USING LOW DOSES OF DEXMEDETOMIDINE FOR PROCEDURAL SEDATION DURING MRI DIAGNOSTICS IN CHILDREN – CASE REPORT

PRIMENA NISKIH DOZA DEKSMEDETOMIDINA ZA SPROVOĐENJE PROCEDURALNE SEDACIJE TOKOM MRI DIJAGNOSTIKE KOD DECE – PRIKAZ SLUČAJA

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Case report
Prikaz slučaja
UDK 615.211-053.2:537.635
https://doi.org/10.2298/MPNS2412348T

Abstract

Introduction. Dexmedetomidine is a potent and highly selective α_2-adrenergic agonist. Its sedative effects are mediated through the inhibition of neuronal activity in the locus coeruleus, which is rich in α_{2A}-adrenergic receptors. In pediatric patients, sedation or general anesthesia is often required for certain diagnostic procedures, such as magnetic resonance imaging, due to limited cooperation in children under six years of age and the intense noise during scanning, ensuring patient tolerance and procedural success. **Case Report.** We present the case of a five-year-old boy referred for magnetic resonance imaging due to headaches and dizziness. Premedication was administered intramuscularly 30 minutes before the procedure, consisting of atropine (0.0125 mg/kg) and midazolam (0.1 mg/kg). Simultaneously, an initial intravenous bolus of dexmedetomidine (2 μg/kg) was infused over 15 minutes. The child was then transferred to the magnetic resonance imaging room, where sedation was maintained with a continuous intravenous infusion of dexmedetomidine at 0.25 μg/kg/h via an infusion pump, without additional anesthetic agents or bolus doses. No adverse effects, including bradycardia, respiratory disturbances, or desaturation were observed. **Conclusion.** Despite the use of a subminimal continuous dexmedetomidine dose compared to recommended dosing regimens, an adequate depth of sedation was achieved, allowing for high-quality magnetic resonance imaging without complications.

Key words: Dexmedetomidine; Hypnotics and Sedatives; Magnetic Resonance Imaging; Diagnostic Imaging; Child

Sažetak

Uvod. Deksmedetomidin je potentan i visokoselektivan α_2 adrenergički agonista. Sedativne efekte ispoljava inhibicijom aktivnosti neurona *locus coeruleus*, u kome se nalazi veliki broj adrenergičkih α_{2A} receptora. Za potrebe pojedinih dijagnostičkih procedura, poput magnetne rezonancije, u pedijatrijskoj populaciji neophodno je pregled sprovesti u sedaciji/opštoj anesteziji, s obzirom na ograničenu saradnju dece mlađe od šest godina, te prisutnu intenzivnu buku tokom snimanja, kako bi dete tolerisalo pregled. **Prikaz slučaja.** Dat je prikaz slučaja dečaka starosti pet godina, koji je na magnetnu rezonanciju upućen pod dijagnozom glavobolje i vrtoglavice. Detetu je ordinirana intramuskularna premedikacija: atropin u dozi od 0,0125 mg/kg i midazolam u dozi od 0,1 mg/kg 30 minuta pre početka sprovođenja pregleda, uz istovremenu primenu i 15-minutne inicijalne bolus doze deksmedetomidina intravenski od 2 μg/kg. Neposredno pre započinjanja pregleda, dečak je premešten u prostoriju za pregled magnetnom rezonancijom. Bez dodatne primene nekog od anestetika, kao i bez dodatnih bolus doza deksmedetomidina, započeta je primena deksmedetomidina kontinuirano intravenski u dozi od 0,25 μg/kg/h preko infuzione pumpe. Nisu zabeležena neželjena dejstva kao bradikardija, poremećaji disanja i desaturacija. **Zaključak.** Iako je primenjena subminimalna kontinuirana doza deksmedetomidina u odnosu na preporučene režime doziranja, postignuta je odgovarajuća dubina sedacije i adekvatan kvalitet slika magnetne rezonancije.

