

# Iatrogenic Central Diabetes Insipidus Induced by Vasopressin Withdrawal

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## Abstract

**Objective:** We would like to report a potential new adverse effect of vasopressin infusions. We had two recent patients with traumatic brain injuries develop central diabetes insipidus temporally related to the discontinuation of vasopressin infusion. Although both patients met diagnostic criteria for DI and required therapeutic interventions (desmopressin and free water replacement), it resolved after a short period of approximately 12-24 hours.

**Data Sources:** Direct patient care in the Trauma ICU at a tertiary care, level 1 trauma center, and review of the electronic medical records.

**Conclusion:** Withdrawal of vasopressin inducing or unmasking diabetes insipidus has not been reported in the literature. This iatrogenic effect may be more likely to occur in brain injured patients but may severely impact their ICU care.

## Keywords

Diabetes insipidus; Vasopressin; Iatrogenic

## Introduction

Vasopressin infusions are used for a variety of therapeutic indications and are known to have adverse effects such as mesenteric ischemia, skin and digital necrosis, and hyponatremia. We would like to report another potential complication: vasopressin withdrawal induced central diabetes insipidus. We recently had two patients with brain injuries who developed polyuria and hypernatremia immediately after having their vasopressin infusions stopped and met all diagnostic criteria for diabetes insipidus.

## Patient A

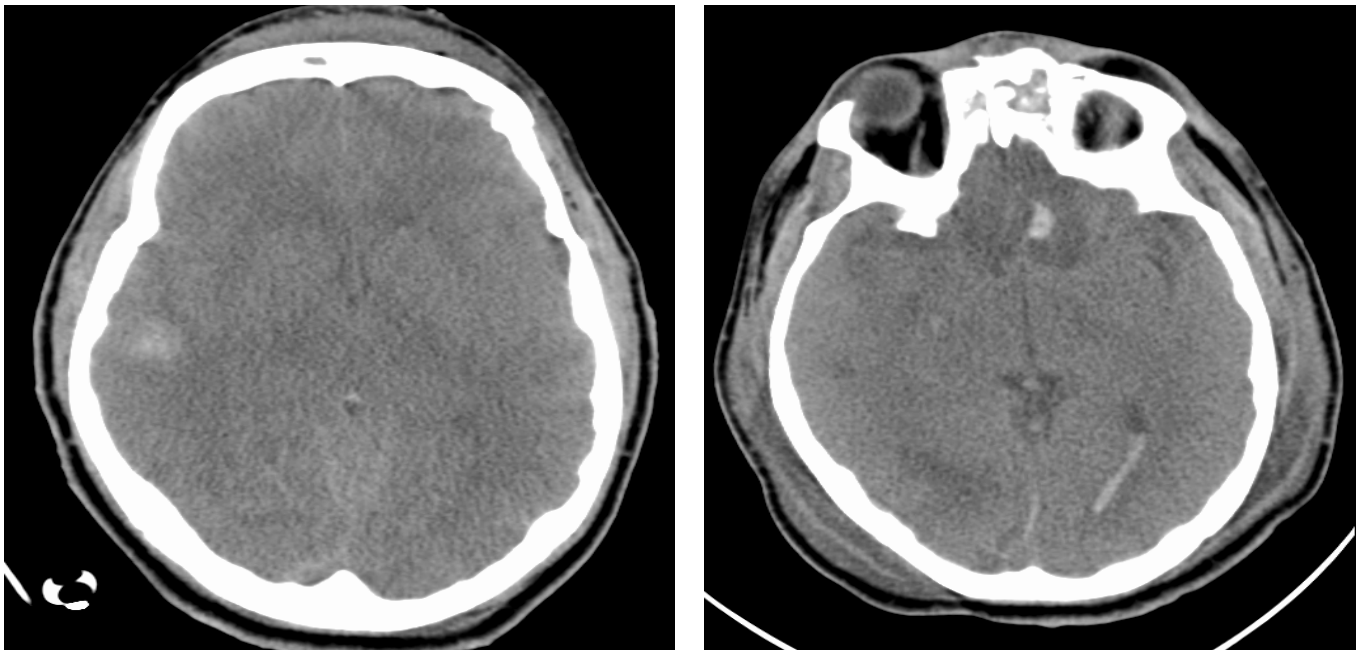
Patient A was a 26 year-old woman with subdural hematoma and intracerebral contusions sustained after fall from a horse. She had a right craniotomy and evacuation of hematoma with placement of EVD. She was sedated and receiving mechanical ventilation. She received norepinephrine and vasopressin infusions (0.04 u/minute) in order to maintain CPP >60 mmHg; although her ICPs were

