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Case Report

Multidisciplinary approach to a diabetes insipidus patient with concomitant conditions: a case report

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ABSTRACT

This case report details the clinical presentation, diagnostic journey, and management of a middle-aged female, aged 52, who sought medical attention in February 2025 for excessive thirst, frequent urination with excretion of large volumes of urine, and weakness. Laboratory findings and investigations revealed few underlying pathologies, which were subsequently consulted by multiple specialists, including neurologists, ophthalmologists, and internists. The final diagnosis of complete central diabetes insipidus was attributed to the results from water deprivation tests followed by a desmopressin test. The patient was discharged with favourable results from the treatment plan, with directives to consume fluids only when thirsty, adhere to the treatment plan, and follow up with scheduled consultations and repeat laboratory test results. This case highlights the importance of a multidisciplinary approach to tailored diagnostic methods and the necessity of evaluating differential diagnoses before treatment in the context of a unique condition.

Keywords: Diabetes insipidus, Desmopressin test, Water deprivation test, Differential diagnosis, Concomitant conditions

INTRODUCTION

Diabetes insipidus is a rare clinical syndrome.[1,2] The prevalence of this condition is 1:25,000, affecting males and females equally, regardless of age.[3] Polyuria-polydipsia syndrome, which is excessive thirst (polydipsia) and excretion of large volumes of diluted urine (polyuria), is one of the main clinical features caused by Diabetes Insipidus.[4] Two main types can be identified in this condition according to the cause: central and nephrogenic forms.[5]

Nephrogenic diabetes insipidus (NDI) is defined by the resistance of terminal distal convoluted tubules and collecting ducts to antidiuretic hormone (ADH).[3] Central diabetes insipidus (CDI) is characterized by impaired production and or secretion of arginine vasopressin (AVP), resulting from the loss or impaired function of vasopressinergic neurons in the hypothalamus posterior pituitary, due to multiple etiologies.[1] While treatment varies according to the differential diagnosis, as the pathophysiological mechanisms of each possible diagnosis are different from each other, apart from the clinical features, urine and serum osmolality, and water deprivation test hold significance in conclusive diagnosis.[6]

This case report explores clinical presentation, investigative pathways with laboratory tests and findings, an approach to excluding differential diagnosis, and therapeutic interventions for a patient diagnosed with central diabetes insipidus, secondary to intracranial pathology with some other concomitant conditions.

CASE REPORT

In February 2025, a 52-year-old woman presented with complaints of weakness, extreme thirst described as drinking up to 7-8 liters of water per day, frequent urination described as urinating every 40 minutes a day with approximately eight liters of urine, and dry skin. In the anamnesis, it was revealed that the symptoms became increasingly disturbing after the death of the mother, for 9 months, after suffering stress. An approximate 10 kg of weight loss in the period of 8 months after the initiation of the above symptoms was also revealed.

Table 1: Laboratory tests with timeline.

Date	Laboratory test	Parameter	Result	Reference range
10.01.2025	Glycosylated hemoglobin	HbA1c	5.4%	4.0-5.6% (normal), <6.5% (non-diabetic)
14.01.2025	Thyroid function tests	Free T4	16.9 pmol/l	9- 22 pmol/l
		TSH	1.8 mIU/ml	0.4- 4.2 mIU/ml
		Anti-TPO	5.9 mIU/ml	0-30 mIU/ml
13.02.2025	General urinalysis	Specific gravity 1000, Ph:7, Transparent, Nitrites negative.		Specific gravity: 1015-25
13.02.2025	Complete blood count (CBC)	WBC	8.69x10^9/l	4-9x10^9/l
		RBC	5.6 x 10^{12}/l	Female: 3.7-4.9 x 10^{12}/l
		HGB	152 g/l	Female: 120-160 g/l
		HCT	46.4%	Female: 32- 47%
		PLT	226x 10^9/l	150-450 x10^9/l
		MCH	27.1	28- 32
13.02.2025	Biochemical blood test	Total protein	73 g/l	65-85 g/l
		Cholesterol	6.0 mmol/l	3.12-5.2 mmol/L
		LDL	3.5 mmol/l	<2.59 mmol/l
		Triglyceride	2.1 mmol/l	<=1.52 mmol/l
		Creatinine	71 µmol/l	53-97µmol/l
		Urea	4.0 mmol/l	1.7-8.3 mmol/l
13.02.2025	Biochemical urine test	Potassium	11.92	25-125 mmol/day
		Sodium	13.87	40-220 mmol/day
		Chloride	14.37	110-250 mmol/day

Table 2: Investigations with timeline.

