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The proof is in the pee: Dexmedetomidine induced diabetes insipidus

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Abstract

Dexmedetomidine induced diabetes insipidus is extremely rare phenomenon and typically seen in surgical cases. There are only a handful of case reports of dexmedetomidine induced diabetes insipidus in non-surgical settings. The mechanism of diabetes insipidus is unclear but thought to be secondary to inhibition of the neurohypophysis release of Antidiuretic Hormone (ADH). It is necessary to evaluate the development of diabetes insipidus in patients being treated with dexmedetomidine to avoid complications such as dehydration, hypotension, coma and death.

Background

Dexmedetomidine is a highly selective α2-adrenergic receptor with minimal side effects in most patients and due to the α2 effect, has minimal cross reactivity. Identification and treatment of dexmedetomidine induced Diabetes Insipidus (DI) is imperative to evade complications of dehydration, coma and death. Recognizing DI and pivoting to other medication choices is an essential step in continuing to provide excellent care for hospitalized patients.

Case presentation

A 62-year-old male with a past medical history significant for hepatocellular carcinoma, pulmonary hypertension, and alcoholic cirrhosis presented to the medical intensive care unit with signs of a gastrointestinal bleed with relative hemodynamic instability.

The patient required increased fluids and blood products to maintain his blood pressure. Pt was transfused 5 units for continued stability. Pt underwent esophagogastroduodenoscopy and colonoscopy which were negative for acute bleeding. Despite the patient's likely hypovolemia, patient continued to be hypertensive and tachycardic. Pt was started on dexmedetomidine for alcohol withdrawal. The following morning, patient was noted to have very large urine output of approximately 3-5 liters in 24 hours.

The patient's sodium was extremely variable throughout the hospital course. The patient's initial sodium value was 141 mmol/L, which increased to 159 mmol/L three days after admission. with large urine output which and a calculated free water deficit of approximately 4-5L on over the following days. The patient was intermittently on and off the precede drip and treatment for alcohol withdrawal was augmented with chlordiazepoxide and lorazepam. Free water was replaced through free water flushes. Patient continued and off the dexmedetomidine drip for tachycardia, hypertension and agitation. The patient was given a dose of desmopressin which resulted in normalization of sodium to 137 mmol/L. The patient's sodium continued to be liable Nephrology was consulted and recommended starting desmopressin. The patient was completely weaned off dexmedetomidine and his sodium resolved to 144 mmol/L.

Investigations

Investigation of patient's hypernatremia started with measurement of the assessment of the patient's volume status, which was considered euvolemic. His serum and urine osmolarity resulted as a urine osmolarity of 130 mOSM/kg and serum of 303 mOSM/kg. Urine sodium was elevated above 20mmol/L on all checks and ranged from 54-126 mmol/L. Further, patient was given desmopressin which subsequently led to more concentrated urine increasing the suspicion for central diabetes insipidus.

Differential diagnosis

Large urine output in patient was thought to perhaps be secondary to an ATN or post obstructive diuresis as the patient came in hypovolemic from a GI bleed. However, the increase in the patient's sodium and his diluted urine was diagnostic for diabetes insipidus.

Treatment

The patient was treated with desmopressin leading to decreased urination. After the cause of the increased urination was determined to be dexmedetomidine, the patient was ultimately treated with stopping the dexmedetomidine infusion.

Outcome and follow-up

Patient survived and was followed up in September of the same year where his sodium was back within normal range at 139 mmol/L. Upon chart review, the patient did not have any further increase in liability of his sodium since discontinuation off dexmedetomidine.

Discussion

Per our review, there are very few cases of dexmedetomidine causing diabetes insipidus. Only one report was of dexmedetomidine being used for alcohol withdrawal, one was in child and the remaining cases were of diabetes insipidus in patients with spinal pathology [1,3-6].

Most cases of dexmedatomidine induced diabetes insipidus included cases of surgical patients in a peri or intraooperative setting and with pharmacologic treatment secondary to either lithium or demeditomidine [2]. However, there is little evidence of dexmedetomidine causing DI in medical settings.

The pathophysiology of diabetes insipidus secondary to dexmedetomidine is unclear at this point. One school of thought are the effect is at the level of the hypothalamus which decreases the excretion of ADH. The second is that dexmedetomidine has an effect directly at the level of the hypothalamus neurohypophyses to decrease ADH secretion via stimulation of a2 adrenergic hormone. The third is the indirect drug effect to decrease the stress experienced by the body leading to a decrease in secretion of ADH [6]. Further, it is observed that inadequate reabsorption of vasopressin leads to a functional loss of aquaporins and increased diuresis increasing the diuretic effect. While dexmedetomidine is highly specific for the α2, the pharmacology of the medication is diverse.

Dexmeditomidine is α2-adrenoceptor agonsit with central nervous system effects of anxiolytic, sympatholytic and analgesic sparing effects. It is much more selective to α2 than α1 leading to more sedation than activation. The pre- and postsynaptic α2-receptors is in the locus coeruleus with hypnotic action through activation of central nervous system. Further, it is metabolized through glucuronidation and hydroxylation of the liver, and metabolism by the CYP system (CYP2A6) which may have some predisposing side effects in patients with liver cirrhosis. This may provide some insight into why our patient had the side effect of diabetes insipidus as perhaps the altered etabbolism predisposes the body to increased side effects. Further, evaluation leads to thoughts that the cardiac output and patient's albumin level, as it is highly protein bound, leads to changes in metabolism [7]. This may have been a contributing factor in our patient as his albumin was 2.7 g/dL at the time of admission. Further his INR was elevated to 1.3 and he had documented cirhosis on imaging. However, when evaluating the case these seem to be the dominate factors that may have contributing to the case.

Learning points

- Dexmedetomidine is a common pharmacologic therapy used in ICU settings for agitation.
- The side effect of diabetes insipidus is rare but should be recognized the patient and vomiting with increased urine output.
- It is unknown what predisposes patients to developing diabetes insipidus with Dex accommodate administration and research is needed in this area.

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