not >20 mmHg but typically 15 mmHg she required vasopressor support to achieve a MAP of approximately 75 mmHg. She was eventually weaned off norepinephrine and then ultimately vasopressin also after a total duration of approximately 96 hours; the vasopressin infusion was first reduced to 0.02 u/minute for one hour before discontinuing it completely. Within 90 minutes of discontinuing the vasopressin infusion, polyuria occurred with urine output > 1L/hour for several hours without diuretic administration. Urinary sodium and osmolality were very low and serum sodium had risen from 151 to 163 mg/dL within 6 hours. We administered desmopressin 2mg subcutaneously once and initiated free water replacement with improvement of urine output and serum sodium. She did not require any additional interventions as the diabetes insipidus appeared to have resolved within 12 hours.



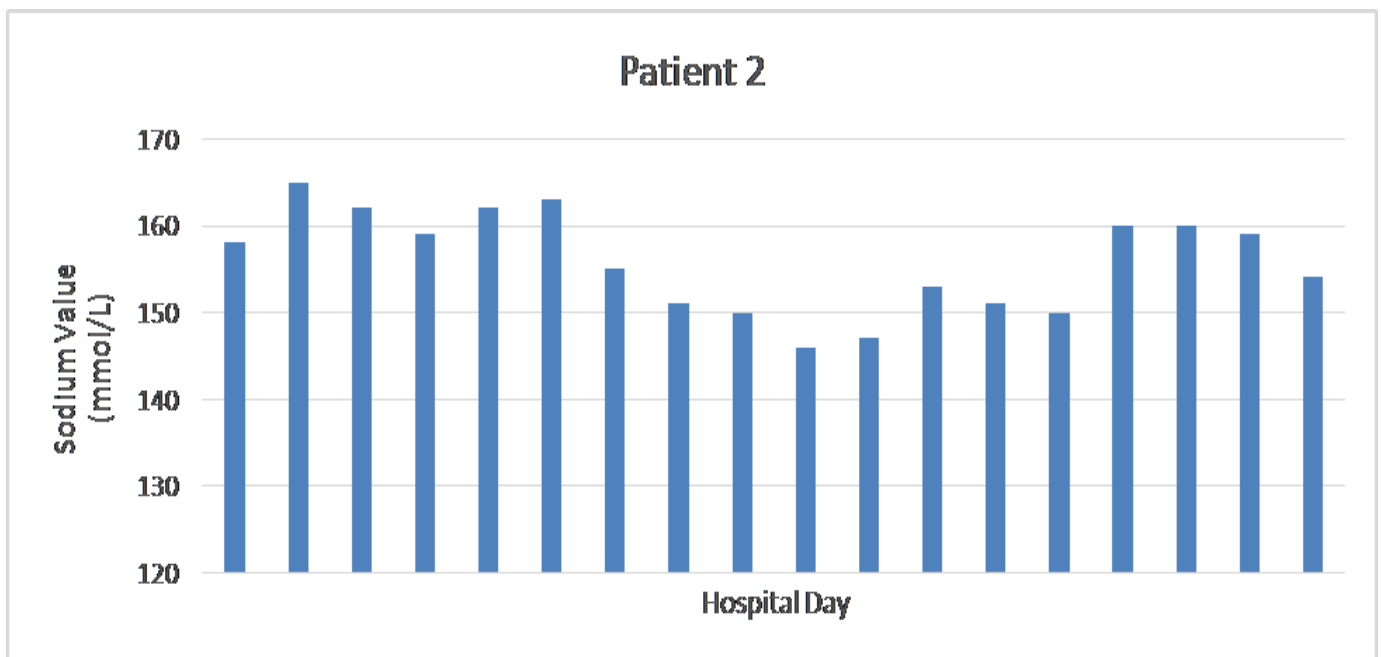
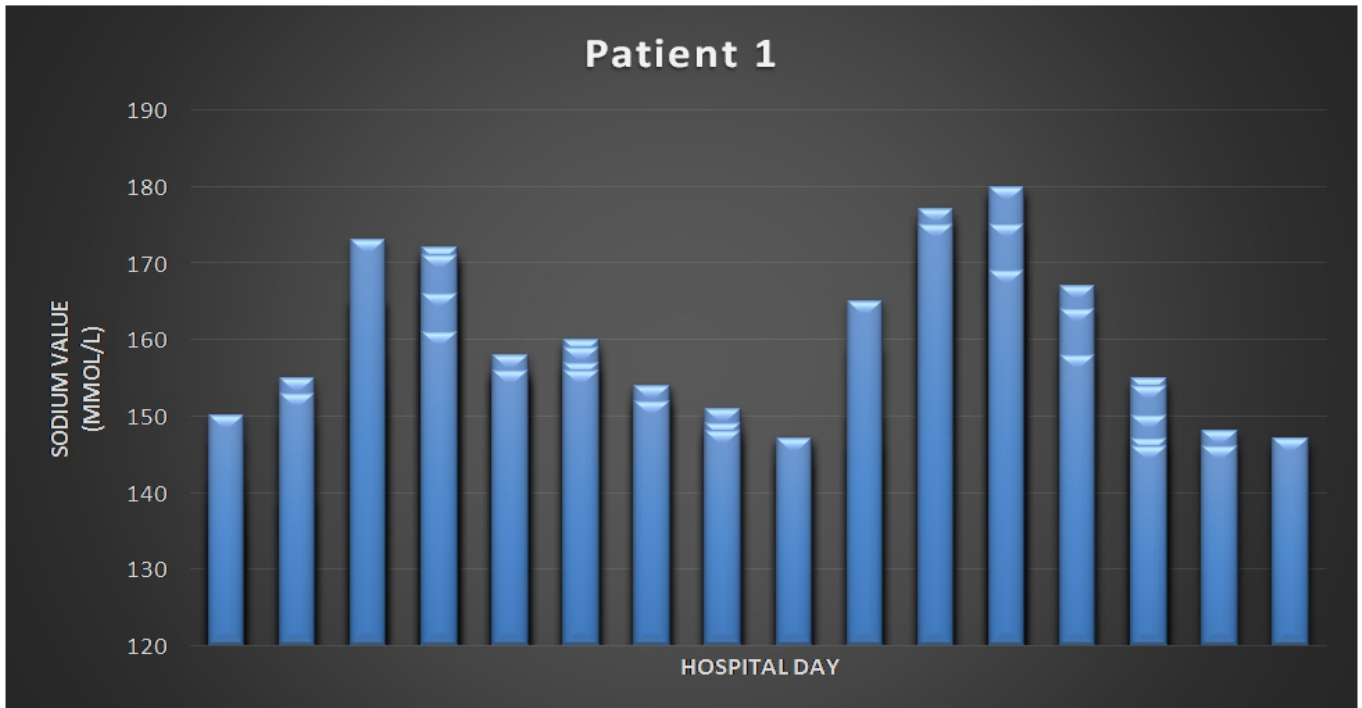
## Patient B

