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Case Report

Adipsic Diabetes Insipidus: Beyond Diabetes, what are the Specific Features?

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Abstract

Adipsic diabetes insipidus (ADI) is a rare form of central diabetes insipidus characterized by hypotonic polyuria associated with absent or impaired thirst, causing a devastating disorder of water balance with significant associated morbidity and mortality. Adipsic diabetes insipidus results from injury to osmosensitive neuroreceptors, mainly located in the paraventricular and supraoptic nuclei, which leads to reduced synthesis and secretion of arginine vasopressin. Key neural circuits governing thirst perception and drive are anatomically colocalized with arginine vasopressin-producing centers and are therefore affected by the same injury, leading to impaired thirst and severe water balance dysregulation. In most patients, the disease arises from destruction of the hypothalamus due to a variety of underlying causes. Adipsic diabetes insipidus has most frequently been linked to surgical clipping of anterior communicating artery aneurysms after subarachnoid hemorrhage, as well as to craniopharyngioma and certain congenital disorders. Meanwhile, suprasellar arachnoid cysts are an uncommon cause. In this case, the suprasellar arachnoid cyst was responsible for hypothalamic-pituitary compression. Diagnosis relies on clinical suspicion, biochemical findings, and imaging. Adipsic diabetes insipidus is a rare but serious condition requiring early recognition. Although often asymptomatic, suprasellar arachnoid cysts can lead to this complication. Management is based on combining hormonal therapy such as arginine vasopressin replacement, careful fluid management, and etiological treatment. Furthermore, keeping serum sodium levels within the normal range is often difficult in these patients, and marked fluctuations in sodium levels frequently lead to recurrent hospitalizations. Thus, a multidisciplinary approach is essential to prevent severe complications and improve outcomes.(Kothari *et al*., 2021a) (Blevins & Wand, 1992)

Keywords: Adipsic diabetes insipidus; Central diabetes insipidus; Suprasellar arachnoid cyst; Hypothalamic-pituitary axis; Polyuria; Desmopressin; Hypernatremia; Hormonal insufficiency.

INTRODUCTION

Water homeostasis is maintained through two critical and complementary mechanisms: the secretion of arginine vasopressin by the neurohypophysis and the thirst response mediated by hypothalamic osmosensitive neurons. Both are primarily regulated by changes in plasma osmolality, allowing osmotic equilibrium to be maintained within a narrow physiological range (normal serum osmolality:285-295mOsm/kg). a rise in plasma osmolality above this threshold simultaneously stimulates arginine vasopressin release, promoting renal free water reabsorption, and activates the thirst response, thereby ensuring coordinated defense against hyperosmolarity and dehydration(Eisenberg & Frohman, 2016).

Diabetes insipidus (DI) is a rare disorder characterized by hypotonic polyuria, most often associated with compensatory polydipsia. It results either from a deficiency of arginine vasopressin in central diabetes insipidus (CDI) or from renal resistance to arginine vasopressin in nephrogenic diabetes insipidus (*Arginine Vasopressin Deficiency (Central Diabetes Insipidus): Etiology, Clinical Manifestations, and Postdiagnostic Evaluation - UpToDate*, n.d.)

A particularly rare form is adipsic diabetes insipidus, in which polyuria is associated with an absence or reduction of thirst sensation. This condition carries a high risk of dehydration and is usually secondary to hypothalamic or hypothalamic-pituitary lesions affecting the thirst center and the

neurohypophysis. Suprasellar arachnoid cysts are often asymptomatic; however, when they enlarge the sella turcica and compress the pituitary parenchyma, they may lead to rare endocrine complications, including central diabetes insipidus and, more rarely, adipsic diabetes insipidus (Eisenberg & Frohman, 2016; Kothari *et al*., 2021b). We describe a rare case of adipsic diabetes insipidus secondary to a suprasellar arachnoid cyst associated with enlargement of the sella turcica, with emphasis on the underlying pathophysiology, diagnosis workup, and therapeutic management. (Yang *et al*., 2024)(Makaryus & McFarlane, 2006)

