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Case Report Open Access

Prolonged Use of Dexmedetomidine Infusion in an Infant for Sedation as Adjuvant Therapy

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Abstract

Dexmedetomidine, a central and peripheral alpha-2 receptor agonist approved by the FDA for the use in patients 18 years old and older, it has been increasingly used in the pediatric population for sedation and analgesia due to its advantage of minimal respiratory depression and lack of abuse/dependence. This is a case report of a successful use of dexmedetomidine in an infant for 15 weeks achieving; symptom control, reduction in the dose of opioids and benzodiazepines by 50% and a safe transition to oral equivalents.

Keywords: Dexmedetomidine; Pediatrics; Dexmedetomidine prolonged use; Opioid adjuvant

Introduction

Dexmedetomidine is a central and peripheral alpha-2 receptor agonist; its analgesic therapeutic effects are mediated centrally. The alpha-2 receptor is located on the vascular pre-synaptic neuron terminals where it inhibits the release of norepinephrine via negative feedback [1].

The medication is approved by the FDA for 24 hours or less for sedation in patients 18 y/o and older [2] in intubated and non-intubated patients in the Intensive Care setting and/or during surgical procedures. Another quality is its analgesic properties, although it may not be sufficient to use it alone, it has been used more and more frequently as an adjunctive agent along opioids.

It has been increasingly used off-label in the pediatric population for sedation and analgesia due to its advantage in safety, minimal to none respiratory depression and lack of dependence/abuse potential compared to other more traditional options (opioids/benzodiazepines) [3-5].

We report the efficacy and safety of the use of Dexmedetomidine infusion for a prolonged duration in an infant beyond current manufacturer and FDA recommendations.

Case Presentation

The patient was a male infant born at 34 weeks of gestational age due to preterm labor with the following diagnosis at birth: Trisomy 21, Esophageal stenosis, Duodenal atresia, Annular pancreas, complete balanced Atrio-Ventricular septal defect. With the following major complications during the hospitalization; Mixed apneas, tracheostomy placement, mechanical ventilation dependence, secondary pulmonary hypertension, venous and arterial clotting episodes leading to Left-above the knee amputation, multifocal brain infarcts and seizures (Figure 1).

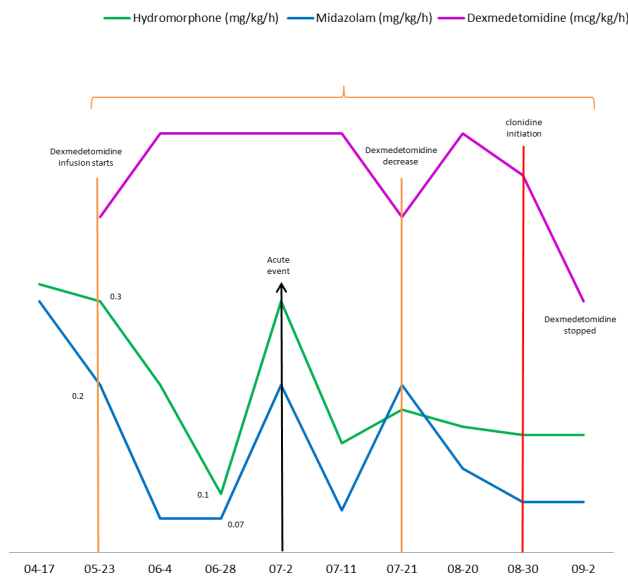

Figure 1: 50% reduction of opioid and benzodiazepine doses overtime.

He required for sedation and analgesia the continuous infusion of; a benzodiazepine (Midazolam – maximum used dose 0.4 mg/kg/h) and an opioid together (Hydromorphone –maximum used dose: 0.7 mg/kg/h). After 11 weeks of unsuccessful pain control/unacceptable comfort level and inability to wean benzodiazepines and opioids dexmedetomidine was added to his regimen. The clinical goals were to decrease pain, improve comfort level and to wean opioids and benzodiazepines. The clinical parameters we followed were; Heart rate (HR), blood pressure (BP) and pain scale (FLACC score) as subrogate for sedation/comfort. The patient was ventilator-dependent therefore

the effects of dexmedetomidine on the respiratory rate could not be evaluated (Figure 2).

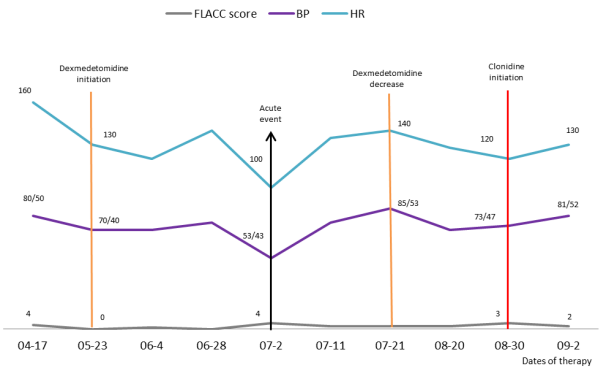

Figure 2: FLACC score - Blood Pressure (BP) - Heart rate (HR) correlation with dose of medications on Figure 1.

Results

In 5 weeks, while on Dexmedetomidine the doses of benzodiazepine and opioid were decreased by at least 50% achieving the same or better degree of pain control/comfort. The doses decreased from 0.2 to 0.1 mg/kg/h for midazolam and from 0.3 to 0.07 mg/kg/h for hydromorphone.

In the 15 weeks of infusion of Dexmedetomidine, the patient did not show side-effects that required medical intervention (Bradycardia, Hypotension) he had one event of bradycardia/hypotension due to sepsis, but not related to the medication proved by the fact he recover his baseline parameters when the infection was controlled without changes in the dose of dexmedetomidine.

The patient was successfully transitioned to another alpha-2 agonist; oral clonidine over a 5-day transition without rebound of hypertension, tachycardia or an increase of the dose of opioids or benzodiazepines.

Discusion

Dexmedetomidine was safely used for longer than 24 hours (15 weeks, in this case) in a pediatric patient without major side-effects showing a potential expansion in the time and age limit of use of Dexmedetomidine. Another potential use of Dexmedetomidine is as an opioid and benzodiazepine adjuvant, allowing reduction to the dose of these medications, decreasing the potential side-effects and dependence on them, along with speeding up the weaning of these two potentially dangerous medications without creating dependence on its substitute. Besides the lack of dependence, the easy transition from an intravenous to an oral medication with similar properties (Clonidine, another alpha-2 agonist) makes the decision to introduce and use dexmedetomidine as an opioid/benzodiazepine adjuvant even more tempting in the ICU setting when aiming for faster transition off of continuous infusions and decreasing the hospital stay in ICU.

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