Cardiac Arrest in a Child Undergoing a Sevoflurane Induction After Receiving Clonidine Preoperatively

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Introduction: Clonidine is an α-2 adrenergic agonist that is used for treatment of childhood hypertension, Tourette syndrome, ADHD and restlessness. Common side effects are sedation and xerostomia. An overdose may cause bradycardia, hypotension and unconsciousness. This report describes a case in which a child receiving oral clonidine sustained a cardiac arrest during anesthesia induction with oxygen (O2), nitrous oxide (N2O) and sevoflurane.

Case Presentation: A 5-year old, 16.8 kg girl was scheduled for baclofen pump placement. Her history was significant for cerebral palsy, severe developmental delay, static encephalopathy, seizures, severe spasticity, periventricular leukomalacia, and G-tube dependency. She had successfully undergone a general anesthetic with O2/N2O/sevoflurane for a lumbar puncture baclofen test dose administration the previous day. She underwent a slow mask induction of anesthesia with O2/N2O/Sevoflurane 2% to 8%. Mask ventilation was easy, and SaO2 was 100%. The patient's heart rate suddenly dropped from 100 beats/min (BPM) to sinus bradycardia in the 20s. Blood pressure (BP) was undetectable. A 22G intravenous catheter was immediately placed in the patient's right arm. N2O and sevoflurane were discontinued, and CPR was initiated. Atropine and epinephrine were administered and the patient was intubated with a 5.5 cuffed endotracheal tube (ETT). The heart rate (HR) rose to 160 BPM and the BP was 95/45 mmHg. After stabilizing, she produced pink frothy fluid through her ETT. This was suctioned and she was sedated with fentanyl, paralyzed with vecuronium and ventilated with 100% O2. A chest x-ray revealed severe diffuse ground glass opacities. The case was cancelled, and the patient was transferred to the PICU. After the resuscitation, it became apparent that the patient's mother had administered Clonidine 0.1 mg (approximately 5 micrograms/kg) via G-tube at 1900, 2300 and 0400 the evening and morning prior to surgery. The patient's neurologist had prescribed this medication on a PRN basis for restlessness. The mother had never given three doses within such a short interval in the past. There was no documentation of this administration and the perioperative medical personnel were unaware of this event. The patient's chart did document a BP of 110/62 mmHg with a HR of 80 at 1700 and a BP of 69/41 mmHg and a HR of 72 at 0100.

In the PICU, an echocardiogram revealed global left ventricular systolic and diastolic dysfunction of unknown etiology. The patient required a subsequent dose of epinephrine as well as dopamine and milrinone infusions in the PICU to maintain adequate perfusion. The patient was extubated on day 4. A follow-up echocardiogram on day 7 revealed LV diastolic but not systolic dysfunction (SF = 45%). The vasopressors were weaned over a 10-day period. A chest x-ray showed no focal parenchymal process. The patient was discharged home on day 11 at her baseline neurological status.

Conclusion: The case demonstrates that large preoperative doses of clonidine may place children at risk of cardiac arrest during induction of anesthesia. 18 mcg/kg of clonidine was administered to this child over nine hours, which is significantly greater than the recommended dose of 5-7 mcg/kg². Excessive dosing of medications may occur in children with developmental delay who are receiving medications from parents as well as medical staff. Effective communication between parents, nurses, anesthesiologist and surgeon is key to properly assessing the patient's regimen. Anesthesiologists should carefully assess patients receiving clonidine for evidence of toxicity prior to inducing general anesthesia.