Ključne reči: deksmedetomidin; hipnotici i sedativi; magnetna rezonanca; dijagnostički imidžing; dete

Introduction

Dexmedetomidine is a potent and highly selective α_2-adrenergic agonist [1], exerting dose-dependent sympatholysis, anxiolysis, sedation, hypnosis, and mild analgesia with minimal impact on respiratory function [2, 3]. Its sedative effects result from neuronal inhibition in the locus coeruleus, which is rich in α_{2A}-adrenergic receptors. Dexmedetomidine also affects the cardiovascular system, with lower doses inducing bradycardia and hypotension, while higher doses may cause more pronounced bradycardia and hypertension. Due to these properties, α_2-adrenergic agonists are widely used for sedation in both diagnostic procedures and intensive care units [4]. In pediatric patients, sedation or general anesthesia is often required for magnetic resonance imaging (MRI) due to limited cooperation, particularly in children

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Abbreviations

MRI – magnetic resonance imaging
CT – computed tomography
MAP – medial arterial pressure

under six years of age, and the intense noise generated during scanning. Adequate sedation ensures patient tolerate while maintaining high-quality imaging [5]. A recent meta-analysis reported an average dexmedetomidine induction dose of 2.8 ± 0.5 μg/kg (administered over 10 minutes) and the maintenance dose of 1.8 ± 0.4 μg/kg/h [6]. Here, we present a case from our clinical practice in which an MRI examination was successfully performed using dexmedetomidine at lower-than-standard doses.

Case Report

A five-year old boy weighing 16 kg was referred for MRI due to headaches and dizziness. His mother reported a one-month history of headaches and four episodes of balance loss. A computed tomography (CT) scan performed in September 2023 suggests the presence of an anterior parasagittal right subarachnoid cyst, measuring 6x2x3 cm and chronic pansinusitis. The child was fully vaccinated for his age, with no history of chronic illnesses, allergies, or previous hospitalizations. On examination, congestion of the left nostril and slight pharyngeal hyperemia were noted. An otorhinolaryngology assessment revealed dried nasal secretions, mild pharyngeal hyperemia, and poor tympanic membrane transparency. Romberg's and Unterberger's tests were negative, and laboratory results were within normal limits.

Per institutional protocol, the mother was instructed to withhold oral intake from midnight, and intravenous Hartmann's solution (50 ml/h) was initiated to optimize cardiovascular volume.

On the day of the MRI, the child was alert and cooperative in the premedication room. He received intramuscular atropine (0.2 mg; 0.0125 mg/kg) and midazolam (1.5 mg; 0.1 mg/kg) 30 minutes before the procedure. Simultaneously, a 15-minute intravenous initial bolus of dexmedetomidine (2 μg/kg, total 32 μg) was administered, inducing sleep within minutes. Just before the MRI, the child was transported with minimal stimulation to the scanning room, remaining asleep throughout. Without additional anesthetic agents or bolus doses, a continuous intravenously infusion of dexmedetomidine (0.25 μg/kg/h) was initiated via an infusion pump. Headphones were placed over his ears to reduce noise exposure and prevent potential hearing damage. Hartmann's solution was infused at 130 ml/h, and the child breathed spontaneously with 100% oxygen (4 l/min) via a face mask. A pulse oximeter was used for monitoring, with blood pressure measurement omitted to minimize stimulation. The child's initial heart rate was 124 bpm, with transcutaneous oxygen saturation at 100%. No adverse effects, such as bradycardia, respiratory disturbances, or desaturation, were observed. Throughout the 45-minute MRI examination, the heart rate remained stable, with the final recorded value at 109 bpm, and saturation maintained at 100%. The total administered dexmedetomidine dose was 2.6 μg, with 100 ml of Hartmann's solution. The child remained adequately sedated, with no unwanted movements or awakening during the procedure. Upon completion, he woke up within minutes without complications.

Discussion

Procedural sedation is increasingly utilized in the pediatric population [7]. Due to ongoing synaptogenesis and central nervous system development, children are particularly sensitive to anesthetics and sedatives, necessitating careful dose selection [8].The ideal sedative should have a rapid and predictable onset, a short half-life, dose-dependent effects, rapid action cessation, spontaneous breathing preservation, hemodynamic stability, painless administration, and a reversible action [9]. Successful MRI sedation depends on two key factors: procedural safety (low incidence of adverse events) and sedation effectiveness (successful completion of MRI) [10].