CASE PRESENTATION

We report the case of a 60-year-old woman presenting with a two-year history of significant polyuria, estimated at 4.5 liters/24 hours (78 mL/kg/24 h), in the context of limited fluid intake of approximately 700 mL/day. The patient did not report any excessive thirst sensation. She also described sleep disturbances due to nocturnal awakenings related to polyuria, associated with right temporal headaches and visual blurring, without nausea or vomiting. On clinical examination, the patient was conscious, with normal hemodynamic parameters and a normal body mass index (BMI : 24 kg/m²). Ophthalmologic evaluation showed no evidence of exophthalmos or ocular dryness. There was no melanoderma, suggesting against primary adrenal insufficiency. The thyroid gland was palpable and slightly enlarged. Hormonal evaluation revealed corticotropic insufficiency, with low serum cortisol (5.88 µg/dL), and central hypothyroidism, with an inappropriately low TSH (0.187 µIU/mL) in the context of low free T4 (8 pmol/L), reflecting global impairment of the hypothalamic-pituitary axis. Gonadotropic evaluation was consistent with menopause (FSH:64IU/L; LH:15 IU/L). Prolactin levels were normal (5.08 ng/mL). Renal function was preserved, with an estimated glomerular filtration rate of 100 mL/min. Serum calcium was within normal range (92.63 mg/L), and HbA1c was 4.9%. Urine osmolality was markedly low (113 mOsm/L) in the presence of normal to slightly elevated natremia, with a complete absence of compensatory thirst, confirming arginine vasopressin deficiency consistent with adipsic diabetes insipidus.

Pituitary MRI demonstrated a suprasellar arachnoid cyst compressing the pituitary parenchyma to a maximal residual thickness of 2.4 mm, with associated enlargement of the sella turcica (Figure 1). Combined management with desmopressin for adipsic diabetes insipidus, levothyroxine for ventral hypothyroidism, and hydrocortisone for corticotropic insufficiency resulted in significant clinical improvement, including a marked reduction in polyuria and an overall improvement in the patient's general condition.

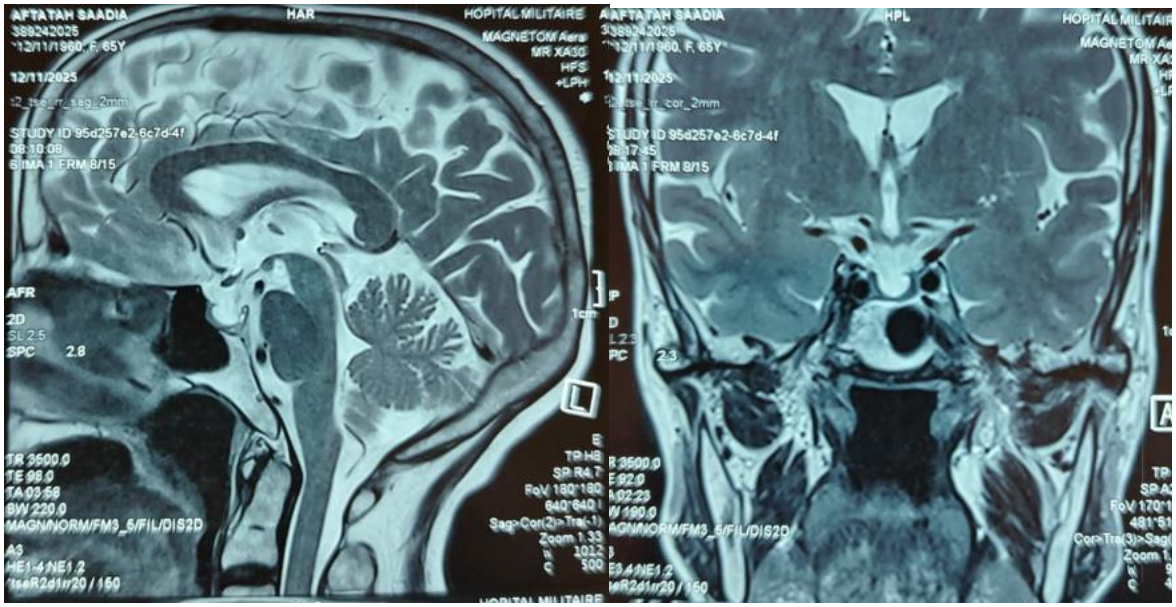

Figure 1: Suprasellar arachnoid cyst

DISCUSSION

Adipsic diabetes insipidus is a rare form of central diabetes insipidus characterized by persistent polyuria associated with an absence or reduction of thirst sensation. This disorder results from combined impairment of the hypothalamic thirst circuitry, encompassing the organum vasculosum of the lamina terminalis (OVLT), the subfornical organ, and the preoptic nuclei, and the neurohypophysis, which is responsible for arginine vasopressin secretion (Wang *et al*., 2023)(*Williams Textbook of Endocrinology - 13th Edition | Elsevier Shop*, n.d.).

