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DEXMEDETOMIDINE-ASSOCIATED POLYURIA MIMICKING DIABETES INSIPIDUS

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ABSTRACT

Dexmedetomidine is a highly selective α2-adrenergic agonist widely used for sedation in the intensive care unit (ICU) and is generally considered well tolerated, with hypotension and bradycardia being the most commonly reported adverse effects. Rarely, dexmedetomidine has been associated with polyuria resembling a diabetes insipidus-like syndrome, particularly during prolonged infusions. We report the case of a 53-year-old male admitted to the ICU for severe alcohol withdrawal who developed profound polyuria during a 5-day dexmedetomidine infusion. Urine output progressively increased to 6.75 litres per day, prompting an extensive evaluation that excluded osmotic diuresis, infection, intracranial pathology, and nephrogenic diabetes insipidus. Laboratory studies demonstrated preserved renal function and normal serum electrolytes. The temporal association between dexmedetomidine initiation and polyuria onset, along with resolution following drug discontinuation, supported the diagnosis of dexmedetomidine-associated polyuria, with a Naranjo adverse drug reaction probability score of 8 indicating a probable association. This case highlights an underrecognized adverse effect of dexmedetomidine. It underscores the importance of monitoring urine output and electrolytes during prolonged infusions to facilitate early recognition, prevent unnecessary diagnostic testing, and avoid complications related to volume depletion and electrolyte imbalance.

KEYWORDS

Dexmedetomidine, polyuria, diabetes insipidus-like syndrome, intensive care unit

LEARNING POINTS

- Dexmedetomidine can, in rare cases, cause profound polyuria mimicking central diabetes insipidus, particularly during prolonged infusions beyond the Food and Drug Administration -approved 24-hour duration.
- Early recognition of dexmedetomidine-associated polyuria can prevent unnecessary diagnostic evaluations, reduce the risk of volume depletion and electrolyte disturbances, and prompt timely discontinuation of the offending agent.
- Internists managing critically ill patients should closely monitor urine output and serum electrolytes during extended dexmedetomidine use, especially in patients with hepatic impairment or refractory delirium.

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INTRODUCTION

Dexmedetomidine is an α2-adrenergic agonist commonly used for sedation in the intensive care unit (ICU). While its most recognized adverse effects are hypotension and bradycardia, emerging evidence highlights a rare but clinically significant complication: unexpected polyuria, often manifesting as a diabetes insipidus-like syndrome[1]. Polyuria is rare in ICU patients and can present a diagnostic challenge. We report a case of dexmedetomidine-associated polyuria in a patient treated for severe alcohol withdrawal, highlighting the clinical course, diagnostic evaluation, proposed mechanisms, and management considerations. Raising awareness of this uncommon side effect may help clinicians recognize and manage it early, potentially preventing complications related to volume depletion and electrolyte disturbances in critically ill patients.

CASE DESCRIPTION

A 53-year-old male with a past medical history of hypertension, alcohol use disorder, depression, and anxiety were brought to the emergency department (ED) following increased aggression and subsequent minimal responsiveness. In the ED, the patient was initially difficult to arouse but later became extremely agitated. He received haloperidol (5 mg twice) and lorazepam (2.5 mg twice) for agitation. Vital signs revealed a blood pressure of 133/106 mmHg, a heart rate of 106 beats per minute, a respiratory rate of 15 breaths per minute, and a temperature of 37.1°C. Initial laboratory results revealed a white blood cell count of 12.2 mm^3 with no bands, an ALT of 65 U/l, an AST of 76 U/l, a glucose level of 231 mg/dl, and a significantly elevated blood alcohol level of 467.2 mg/dl. Urine toxicology was unremarkable. The patient was diagnosed with acute alcohol intoxication with impending withdrawal and was admitted to the medical floor. However, due to escalating agitation, he was transferred to the ICU and started on continuous infusion of dexmedetomidine. He was concurrently managed with intravenous diazepam and phenobarbital for agitation control. Over the ensuing 5 days of dexmedetomidine infusion, the patient developed progressively increasing urine output. Urine output was minimal on day one, increased to approximately 1.5 litres by day 2, and peaked at 6.75 litres on day 5. Urine osmolality was measured at 322 mOsm/kg H_2O. Random urine sodium increased from 20 mmol/l to 125 mmol/l over this period.

