Article published online: 2024-12-04

Intranasal Dexmedetomidine for Sedation in Acute Ischemic Stroke: A Case Report

Antonio Romanelli[1] Antonella Langone[1] Flora Minichino[1] Renato Gammaldi[1]

[1] Department of Anaesthesia and Intensive Care, A.O.U. "San Giovanni di Dio e Ruggi d'Aragona", Salerno, Italy

Address for correspondence Antonio Romanelli, MD, Department of Anaesthesia and Intensive Care, A.O.U. "San Giovanni di Dio e Ruggi d'Aragona", Salerno, Italy (e-mail: antonioromanelli86@gmail.com).

J Neuroanaesthesiol Crit Care

Abstract

This case report presents the innovative use of intranasal dexmedetomidine (IN-DEX) for sedation in an 82-year-old female patient undergoing endovascular treatment (EVT) for acute ischemic stroke (AIS). The patient, with a history of chronic atrial fibrillation, arterial hypertension, and chronic kidney failure, presented with sudden left-sided weakness. IN-DEX was administered to manage her agitation, achieving stable sedation without respiratory depression, facilitating EVT, and preserving the patient's cooperative state for accurate neurological assessments. The procedure was completed successfully, with stable vital parameters and no complications. This report highlights IN-DEX's potential as a noninvasive, effective sedative alternative in emergency neurological settings, emphasizing its advantages in maintaining respiratory function and reducing stress responses in AIS patients. Further studies are recommended to validate these findings and explore the broader applicability of IN-DEX in AIS management.

Keywords

- dexmedetomidine
- intranasal administration
- acute ischemic stroke
- endovascular treatment
- sedation

Introduction

The management of acute ischemic stroke (AIS) encompasses a multidisciplinary approach involving neuroprotective strategies, reperfusion therapies, and the meticulous control of physiological parameters to ameliorate outcomes. Sedation is pivotal in managing agitation, reducing metabolic demand, and facilitating necessary diagnostic and therapeutic endovascular treatment (EVT).

Due to its unique pharmacological profile, dexmedetomidine (DEX), a highly selective α_2-adrenergic agonist, inhibits the release of norepinephrine and reduces the electrical activity of the brain by acting on the α_2 adrenergic receptor of the nucleus coeruleus.[1] Unlike traditional sedatives, endovenous DEX provides sedation without respiratory depression, preserves a state of easy arousability, and exerts anxiolytic and analgesic effects. Moreover, DEX facilitates cooperative sedation, which is particularly advantageous in neurological assessments and neuroimaging procedures where patient cooperation is indispensable.[2] Furthermore, this drug can significantly reduce the amount of sedatives and opioids, reducing the risk of conversion to general anesthesia, and alleviating hemodynamic response to periprocedural stress.[3]

Intranasal DEX (IN-DEX) is an attractive alternative to endovenous administration. It offers a noninvasive, rapid, and reliable method of drug delivery, bypassing the first-pass metabolism and ensuring higher bioavailability. IN-DEX has been previously documented for procedural sedation in pediatric populations and premedication purposes.[4]

Due to its favorable pharmacologic profile and easy administration, IN-DEX can be considered an interesting option in patients with AIS undergoing EVT. However, its use in this setting has yet to be explored. Here, we reported the case report of a patient sedated with IN-DEX requiring EVT for AIS treatment.

DOI https://doi.org/10.1055/s-0044-1791687.
ISSN 2348-0548.

Thieme Medical and Scientific Publishers Pvt. Ltd., A-12, 2nd Floor, Sector 2, Noida-201301 UP, India

Case Report

An 82-year-old female patient (weight 55 kg) presented at the emergency department with sudden weakness in the left arm and leg (National Institutes of Health Stroke Scale 4 points). Her past medical history includes chronic atrial fibrillation, arterial hypertension, chronic kidney failure (estimated glomerular filtration rate 34 mL/min), gastroesophageal reflux, hyperthyroidism, and colon diverticulosis. Her home therapy consisted of edoxaban, tapazole, metoprolol, atorvastatin, and furosemide. Her vital parameters were: heart rate (HR) 55 beats per minute (bpm), noninvasive blood pressure (NIBP) 170/50 mm Hg, and SpO_2 99% in room air. A brain computed tomography (CT) scan highlighted blood stacking from proximal M2 stasis in the right middle cerebral artery with a small hypodensity area in the parietal region, suggesting AIS.

After the CT scan, the patient was anxious, with movements not aggressively vigorous (Richmond Agitation Sedation Scale [RASS] +1). In the preprocedure room, the attending anesthesiologist administered IN-DEX (1 mcg/kg) plus ephedrine (1 mg) for a total volume of 2 mL (1 mL per nostril) with a nasal nebulizer (Aluneb MAD Nasal, ►**Fig. 1**). The administration time was just a few seconds, and vital parameters were strictly monitored.

After 17 minutes, mechanical thrombectomy treatment began. When the patient was positioned on the operating table, RASS was 0, and her vital parameters were stable (HR 50 bpm, NIBP 163/48 mm Hg, and SpO_2 100% with O_2 2.0 L/min). Endovenous midazolam (2 mg) was administered because she reported pain when inflating the blood pressure cuff.

