JCEM Case Reports, 2026, **4**, luag132
https://doi.org/10.1210/jcemcr/luag132
Published: 18 May 2026
Image in Endocrinology

Glucocorticoid-induced arginine vasopressin deficiency and neurohypophyseal T1-hyperintensity transient switch-off

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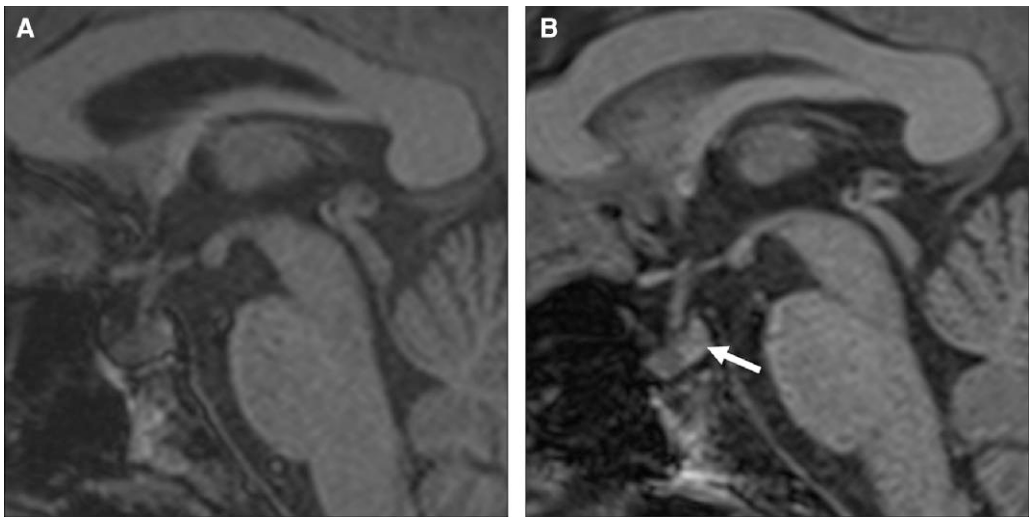
Key Words arginine vasopressin (AVP), antidiuretic hormone (ADH), AVP deficiency, central diabetes insipidus, glucocorticoids

Image legend

A 57-year-old-man presented with polyuria, polydipsia, and weight loss 1 week after betamethasone injection because of back pain. His medical and drug history was unremarkable. His laboratory work-up revealed hypernatremia with serum sodium of 146 mEq/L (SI: 146.00 mmol/L; reference range, 136-145), and normal serum levels of glucose (97 mg/dL; SI: 5.38 mmol/L), urea (40 mg/dL; SI: 14.28 mmol/L) and potassium (4.2 mEq/L; SI: 4.20 mmol/L), with a serum osmolality calculated at 312 mOsm/kg (SI: 312 mmol/kg; reference range, 285-295). His baseline pituitary function was normal. Pituitary magnetic resonance imaging (MRI) showed absent neurohypophyseal hyperintensity on T1-weighted images (panel A). He commenced oral desmopressin (60 mcg sublingual tablet at bedtime) with immediate



Received: 5 March 2026. **Accepted:** 20 April 2026. **Corrected and Typeset:** 18 May 2026
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improvement. Two weeks later, he noted decreased thirst and diuresis, which led him to withdraw desmopressin. After discontinuing desmopressin, he remains asymptomatic and his pituitary MRI 6 months later showed reappearance of the neurohypophysis T1-hyperintensity (panel B, arrow). T1-hyperintense neurohypophysis "bright spot" represents the presence of stored arginine vasopressin (AVP), and its absence indicate failure to produce/store AVP, a key MRI finding of AVP deficiency (central diabetes insipidus). Glucocorticoids inhibit AVP secretion centrally, and induce renal AVP resistance, leading to polyuria-polydipsia [1, 2]. There are a few polyuria-polydipsia cases reported following systemic corticosteroid treatment, where both AVP deficiency and resistance mechanisms were postulated [2]. In our case, neurohypophysis T1-hyperintensity reappearance after glucocorticoid washout, and the response to desmopressin in acute phase, support a glucocorticoid-induced central AVP suppression mechanism rather than AVP resistance or AVP-independent mechanisms. This case alerts for the importance of recognizing the entity of transient AVP deficiency following systemic corticosteroids to avoid mismanagement and highlights the usefulness of pituitary imaging in the differential diagnosis and clarification of pathophysiological mechanisms.

Contributors

All authors made individual contributions to authorship. P.M., L.F., I.S.: were involved in the clinico-biochemical diagnosis and management of this patient. L.N.: was involved in the imaging studies of this patient. All authors reviewed and approved the final draft.

Funding

No public or commercial funding.

Disclosures

The authors have nothing to disclose.

Informed patient consent for publication

Signed informed consent was obtained directly from the patient.

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