Date	Investigation	Findings
28.01.2025	MRI brain	Meningioma in the right parietal region, calcifications.
30.01.2025	Abdominal ultrasound	No pathologies were detected. No structural or size abnormalities.
03.02.2025	Neurologist consultation	Recommended neurosurgical evaluation, EEG, and ophthalmologic exam.
05.02.2025	MSCT brain	Hypodense focus on subcortical nuclei, advised MRI for confirmation.
13.02.2025	EEG	Desynchronization of bioelectric activity, no epileptiform waves detected.
17.02.2025	Ophthalmologist consultation	Normal visual function, no pathology detected.
19.02.2025	Water deprivation test and desmopressin test	(results discussed in a more detailed manner under case presentation)
20.02.2025	Consultation - internal medicine	Confirmed diabetes insipidus, recommended desmopressin therapy.
24.02.2025	Colonoscopy	A submucosal lipoma in the cecum dome was found. Polyp excised.

The patient denied any history of tuberculosis or viral hepatitis, any contact with infectious individuals, or recent travel history. A meticulous inquiry into her medical history revealed she had been taking phenibut (an antidepressant) after the stress caused by the loss of her mother, which is now replaced by prescribing Grandaxin. Furthermore, stage 2 arterial hypertension with a complete block of the left leg of His bundle in the ECG, and surgery for extirpation of the uterus with appendages were revealed. Hereditary history disclosed glioblastoma in the father, Alzheimer's disease, and high blood pressure in the mother. Allergies, transfusions, or indications of intestinal infection within the preceding 10 days were denied.

The patient's condition upon admission was satisfactory; the patient was conscious and had a clear, calm mental status. Weight and height upon admission were 105 kg and 177 cm, respectively. Stable blood pressure of 120/70 mmHg, and a 36.5 °C temperature was measured. The pulse rate and respiratory rate were 78 bpm and 17 breaths per minute, respectively. No edema was found, and the skin and mucosa were found to be normal. No murmurs or abnormal heart sounds were detected while the pulse was rhythmic. The respiratory system was assessed and clear for abnormal lung sounds and wheezing. Inspection of the abdomen revealed soft, non-tender palpation and normal intestinal motility.

As shown in Tables 1 and 2, multiple laboratory tests and investigations were conducted to identify the root cause of the symptoms. These revealed multiple underlying conditions that led to the final diagnosis. For the conclusion of the final diagnosis, the desmopressin test and the water deprivation test were crucial, as the indirect water deprivation test is considered the gold standard for diagnosing diabetes insipidus. The patient was first restricted from fluids under close medical supervision for the water deprivation test. Here, the body weight, urine volume, urine osmolality, and plasma osmolality were measured at regular intervals.

During the test, the patient was not allowed to drink anything, and it can also be advisable to limit food intake. Tests were conducted during the first 8 hours. However, feeding food will not preserve much of the necessary water and easily digestible carbohydrates. Boiled eggs, bread, lean grain-based meats, and fish were recommended. The test was meant to stop when either one of the following conditions was met:

Loss of more than 5% of body weight

Unbearable thirst

The patient's condition is objectively severe

An increase in blood sodium and osmolality above the normal limits

An increase in urine osmolality above 600 mOsm/kg

Subsequently, the desmopressin test was performed for further clarification of the differential diagnosis on the next day. After measuring baseline measurements of urine volume, urine osmolality, and plasma osmolality, desmopressin (DDAVP) was administered orally at 0,2 mg. Urine output and urine osmolality were monitored over the next 2-4 hours. The desmopressin test was helpful in the differential diagnosis.

On 19 February 2025 water deprivation test was conducted. The test results are as follows.

The general urine analyses showed straw-yellow colored, transparent urine in both samples. The first sample demonstrated a low relative density of 1003 (1018-30) and a urine osmolality of 99.9 mOsm/kg. The second sample had a relative density of 1002 (1018-30) with a urine osmolality of 66.6 mOsm/kg. Both analyses were otherwise unremarkable (not clinically significant), showing no protein, glucose, oxalates, mucus, or bacteria, with only a single squamous epithelial cell, a single red blood cell, and a single leukocyte detected in each sample. Biochemical blood tests were performed at 3 different times of the day. The first sample revealed blood osmolality of 306.3 mOsm/kg (275-95 mOsm/kg), the second of 314.5 mOsm/kg (275-95 mOsm/kg), and the third of 298.4 mOsm/kg (275-95 mOsm/kg). Other parameters like total protein, urea, sodium, potassium, and chlorides were within the normal range in all three samples except for a blood glucose rise in the second sample of 7.4 mmol/L (3.5-6.2 mmol/L).