An extensive evaluation of the cause of polyuria was performed. A computed tomography scan of the head showed no evidence of intracranial or pituitary pathology. Serum electrolytes remained within normal limits, and the patient was clinically euvolemic on examination. Laboratory values included blood urea nitrogen of 7 mg/dl, creatinine of 0.72 mg/dl, serum glucose of 130 mg/dl, sodium of 142 mmol/l, potassium of 3.5 mmol/l, and calcium of 7.2 mg/dl. Urine electrolyte analysis revealed sodium at 125 mmol/l, potassium at 13 mmol/l, chloride at 22 mmol/l, and creatinine at 25.2 mg/dl. No medications known to cause nephrogenic diabetes insipidus were identified, and osmotic diuresis was excluded.

Based on an extensive negative workup, along with the temporal association between the onset of polyuria and the initiation of dexmedetomidine and its resolution upon discontinuation, we concluded that the polyuria was likely due to central diabetes insipidus, induced by the dexmedetomidine infusion. Application of the Naranjo adverse drug reaction probability scale yielded a score of 8, consistent with a probable adverse drug reaction.

DISCUSSION

Dexmedetomidine is generally considered well-tolerated. The most commonly reported side effects include hypotension, hypertension, and bradycardia[1]. This case illustrates a rare but recognized side effect of dexmedetomidine: drug-induced polyuria resembling diabetes insipidus, which occurred with prolonged sedation for alcohol withdrawal symptoms. In a systematic review of sedation-related diabetes insipidus in the ICU, dexmedetomidine was implicated in 42.9% of all cases, with 81.8% of patients who were given dexmedetomidine developing this complication (95% CI 48.2-97.7%)[1].

The temporal relationship in our case between the 5-day dexmedetomidine infusion and the onset of polyuria, followed by normalization after discontinuation, strongly suggests a causal association[2]. The Naranjo algorithm offers a standardized and objective method for assessing the probability of causation between a suspected drug and an adverse clinical event[3]. When applying the Naranjo score to this case, we got a value of 8, which represents a “probable” adverse drug reaction to dexmedetomidine. Prolonged infusion duration beyond the Food and Drug Administration (FDA)-approved 24-hour maximum appears to be the primary risk factor. Most reported cases involve continuous infusions lasting multiple days, as in the presented case with a 5-day infusion[1,2,4]. Higher doses also increased risk, as dexmedetomidine induces polyuria in a dose-dependent fashion[2].

The proposed mechanism of dexmedetomidine-associated polyuria involves α2-adrenergic receptor-mediated suppression of arginine vasopressin release from the hypothalamic paraventricular nucleus[2,4]. This central effect may reduce circulating vasopressin levels, resulting in impaired free water reabsorption and increased urine output[2]. Additionally, peripheral renal effects have been described, including increased collecting duct permeability independent of vasopressin, further contributing to free water loss in a dose-dependent manner[2]. Together, these mechanisms can produce a clinical picture resembling central diabetes insipidus, though laboratory findings may be variable.

Recognition of this adverse effect is critical, as persistent polyuria may lead to hypovolemia, electrolyte disturbances, and hemodynamic instability. Management primarily

involves early identification and discontinuation or dose reduction of dexmedetomidine. Close monitoring of urine output, serum sodium, serum osmolality, urine osmolality, and urine specific gravity is recommended, particularly when dexmedetomidine infusions are continued beyond 24 hours[1,5]. Resolution typically occurs within 2-24 hours of discontinuing dexmedetomidine[1,6,7] In severe cases with dangerous hypernatremia, temporary vasopressin analogue therapy (desmopressin or pituitrin) may be considered, though most cases resolve spontaneously with drug cessation[6].

Prevention strategies include limiting infusion duration to the FDA-approved 24 hours whenever possible, using the lowest effective dose, and considering alternative sedation strategies for patients who require prolonged sedation[1,5]. Given the widespread use of dexmedetomidine, increased awareness of this uncommon adverse effect may reduce unnecessary diagnostic testing and facilitate timely management.

CONCLUSION

Dexmedetomidine-associated polyuria is a rare but clinically significant adverse effect that may mimic diabetes insipidus in critically ill patients. Prolonged infusion appears to be a key risk factor. Clinicians should maintain a high index of suspicion for this diagnosis in patients who develop unexplained polyuria while receiving dexmedetomidine. Prompt recognition, careful monitoring, and timely discontinuation of the drug can lead to rapid resolution and prevent associated complications.

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