Selective catheterization of the right internal carotid artery was performed by right femoral artery access. Angiography confirmed the occlusion of the M2 segment. Using a stent retriever with an aspiration catheter, complete recanalization was achieved. Before aspiration, 1 mg of midazolam was administered to avoid the patient's head movement.

Fig. 1 The figure shows the Aluneb MAD Nasal, a drug nebulizer for intranasal mucosa. The cap completely seals the nostril for rapid and painless administration of drugs.

The procedure lasted approximately 40 minutes. The patient's vital parameters were stable during the procedure, with RASS ranging from –1 to –2, without the occurrence of hypotension, severe bradycardia, breath depression, or agitation. The anesthesiologist administered flumazenil (1 mg), reaching a RASS 0, and the postprocedural neurological examination showed no modifications compared to the preprocedure status.

The patient was transferred to the stroke unit in stable conditions (HR 65 bpm, NIBP 153/75 mm Hg, and SpO_2 98% in room air). After 6 hours postprocedure, the patient showed RASS 0, with stable parameters (HR 52 bpm, NIBP 147/61 mm Hg, and SpO_2 96% in room air) and fully recovered neurological status.

Discussion

This case report provides an interesting perspective on the use of IN-DEX as a noninvasive, quick, and reliable option for sedation in emergency neurological situations. It could be a useful addition to the management of AIS patients.

IN-DEX demonstrated a promising alternative to traditional route administration for sedative agents, offering the advantage of providing sedation without respiratory depression, a critical consideration in AIS patients who may already have compromised respiratory function. DEX's anxiolytic and analgesic properties contribute to patient comfort, potentially reducing the stress response, which could exacerbate ischemic injury.[5] IN-DEX does not require an infusion pump, allowing its administration in a nonoperating room setting and gaining time to reach maximum effect when starting the procedure. The cooperative sedation facilitated by DEX allowed for accurate neurological assessments and smooth execution of necessary neuroimaging procedures, emphasizing the drug's utility in the nuanced management of AIS.

In the pediatric population, IN-DEX (2.0 mcg/kg) has a relatively predictable onset time, approximately 25 to 30 minutes after administration, achieving mild to moderate sedation lasting up to 2 hours. [6] Although the exact mechanisms underlying intranasal drug delivery to the central nervous system (CNS) are not entirely understood, drugs can rapidly access the CNS following olfactory nerve pathways leading from the nasal cavity directly to the CNS.[7] In our case, we also administered a vasoconstrictor. Vasoconstrictors, reducing the vascular supply to the mucosa, prevent the drug from dispersing into the systemic circulation, thus reaching higher local concentrations and avoiding systemic effects. [8] Although bradycardia and hypotension may be a concern, meta-analyses showed a relatively low occurrence (3.0%) of bradycardia in the pediatric population, typically manageable and without severe clinical consequences. [9,10]

To the best of our knowledge, this is the first report of IN-DEX administration for the management of AIS patients. Further research, through randomized controlled trials and larger observational studies, is needed to establish the dose, the sedative effect-sparing, efficacy, and safety of IN-DEX in patients with AIS and requiring EVT.

Conflict of Interest
None declared.

References

1 Patel A, Davidson M, Tran MC, et al. Dexmedetomidine infusion for analgesia and prevention of emergence agitation in children with obstructive sleep apnea syndrome undergoing tonsillectomy and adenoidectomy. Anesth Analg 2010;111 (04):1004–1010
2 Bao N, Tang B. Organ-protective effects and the underlying mechanism of dexmedetomidine. Mediators Inflamm 2020; 2020:6136105
3 Nii K, Hanada H, Hiraoka F, Eto A, Mitsutake T, Tsutsumi M. Usefulness of consciousness sedation with dexmedetomidine and pentazocine during endovascular treatment for acute stroke. Neurol Med Chir (Tokyo) 2018;58(02):79–84
4 Zhang G, Xin L, Yin Q. Intranasal dexmedetomidine vs. oral midazolam for premedication in children: a systematic review and meta-analysis. Front Pediatr 2023;11:1264081
5 Terceño M, Silva Y, Bashir S, et al; Catalan Stroke Code and Reperfusion Consortium (Cat-SCR) Impact of general anesthesia on posterior circulation large vessel occlusions after endovascular thrombectomy. Int J Stroke 2021;16(07):792–797
6 Li BL, Guan YP, Yuen VM, et al. Population pharmacokinetics of intranasal dexmedetomidine in infants and young children. Anesthesiology 2022;137(02):163–175
7 Crowe TP, Hsu WH. Evaluation of recent intranasal drug delivery systems to the central nervous system. Pharmaceutics 2022;14 (03):629
8 Dhuria SV, Hanson LR, Frey WH II. Intranasal delivery to the central nervous system: mechanisms and experimental considerations. J Pharm Sci 2010;99(04):1654–1673
9 Gong M, Man Y, Fu Q. Incidence of bradycardia in pediatric patients receiving dexmedetomidine anesthesia: a meta-analysis. Int J Clin Pharm 2017;39(01):139–147
10 Marra P, Di Stadio A, Colacurcio V, et al. Sedation with intranasal dexmedetomidine in the pediatric population for auditory brainstem response testing: review of the existing literature. Healthcare (Basel) 2022;10(02):287

