role in preventing and managing tet spells in TOF patients.[9] While it may appear intuitive that dexmedetomidine would have prevented tet spells in our TOF patients, the importance of a productive liaison of the drug combination can simultaneously not be undermined. Alongside the avoidance of a decrease in the SVR with ketodex, the lack of any significant escalation in pulmonary vascular resistance (PVR) attributable to the drug combination, can be a particularly useful pharmacological attribute.[6,8,9] Moreover, both the drugs are associated with minimal respiratory depression, with mechanistically distinct additive analgesic effects, further beneficial in attenuating any untoward PVR elevation.[3,6,8]

On comparing the literature on the recovery time following MRI sedation, it appears to be compounded by factors such as type of drug, dosage and heterogeneity in the definitions of time to recovery.[2,4,10] Reduced recovery time was observed in the present case-series unlike Abdellatif *et al*.[8] who used ketamine 1.5 mg/kg + dexmedetomidine 1 μg/kg/hour for 10 min followed by 0.6-1 μg/kg/hour. This could be explained by the lower dexmedetomidine dose employed in our high-risk subset. However, the time to discharge in the present case-series was longer compared to that observed by Tammam *et al*.[6] who used intramuscular ketodex (ketamine 2 mg/kg + dexmedetomidine 1.5 μg/kg). This can possibly be attributed to the different route and dose of ketodex administered.

In addition, certain properties of dexmedetomidine can significantly improve the recovery profile.

While ketamine is emetogenic and can produce emergence delirium, dexmedetomidine is found to decrease the incidence of PONV and emergence delirium.[11] Dexmedetomidine also has a role in the alleviation of perioperative shivering which can be detrimental in cardiac patients.[12] Nevertheless, no patient in the present-case series had PONV or shivering.

CONCLUSION

Ketodex appears to be a safe agent for MRI sedation in syndromic patients with congenital cardiac anomalies backed by meticulous dose titration, individualisation appropriate to the clinical context and vigilant monitoring.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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