Paradoxical Hypertension Following Administration Of Dexmedetomidine During Embolization Of A Congenital Arteriovenous Malformation In A 15-Month-Old Male

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Citation

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Abstract

We present a 15-month-old boy with a past medical history significant for a left-sided thalamic arteriovenous malformation, status post multiple staged embolizations. During a repeat embolization under general anesthesia, dexmedetomidine was given to deepen the anesthesia during periods of increased stimulation. On three separate occasions, the patient developed paradoxical hypertension consistent with an acute alpha-1 and alpha-2b agonist response, which subsided with immediate discontinuation of dexmedetomidine and intervention with propofol and fentanyl. Care should be taken while using dexmedetomidine in children.

CASE REPORT

This is a 15-month-old boy, dichorionic diamniotic twin gestation, who had been born via repeat cesarean section at 26 weeks 5 days, birth weight 1151 grams, for preterm labor, with a maternal history of methamphetamine use in early pregnancy. He presented in infancy with multiple generalized tonic clonic seizures. Brain MRI revealed a large left thalamic arteriovenous malformation, so he was scheduled for staged endovascular embolization over three sessions. At our encounter, he remained symptomatic and the target of the current treatment was the anterior choroidal artery.

The patient was brought to the interventional radiology suite and positioned supine on the fluoroscopy table. After standard monitoring was placed, he underwent uneventful induction with sevoflurane, fentanyl, propofol, and vecuronium. The patient was intubated with a 4 mm endotracheal tube, and a left radial arterial line was established. He was maintained on sevoflurane and propofol 100 mcg/kg/min and remained hemodynamically stable with heart rate 80-110 bpm and blood pressure 90s/50s. With neuromonitoring, the neurosurgery team proceeded with diagnostic cerebral angiogram and roadmap creation.

Prior to embolization of the lesion with Onyx 18, the surgical team requested increasing the patient's anesthetic depth for anticipated stimulation. We elected to use a bolus of dexmedetomidine 1 mcg/kg administered as an IV push. The blood pressure suddenly increased dramatically to 120/80 and the heart rate increased to 105 bpm. They returned to baseline within 10 minutes with administration of fentanyl 30 mcg and propofol 20 mg, and remained stable at baseline without further intervention. At two other points 90 minutes after this initial episode, the surgeons again requested deeper anesthetic, so dexmedetomidine was administered at lower doses of 0.7 mcg/kg and 0.5 mcg/kg (Figure 1). Despite these lower doses, the heart rate and blood pressure again increased dramatically and resolved with discontinuation of dexmedetomidine and administration of fentanyl and propofol boluses. The surgery team successfully embolized the lesion and there were no adverse neuromonitoring events during the procedure. The patient remained intubated and was transported to the pediatric ICU post-operatively.

DISCUSSION

Dexmedetomidine is an important non-opiate sedative analgesic. Currently it is only US FDA approved for sedation for < 24 hours in adults under mechanical

ventilation and for sedation of non-intubated patients prior to and/or during surgical and other procedures. However, it is commonly used for pediatric sedation for imaging studies, long-term ventilator management in the ICU, and painful procedures such as burn dressing changes. It is popular in multimodal analgesia for its sedative and anxiolytic properties and minimal respiratory depression.7 Unlike narcotics, it does not act at the opioid receptors and does not cause constipation and pruritus. Centrally it agonizes alpha-2 adrenoreceptors at the local ceruleus in the pons to cause sedative and anxiolytic effects. More peripherally it agonizes alpha-2 receptors in the dorsal horn of the spinal cord and inhibits substance P release for anti-nociceptive and opioid synergistic effects. Dexmedetomidine has a higher specificity ratio for alpha-2 compared to alpha-1 of 1620:1.5

Dexmedetomidine is most known for causing refractory hypotension and bradycardia. It typically occurs with loading doses or high infusion rates.7 The hypertension is theorized to be due to an initial, direct stimulation of alpha-1 or alpha-2b receptors peripherally, resulting in vasoconstriction. This phenomenon resolves with subsequent central alpha-2a receptor stimulation, which exerts a stronger effect causing hypotension and bradycardia. This is known as a biphasic blood pressure response. With infusions, time to effect ranges from 10-15 minutes on average, so dexmedetomidine boluses are frequently given to speed onset of sedation. Generally it is advised that dexmedetomidine boluses be loaded over 10 minutes to prevent profound hypotension and bradycardia that can be refractory to fluid resuscitation and vasopressor support. This is especially dangerous in children where cardiac output is largely heart rate dependent.

Until now, there have only been two published pediatric case reports of dexmedetomidine-induced hypertension. One was a 32-month-old boy with acute transverse myelitis who received a dexmedetomidine loading dose and infusion as sedation for MRI.6 The other was an 18-year-old boy with acute traumatic brain injury and intermittent spikes in intracranial pressure who was being maintained on dexmedetomidine infusion for mechanical ventilation in the ICU.1 The reported incidence of pediatric hypertension with dexmedetomidine varies from 0 to 8%.7 Some studies are confounded by concurrent anxiety and crying by the child.4 In others, blood pressure data is limited to every 5 minutes to avoid interference with MRI signals. From one retrospective review of dexmedetomidine for sedation for radiologic studies, risk factors include > 2 boluses of 3 mcg/kg and younger age < 1 yo.2,3 Another large cohort study looking specifically at the incidence of hypertension with high-dose dexmedetomidine for MRIs noted a 4.9% incidence, with risk factors including age < 1 year old, high dose infusion > 2 mcg/kg/hr, and/or receiving multiple initial boluses. No study has clearly reported an association between the dose and frequency or severity of hypertension.

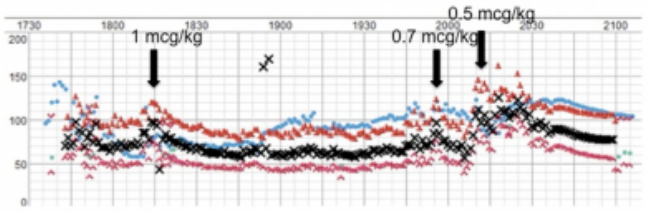
Our case report describes this phenomenon recurring three times with each administration of precedex even at successively lower doses under general anesthesia. The boluses were administered as IV pushes and there was clear hypertension delineated by arterial blood pressure waveform monitoring throughout the case. We present this case to caution practitioners about this uncommon side effect. Consequences of uncontrolled intraoperative hypertension include cerebrovascular events such as intracerebral hemorrhage, stroke, and elevated intracranial pressure; bleeding into the surgical field that limits surgeons' view; myocardial infarction; pulmonary edema.

LEARNING POINTS

1. Paradoxical hypertension is a rare event that can occur with bolus administration of dexmedetomidine.
2. Initial hypertension is likely due to alpha-1 and alpha-2b agonist activity of dexmedetomidine.
3. Dexmedetomidine boluses should be administered slowly and with caution.

Figure 1

Vital signs graph depicting a sudden increase in blood pressure and heart rate with successively reduced boluses of dexmedetomidine



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