In view of persistent polyuria during water deprivation, a gradual decrease in blood osmolality with fluid restriction, and urine osmolality remaining less than 300 mOsm/kg during the test, diabetes insipidus was confirmed as the main diagnosis.

On 20 February 2025. water deprivation test followed by a desmopressin test was conducted, and the results are given below.

The first general urine analysis presented straw-yellow colored, transparent urine with a relative density of 1003 (1018-30) and a urine osmolality of 99.9 mOsm/kg. No protein, glucose, oxalates, mucus, or bacteria were detected in it, and only a single squamous epithelial cell, a single red blood cell, and a single leukocyte were detected.

Later, after administration of desmopressin, another general urinalysis was conducted, which revealed the same results except for relative density of 1010 (1018-30) and urine osmolality of 333 mOsm/kg.

Considering the urine osmolality changes of these tests, the diagnosis was further clarified as given below. Here, urine osmolality being elevated with an increase of >50% was a key feature in attaining the final diagnosis of which type of diabetes insipidus.

Diagnosis

The final diagnosis of complete CDI was concluded with laboratory and clinical findings. Comorbidities diagnosed were meningioma of the right parietal region, non-toxic nodular goiter, kidney cysts, submucosal lipoma of the cecum dome, and hypertension.

Treatment

The patient received hospital care, including diagnostic assessments and treatment, from February 12, 2025, to February 25, 2025. With the confirmed diagnosis, a pharmacological management plan was executed. Desmopressin (0.1 mg, twice daily, 30 minutes before meals for 3 months, with dose adjustment as needed) was the main drug prescribed to control polyuria by enhancing renal water reabsorption. Lisinopril (10 mg, twice daily) was administered for blood pressure stabilization. Grandaxin (50 mg, twice daily for one month) was provided for stress management following the patient's emotional distress situation. The patient was advised to drink only when she felt thirsty and to avoid excessive fluid intake to prevent overhydration. It was taken into consideration to monitor the daily fluid intake and urine output to assess the efficacy of the treatment. Serum electrolytes, blood glucose, creatinine, and sodium levels were also periodically evaluated. Regular blood and urine tests (osmolality tests) were performed to adjust medication dosage accordingly.

The patient was discharged on 25 February 2025, with significant improvement in the patient's condition after being able to reduce the diuresis to manageable levels. It is to be noted that with the treatment plan specifically following desmopressin therapy, diuresis was reduced from 8.5 L/day to 4.5 L/day. Body weight and the frequency of urination at night were monitored. Serum sodium levels were stabilized between 140-47 mmol/l. The patient's complaints, like severe thirst perception, were controlled, and the fluid balance was improved. Observations by an endocrinologist and a neurologist were conducted as treatment recommendations. The patient was discharged with conditions of strict fluid intake regulation, continuation of desmopressin and lisinopril as prescribed, self-monitoring of diuresis to evaluate disease progression, and follow-up care. Neurological consultation with an MRI after six months to monitor the progression of the meningioma was also recommended. Also, in follow-up care, a general urinalysis and biochemical blood test of sodium, potassium, glucose, and creatinine are specifically recommended to be taken within a 6-months. After receiving the results of the histopathological examination, it was recommended to consult a gastroenterologist.

DISCUSSION

This case describes the unique presentation of a 52-year-old female patient with main complaints of persistent polyuria and polydipsia, which were ultimately diagnosed as CDI with concomitant diseases. Though the patient presented typical symptoms of diabetes, several investigations needed to be conducted to exclude all the differential diagnoses of the complaints. Systemic evaluation was undertaken to rule out such alternative causes, which revealed other underlying concomitant conditions. Below is the list of each considered condition, carefully discussed with how it was excluded.