Patient B was a 47 year-old man who sustained a traumatic subarachnoid hemorrhage and intracerebral contusions during a motor cycle collision; he also sustained bilateral rib fractures and pulmonary contusions with hemo-pneumothoracies, thoracic vertebral fractures (T4 and T5) a closed left femur fracture and open left tibia fracture. In addition to crystalloid and blood product administration, he was placed on norepinephrine and vasopressin infusions (also 0.04 u/minute) for blood pressure support to maintain MAP > 80 in order to optimize spinal perfusion pressure. His vasopressin infusion was reduced to 0.02 u/minute for one hour before being stopped after a total time of approximately 72 hours; one hour later he developed polyuria (up to 2 L urine output per hour without diuretic administration) and his serum sodium increased from 147 to 158 mg/dL within 6 hours. His urinary sodium and osmolality were also low consistent with diabetes insipidus. He received a subcutaneous dose of desmopressin and free water replacement with resolution of the polyuria. His case was complicated by the ICU team responding to his serum sodium decreasing over 6-12 hours below 150 and concern over increasing ICP due to cerebral edema, so that hypertonic saline was then given as an intravenous bolus and an infusion started. His serum sodium thus increased again but he did not require any additional doses of desmopressin.



## Conclusion

When administered for septic shock, abruptly discontinuing vasopressin infusion can cause a rapid drop in vasopressin levels leading to hypotension. It is not clear if this effect is due to suppression of endogenous vasopressin production or release.<sup>1</sup> We were not able to find any previous reports of the occurrence of diabetes insipidus after the discontinuation of vasopressin infusion. It appears that these two patients had a brief suppression of their endogenous vasopressin levels due to the exogenous administration and developed central diabetes insipidus that met diagnostic criteria and required treatment intervention. A second possibility is the unmasking of unrecognized DI that was being treated while on vasopressin infusion. We believe that this potential adverse effect may occur in critically ill patients, possibly specifically those with brain injuries, and providers for these patients should be made aware.



Both patients had hypernatremia initially due to hypertonic saline loading as treatment for elevated ICP due to cerebral edema; then their sodium levels were allowed to decrease as their condition improved. When the Vasopressin infusions were stopped, their sodium values increased rapidly without hypertonic saline administration.

## References

1. Russell, JA. Bench-to-bedside review: vasopressin in the management of septic shock. *Critical Care* 2011;15:226-245

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