Dexmedetomidine is well-suited for MRI sedation, either alone or in combination with other anesthetics such as midazolam and propofol. Heard et al. [11] compared dexmedetomidine monotherapy (1 μg/kg bolus followed by 0.5 μg/kg/h infusion) with a combination of dexmedetomidine and midazolam (0.1 mg/kg). The dexmedetomidine-only group exhibited a shorter recovery time. Similarly, Koroglu et al. [12] compared dexmedetomidine (1 μg/kg bolus, 0.5 μg/kg/h infusion) to propofol (3 mg/kg bolus, 100 μg/kg/min infusion). Although propofol led to more frequent hypotension and desaturation, it also provided faster onset and recovery. Propofol, was associated with higher heart rate values but lower medial arterial pressure (MAP) and respiratory rates compared to dexmedetomidine, though these differences lacked clinical significance. Dexmedetomidine is characterized by prolonged recovery time [13]. Ahmed et al. [14] administered an initial 2 μg/kg dexmedetomidine bolus over 10 minutes, followed by 1 μg/kg/h dexmedetomidine infusion. If sedation was inadequate, an additional 2 μg/kg bolus was given. Bradycardia was

observed in 4.5% of cases, primarily in children aged 1– 3 years.

Although some studies suggest that lower dexmedetomidine doses (0.1-0.7 μg/kg/h) can achieve sedation, higher doses are generally recommended for MRI procedures to ensure procedural success [15, 16]. Recent findings support that the use of higher dexmedetomidine doses for MRI sedation, demonstrating good tolerability [17]. A retrospective study [18] administered a dexmedetomidine in an initial bolus dose of 2 μg/kg over 10 minutes, followed by a continuous 2 μg/kg/h infusion. If sedation was inadequate, an additional 2 μg/kg bolus was given, with midazolam or fentanyl added if necessary. This approach achieved successful sedation in 99.8% of cases - 78.5% with dexmedetomidine alone and 21.5% with adjunctive medications. No significant respiratory depression was observed, although apnea occurred in 0.1% of cases. A revised protocol by Ahmed et al. [14], increased the initial 10-minute loading dose of dexmedetomidine from 2 to 3 μg/kg and the continuous infusion from 1 to 1.5-2 μg/kg/h. raising the sedation from 91.8% to 97.6%. The Boston Sedation Protocol recommends a 3 μg/kg dexmedetomidine bolus over 10 minutes, followed by a 2 μg/kg/h infusion [19]. Liaudanskytė et al. [20] reported a 94.3% sedation success rate using a protocol with intravenous midazolam (0.15 mg/kg), a 3 μg/kg dexmedetomidine bolus over 10 minutes, and a continuous 2 μg/kg dexmedetomidine infusion. In the remaining 5.7% of cases, additional boluses of dexmedetomidine, midazolam, propofol or fentanyl were required.

In our experience, adequate MRI sedation is typically achieved with a 1.5-2 μg/kg dexmedetomidine bolus over 15 minutes, with or without intramuscular atropine and midazolam, followed by a 1.5-2 μg/kg/h infusion. When deeper sedation is required, additional dexmedetomidine and propofol boluses are preferred. This dosage regimen aligns with most published protocols. However, to our knowledge, the present case report is the first to document successful MRI sedation with a significantly lower continuous infusion of dexmedetomidine. The sedation was achieved with an initial 2 μg/kg bolus of dexmedetomidine, supplemented with intramuscular atropine and midazolam, followed by continuous administration of 0.25 μg/kg/h of dexmedetomidine.

Conclusion

This case demonstrated that magnetic resonance imaging sedation can be effectively achieved with subminimal dexmedetomidine infusion (0.25 μg/kg/h), without adverse effects and while maintaining image quality. Potential factors contributing to this successful outcome include additional applied premedication, patient-specific physiological characteristics, and an individual response to the medication.

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Rad je primljen 7. XII 2024.
Recenziran 14. I 2025.
Prihvaćen za štampu 14. I 2025.
BIBLID.0025-8105:(2024):LXXVII:9-12:348-351.