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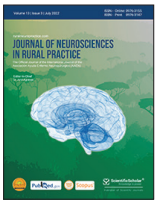

*Letter to Editor*

# Unexpected polyuria in a neurosurgical patient undergoing cerebellopontine angle tumor surgery: Can dexmedetomidine be the culprit?

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Dear Editor,

Dexmedetomidine is dextro-optical isomer of an $\alpha_2$-adrenoreceptor agonist medetomidine. It is frequently implemented as one of the components of a multimodal approach to general anesthesia. Furthermore, it is a promising drug for intraoperative neuromonitoring because of the lack of its impact on the evoked potentials. Although infrequently reported, dexmedetomidine has the potential to cause massive polyuria during intraoperative period.[1-3] Herein, we describe a case of severe intraoperative diuresis attributable to dexmedetomidine administration.

A 49-year-old female of the American Society of Anesthesiologists class 1, weighing 61 kg, having a body mass index of 26.5 kg/m$^2$, was scheduled to undergo resection of the right cerebellopontine angle tumor. She had no known comorbidities, and her investigations were unremarkable. General anesthesia was induced with fentanyl 120 μg, propofol 120 mg, and atracurium 30 mg. Standard monitoring, including invasive blood pressure monitoring, was also performed. Intraoperative neuromonitoring of the seventh, ninth, and eleventh nerves was planned. A loading dose of dexmedetomidine (1 μg/kg/h) was administered over 10 min. To achieve an adequate intraoperative depth of anesthesia, the bispectral index was targeted between 40 and 50 using the titrated infusion of propofol (50–150 μg/kg/min), dexmedetomidine (0.5 μg/kg/h), and fentanyl (1 μg/kg/h). Owing to intraoperative neurophysiological monitoring, we did not use a maintenance dose of muscle relaxant.

Approximately 30 min after starting infusions, the patient's urine output (UO) began to rise, reaching approximately 1200 mL within 1 h. Upon introspection, no known diuretic agents were administered including osmotic diuretics such as mannitol, the blood sugar level was 108 mg/dL, the core body temperature was 37.4°C, and only 750 mL of balanced salt solution was administered. After ruling out the aforementioned causes, we decreased the dexmedetomidine infusion to 0.3 μg/kg/h. Surprisingly, at the end of 2$^{nd}$ h, the UO was reduced to 700 mL. Dexmedetomidine infusion was then completely stopped, and concurrently fentanyl infusion was increased to 2 μg/kg/h. Afterward, UO further decreased to 400 mL in the 3$^{rd}$ h. In the subsequent hours of surgery, UO slowly returned to <120 mL. The total volume of fluid used was 3 L, including two packed red blood cells, over a period of 5 h. At the end of the surgery, there was a positive fluid balance of 460 mL.

Serum osmolarity was 305 mOsm/kg, while urine osmolarity was 190 mOsm/kg at the end of 1$^{st}$ h. The sodium level increased from baseline (137 mEq/L) to 143 mEq/L at the end of the 1$^{st}$ h and then decreased to 139 mEq/L by the end of the procedure. Subsequently, the patient was shifted to neurosurgical intensive care for elective mechanical ventilation. Post-operatively, no further increase in UO and sodium level was recorded, and the remaining hospital stay was uneventful.

Polyuria in adults is either due to the excretion of non-absorbable solutes including glucose (solute diuresis) or following the excretion of water from abnormalities in the production of arginine vasopressin or responsiveness of the renal system (water diuresis).[2] There is a plethora of differential diagnoses for polyuria in neurosurgical patients [Figure 1].