Under normal conditions, thirst perception and arginine vasopressin secretion are triggered by osmosensitive neurons responsive to rising plasma osmolarity, ensuring coordinated water balance. In adipsic diabetes insipidus, a hypothalamic lesion such as

a suprasellar arachnoid cyst disrupts these centers simultaneously, abolishing the perception of thirst despite progressive hyperosmolarity. In the absence of compensatory thirst mechanisms, patients are at high risk of severe dehydration and life-threatening hypernatremia, unlike a typical diabetes insipidus, where polydipsia helps limit these complications. This lack of compensatory mechanisms explains why patients develop marked polyuria and are exposed to severe hypernatremia, even with limited fluid intake. Initial symptoms may be subtle, including fatigue, headaches, cognitive blurring, or sleep disturbances, which can delay diagnosis.(Dalan *et al*., 2019)(Eisenberg & Frohman, 2016; Kothari *et al*., 2021b)

The most common causes of adipsic diabetes insipidus are hypothalamic or hypothalamic-pituitary lesions, including tumors, trauma, infections, or congenital abnormalities(Kothari *et al*., 2021c). In our case, a suprasellar arachnoid cyst was identified as the underlying etiology. Although often asymptomatic, such cysts may lead to significant endocrine dysfunction when they expand the sella turcica and compress the pituitary parenchyma, as observed in our patient. Pituitary MRI revealed an arachnoid cyst compressing the pituitary gland, with a residual maximal thickness of 2.4 mm and enlargement of the sella turcica, providing a clear anatomical substrate for the combined hypothalamic-pituitary dysfunction.

Adipsic diabetes insipidus carries a broad spectrum of potentially serious complications, largely attributable to the inability to mount an appropriate thirst response and the resulting impairment of autonomous fluid regulation. Dysnatremia is the most frequent complication, with hypernatremia predominating, occurring in nearly one-fifth of outpatient measurements and exceeding one-quarter during hospitalization. Beyond electrolyte disturbances, hypernatremic dehydration promotes hemoconcentration-induced hypercoagulability, with venous thromboembolism reported in approximately one-third of patients, justifying prophylactic anticoagulation in admitted patients with hypernatremia and elevated hematocrit. Given the frequent hypothalamic-pituitary etiology, anterior hypopituitarism commonly coexists and must be systematically corrected. Neurological complications, including seizures and sleep-disordered breathing, are also prevalent; the latter is of particular prognostic significance, as respiratory failure in the context of sleep apnea accounted for many premature deaths, underscoring the importance of routine screening and active treatment. Rhabdomyolysis and thermoregulatory dysfunction, although less frequent, represent additional sources of morbidity. Collectively, these complications underscore the necessity of structured multidisciplinary follow-up for optimal long-term management of adipsic diabetes insipidus(Cuesta *et al*., 2017). Management of adipsic diabetes insipidus relies on several complementary measures. Arginine vasopressin replacement with desmopressin is essential to control polyuria and prevent dehydration. Fluid intake must be strictly prescribed based on body weight or plasma osmolality targets rather than thirst sensation, as patients are unable to self-regulate their water intake, which renders them particularly vulnerable to both hypernatremia and iatrogenic hyponatremia. It is also crucial to correct associated hormonal deficiencies, such as corticotropic and thyrotropic insufficiencies, to restore overall endocrine balance. Regular biochemical monitoring of serum sodium and plasma osmolality is mandatory to guide treatment adjustments and prevent electrolyte imbalances. Finally, when the underlying cause is a compressive lesion such as a symptomatic suprasellar arachnoid cyst, surgical decompression (through endoscopic fenestration or cystoperitoneal shunting), in some cases, radiotherapeutic management may be considered to relieve compression and limit hypothalamic-pituitary damage. This case highlights the importance of early recognition of adipsic diabetes insipidus in patients presenting with polyuria in the absence of polydipsia, and underscores the need for a multidisciplinary approach involving endocrinology, neurosurgery, and radiology for optimal long-term management.

CONCLUSION

Adipsic diabetes insipidus is a rare complication of hypothalamic-pituitary disorders, associated with a high risk of dehydration and electrolyte imbalance due to the absence of compensatory thirst. The present case illustrates that a suprasellar arachnoid cyst, although often asymptomatic, can lead to secondary adipsic diabetes insipidus when it compresses the pituitary parenchyma and enlarges the sella turcica. This case highlights the importance of early diagnosis based on clinical, biological, and imaging findings, as well as the need for a multidisciplinary approach combining hormonal replacement therapy, tailored hydration, and, when appropriate, treatment of the underlying lesion. Clinical vigilance in cases of polyuria without polydipsia is essential to prevent serious complications and improve patient outcomes.

COMPLIANCE WITH ETHICAL STANDARDS

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Disclosure of Conflict of Interest: The authors declare no conflict of interest.

Statement of Ethical Approval: The present research work does not contain any studies performed on animals/human's subject by any of the authors.

Statement of Informed Consent: Informed consent was obtained from all individual participants included in the study.

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