For a patient with polyuria and polydipsia, diagnoses can include diabetes insipidus (central DI, nephrogenic DI, gestational DI, dipsogenic DI), solute diuresis (uncontrolled diabetes mellitus, high sodium administration, mannitol, radiocontrast dye, salt-wasting nephropathies), and an appropriate diuretic response.[3-7]

Central diabetes insipidus

This is characterized by inadequate ADH synthesis or secretion from the posterior pituitary, leading to impaired renal water absorption.[3] It can be idiopathic or secondary to intracranial pathology such as traumatic brain injuries, infections, loss of blood from the posterior pituitary or hypothalamus, neurosurgery, and tumors.[8]

Reasons that helped in confirming the diagnosis of CDI were as follows. Patient's complaints about polyuria (urine output exceeding 3 L/day) and polydipsia (fluid intake exceeding 3 L/day), and symptoms of dehydration and hypernatremia: weakness, dry skin. The key factor was the results from the water deprivation test and desmopressin test. Here, during the fluid restriction period, urine osmolality remained below 300 mOsm/kg which confirmed diabetes insipidus, and with the injection of desmopressin (exogenous synthetic AVP), urine osmolality elevated with an increase of >50%, which then confirmed the diagnosis of complete CDI.[3] MRI findings of right parietal meningioma, suggesting possible disruption to the function of the hypothalamic-pituitary axis, can also be considered as a confirming factor. A good prognosis with a favorable outcome of the condition after treatment further proved the accuracy of the diagnosis.

Nephrogenic diabetes insipidus

Nephrogenic DI is caused by a defect in the terminal distal convoluted tubule and collecting duct, which leads to insensitivity to the antidiuretic effect of physiological ADH levels, resulting in dilute urine.[8] No history of nephrotoxic drug use (e.g., Lithium), normal serum calcium and potassium levels, and absence of abnormalities suggesting congenital NDI or nephrogenic pathology were some of the factors that contributed to excluding nephrogenic DI from this patient's diagnosis. Results from the Fluid restriction test followed by the Desmopressin test were again the key factor in the exclusion of this pathology. If the increase in urine osmolality after injection of desmopressin during the tests was less than 50%, the diagnosis could be complete nephrogenic DI.[3]

Primary polydipsia

Primary polydipsia, also known as dipsogenic DI, has an abnormally low thirst threshold, which leads to increased fluid intake, resulting in excretion of diluted urine exceeding 40-50 ml/kg body weight and risk of hyponatremia.[8] This condition is common in patients with psychotic or neurodevelopmental disorders. While patients with primary polydipsia usually have uninterrupted sleep at night, they wake up at night mostly to drink water rather than to pass urine, which was the opposite for this patient. Even though the normal sodium levels align with this differential diagnosis, the results from the stimulation test conducted confirmed that the diagnosis proved otherwise. During the water deprivation test, when urine osmolality increases between 300 and 800 mOsm/kg, the underlying cause is primary polydipsia, which was not the case for this patient. Furthermore, improvement in urine osmolality of the patient after the desmopressin administration, with no psychiatric or behavioral evidence of compulsive drinking, excludes this differential diagnosis.

Gestational diabetes insipidus

This is caused by an exaggerated concentration of placental vasopressinase during pregnancy, which degrades maternal AVP, resulting in dilute polyuria.[8] As the patient is not pregnant and has undergone uterine extirpation previously, this differential diagnosis was dismissed, as it is physiologically unlikely to happen.

Solute diuresis

In general, this involves several conditions that result in osmotic diuresis, mainly due to increased solute load in the renal tubules. Uncontrolled diabetes mellitus was excluded due to normal values in the fasting glucose levels, HbA1c, and absence of glucosuria on repeated urine analyses. No history of sodium supplement or hypertonic saline usage was found, while the dietary intake of sodium and laboratory testing were reported to be normal, excluding high sodium administration. Mannitol and radio-contrast dye exposure were not recorded. Moreover, renal tubular disorders were not suspected, as serum electrolytes and renal function tests were normal with no proteinuria and no history of nephrotoxic exposure or genetic renal disease.

Appropriate diuretic response

Polyuria can be caused by the use of pharmacological agents like diuretics. This patient does not have any history of using such medicine prior to symptom onset.

CONCLUSION

This case report underscores the atypical presentation of CDI in a middle-aged female, manifested with underlying concomitant diseases. The diagnostic journey, which outweighed different diagnoses typical for presenting symptoms of polyuria and polydipsia, finally revealed complete CDI. In the attribution of the final diagnosis, the fluid restriction test and desmopressin test were key investigations. The patient managed comprehensively with desmopressin and demonstrated a favorable response, highlighting the efficacy of this diagnostic approach. This case report adds valuable insights to the literature, emphasizing the significance of evaluating differential diagnoses and establishing the final diagnosis using the standard diagnostic method, even with underlying concomitant conditions. Ultimately, it reinforces the importance of continuous education for healthcare providers in recognizing and managing a patient with diabetes insipidus.

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