[NM-187] Polyuria induced by dexmedetomidine in a pediatric patient for spine surgery

Rajashekara S, Carling N, Nguyen K
Texas Children's Hospital , Houston , TX, 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:
A 14 y/o 68kg female with thoracolumbar scoliosis presented for posterior spinal fusion surgery. After standard ASA monitors were applied, the patient received an inhalational induction. Sevoflurane was then discontinued and anesthesia was maintained with dexmedetomidine infusion at 0.5 mcg/kg/hr, propofol at 100mcg/kg/min, and fentanyl boluses. The patient's urine output was noted to be 400 ml within 20 minutes after the dexmedetomidine was started. At one hour, an additional 600ccs of urine was noted. Serial blood gas measurements showed increasing plasma sodium from 140 to 149, increasing serum osmolality to 305, and decreasing urine osmolality to 131. A preliminary diagnosis of dexmedetomidine-induced polyuria syndrome was made. Adequate crystalloid was 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 sequalae. Total urine output was 2300 ml at the end of the six hour surgery, blood loss was 500 ml, and total fluid administered was 3400 ml. The patient was discharged home after an appropriate recovery.

Discussion:
Dexmedetomidine infusions causing a polyuria-type syndrome have been reported in adult patients1-3. Previous work by Shirasaka4 in animal models have described dexmedetomidine as both preventing arginine vasopressin (AVP) release from rat hypothalamic neurons as well as inhibition of AVP dependent channels in the cortical collecting duct.

Although animal studies support a dexmedetomidine induced polyuria, the human literature on this phenomenon remains sparse. This is the first reported case of polyuria induced by dexmedetomidine in a pediatric patient. Of note, urine output normalized within hours upon discontinuation of the dexmedetomidine infusion. The temporal relationship between the polyuria and initiation of dexmedetomidine would suggest that dexmedetomidine was the causative agent. Despite the significant diuresis, there were no adverse events noted in our patient. 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.

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2.Prat et al. Polyuria related to Dexmedetomidine. Anesthesia and Analgesia July 2013.117(1):p150-152
3.Fuhai et al. Intraoperative hypernatremia and polyuric syndrome induced by dexmedetomidine. Journal of Anesthesiology 2013. 27:599-603.
4.Shirasaka T et al. Activation of a G protein-coupled inwardly rectifying K+ current and suppresion of Ih contribute to dexmedetomidine-induced inhibition of rat paraventricular nucleus neurons. Anesthesiology 2007. 107:605-15.