The sudden onset of massive polyuria, resulting from water diuresis, i.e., a decreased urine specific gravity, reduced urine

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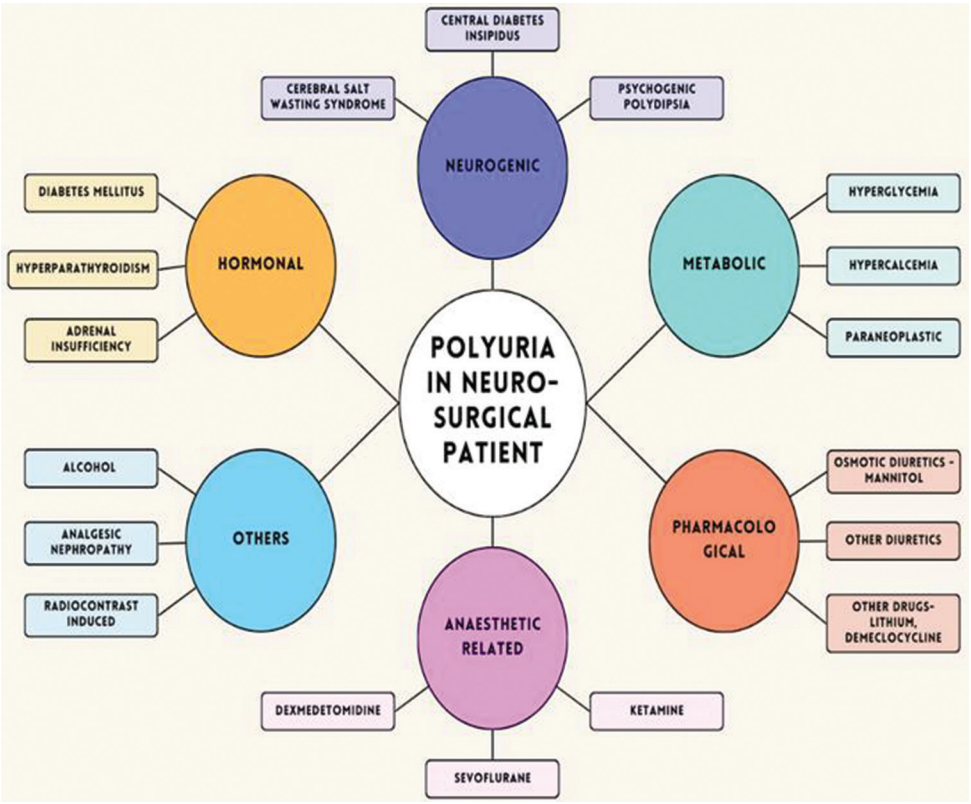


**Figure 1:** Causes of polyuria in neurosurgical patients.

osmolality concurrent with elevated serum osmolality, and sodium, was the most remarkable intraoperative event in the indexed case. In addition, polyuria was not observed before or after surgery. The potassium, calcium, and creatinine levels were in the normal range. Van Decar *et al*., in a review, have recommended considering anesthetic agents as the cause of polyuria after excluding other possibilities.[4] Hence, the same was considered in our case; further, polyuria resolved after discontinuing dexmedetomidine infusion.

Various animal studies have reported the potential of dexmedetomidine to induce diuresis of free water. The mechanism is probably from the inhibition of vasopressin by the pituitary and/or inhibition of hypothalamic paraventricular nucleus magnocellular neuronal signaling.[5-7] In humans, a handful of cases of dexmedetomidine-induced diuresis have been reported in the non-neurosurgical population.[1-3]

Urine osmolality helps to distinguish the cause of polyuria. UO exceeds 3 L/day with osmolality <250 mOsm/L, i.e., dilute urine indicates water diuresis. It occurs either because of polydipsia caused by inadequate vasopressin secretion, as occurs in central diabetes insipidus, or because of failure of vasopressin to act on renal tubules as seen in nephrogenic diabetes insipidus.[4]

Worldwide, dexmedetomidine is used for the contemplation of intraoperative neuromonitoring during neurosurgery. As a neuroanesthetist, we should be vigilant about dexmedetomidine-associated polyuria after excluding all possible causes of polyuria in neurosurgical patients [Figure 1]. Vigilant UO and serum sodium level monitoring should be contemplated for all neurosurgical patients receiving an infusion of dexmedetomidine.

### Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

### Conflicts of interest

There are no conflicts of interest.

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