Polyuria induced by dexmedetomidine in a pediatric patient for spine surgery

Sheila Rajashekara MD, Nicholas Carling MD, Kim Nguyen MD

Department of Pediatric Anesthesiology, Texas Children's Hospital, Baylor College of Medicine, Houston, Texas, USA

Introduction:
Dexmedetomidine infusions are increasingly being used during spine surgery as an adjunct to total intravenous general anesthesia. We present a case in which a dexmedetomidine infusion led to possible polyuric syndrome in a pediatric patient.

Case 1:
A 14 y/o 68kg female with thoracolumbar scoliosis presented to the hospital for posterior spinal fusion surgery. After mask induction with sevoflurane, an infusion of dexmedetomidine at 0.5 mcg/kg/hr and propofol at 100 mcg/kg/min was started to maintain total intravenous anesthesia for the case. 30 minutes after the infusion was started, a urine output of 400 mLs was noted. An additional 600 mLs of urine was recorded at 3.5 hours. ABGs showed increasing plasma sodium from 140 to 149 during the time that polyuria was recorded. Serum osmolality was elevated to 305, while urine osmolality was decreased to 131. A preliminary diagnosis of dexmedetomidine-induced polyuria syndrome was made, and adequate crystalloid and colloid infusions were given to maintain hemodynamic stability. The dexmedetomidine infusion was stopped at the end of surgery and the patient was extubated. Neurological examinations were at baseline with no adverse sequelae. The patient had a total urine output of 2500 mLs at the end of the six hour surgery and total fluid administered was 2900 mLs.

Discussion:
• Dexmedetomidine infusions causing a polyuria-type syndrome, characterized by an increase in plasma sodium, low urine specific gravity and a high plasma osmolality, have been reported in adult patients. Previous work by Shirasaka and Rouch in animal models have described dexmedetomidine as both inhibiting arginine vasopressin (AVP) release from rat hypothalamic neurons as well as inhibition of AVP dependent channels in the rat cortical collecting duct.

• Since levels of vasopressin were not measured during this surgery, we cannot definitively attribute this effect as secondarily to diabetes insipidus.

• Our patient was not taking any other medication that would cause significant diuresis. In addition, urine output normalized within hours upon discontinuation of the dexmedetomidine infusion.

Conclusion:
Careful monitoring of the urine output with serial sodium levels is warranted in pediatric patients undergoing complex spine surgery with dexmedetomidine infusion as an adjuvant agent. Fluid management should be adjusted accordingly to maintain hemodynamic stability during these cases.

References: