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INTRODUCTION

Hyponatremia is one of the rare side effects of non steroidal anti-inflammatory drugs (NSAIDs) which can be potentiated in combination with desmopressin (DDVAP). Here we describe a case of hyponatremia induced by meloxicam in a patient with acquired central diabetes insipidus on DDAVP.

CASE REPORT

A 56-year-old female was admitted to the emergency department with confusion and aphasia over the past 12 hours. Her past medical history is significant for panhypopituitarism and central diabetes insipidus secondary to transphenoidal resection of a pituitary adenoma. Her medications include intranasal DDAVP (10 mcg/spray every 8 hours), hydrocortisone (15 mg in the morning and 5 mg in the afternoon) and levothyroxine (200 mcg daily). 5 days before admission she was prescribed meloxicam 15 mg daily for back pain. Since then she noticed progressive weight gain, lower extremity swelling and bloating. Patient reported no history of hyponatremia, smoking, alcohol or illicit drug use.

Her physical exam on admission was normal except for some altered orientation.

The diagnostic work up revealed: sodium, 118 mmol/L; potassium 4.2 mmol/L; chloride, 90 mmol/L; blood urea nitrogen 10 mg/dl; creatinine, 0.8 mg/dl; glucose, 94 mg/dl and serum osmolarity 249 mosm/kg (285-295 mosm/kg). Urine osmolarity 548 mosm/kg and urine sodium 178 mEq/L. Free T4 was normal at 1.4. The results of computed tomography of the head showed no acute findings.

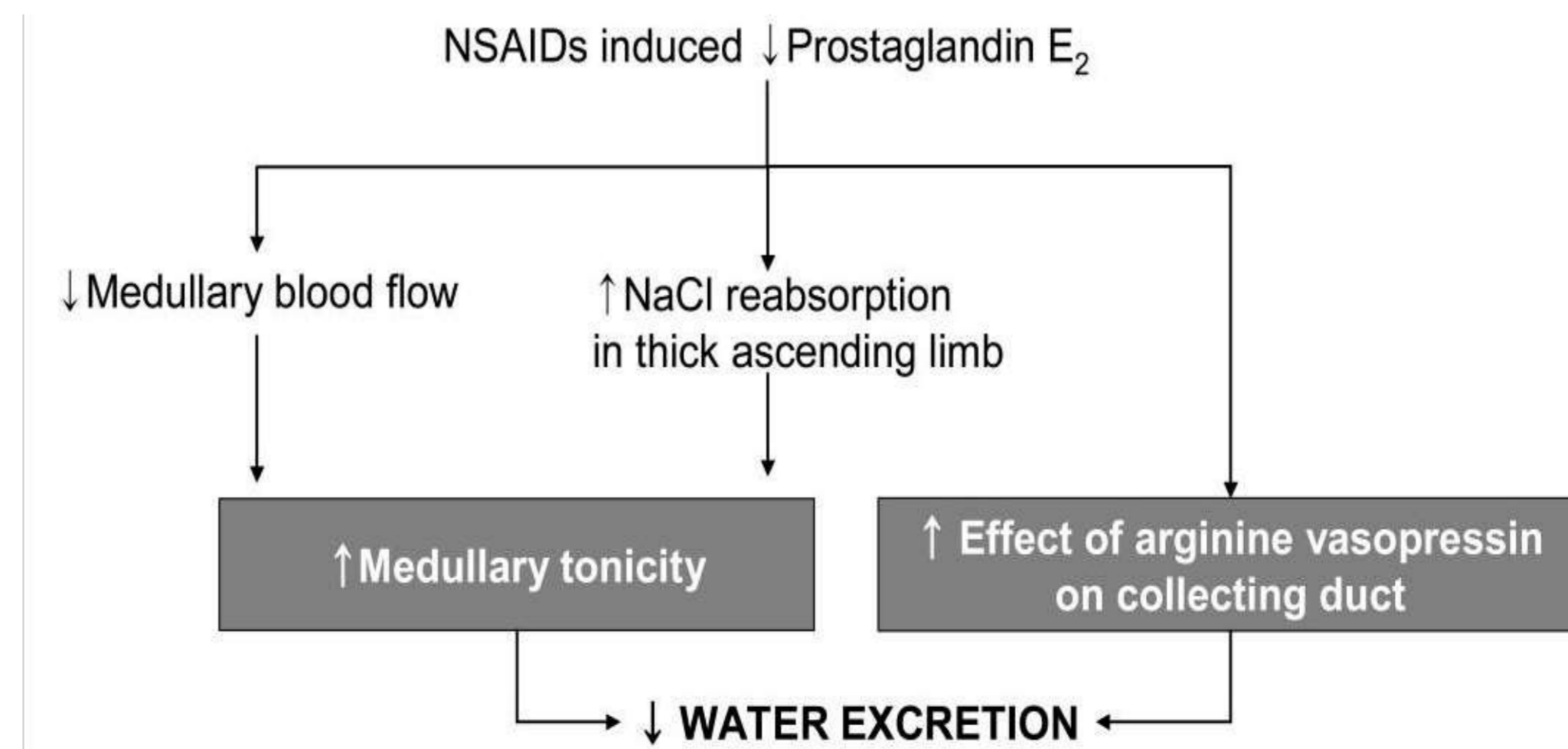
During hospitalization, DDAVP and meloxicam were held allowing over diuresis. Hyponatremia resolved over the next 3 days with sodium level of 135 mmol/L on discharge. DDAVP was resumed at reduced dose (10 mcg/spray every 12 hours).

DISCUSSION

NSAIDs are commonly prescribed medications and one of their rare side effects include hyponatremia. NSAIDs facilitate the effect of antidiuretic hormone (ADH) by diminishing the normal inhibitory effect of prostaglandins on the activity of ADH causing water retention and thus hyponatremia. Patients taking DDVAP are at a higher risk of hyponatremia due to the increased antidiuretic effect of DDVAP potentiated by NSAIDs. Other factors contributing to increased risk of hyponatremia include advanced age, female sex, increasing doses of DDVAP. This serious interaction between DDVAP and NSAIDs has been rarely reported in the literature, making this case interesting. Also most cases have been described in patients taking DDVAP for nocturnal enuresis and coagulopathies rather than replacement therapy in central diabetes insipidus which makes it more unique.

CONCLUSION

Clinicians should recognize the severe implication of hyponatremia associated with concomitant use of DDVAP and NSAIDs. Patients taking DDVAP should be advised to use alternative therapeutic interventions to NSAIDs when possible.



REFERENCES

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