

# A Case of Psychogenic Polydipsia in an Elderly: An Overlooked Diagnosis

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## ABSTRACT

Primary polydipsia, or psychogenic polydipsia, is characterised by excessive fluid intake without an underlying physiological cause. If left untreated, primary polydipsia may result in life-threatening hyponatremia and can lead to complications such as nausea, vomiting, confusion and seizure episodes, potentially becoming life-threatening if not recognised and managed early. The aetiology of this condition is incompletely understood and is frequently attributed to psychiatric disorders, most commonly chronic schizophrenia. Psychogenic polydipsia occurs in up to 20% of psychiatric patients, and this case serves as a reminder to be cognisant of water overconsumption. This case report presents a 74-year-old male with a three-year history of excessive thirst and polyuria, which worsened following the death of his elder brother. Laboratory investigations showed normal random blood sugar and HbA1c levels, normal renal and liver function tests and mild hyponatremia, while radiological investigations were grossly normal. Ultimately, a water deprivation test ruled out diabetes insipidus, leading to a diagnosis of primary polydipsia. The patient was started on oral medication and psychotherapy after diagnosis, and he improved significantly with treatment. He was discharged on oral atypical antipsychotics, with water restriction and regular psychotherapy sessions for further improvement. This case highlights the importance of a thorough diagnostic evaluation of excessive thirst in elderly patients and the necessity of initiating appropriate treatment, which can prevent complications such as severe dilutional hyponatremia in this population.

**Keywords:** Chronic polydipsia, Polyuria, Water deprivation test

## CASE REPORT

A 74-year-old male, retired, non alcoholic and non smoker, was admitted to the hospital with complaints of excessive thirst and dryness of the mouth and throat, which was relieved by water intake for the past three years. These symptoms were associated with an increased frequency and urgency of micturition without urinary incontinence. The patient's relatives reported that after the death of the patient's elder brother six months ago, he experienced a worsening of these symptoms, accompanied by low mood, decreased social interaction, decreased interest in performing daily activities and fragmented night-time sleep.

The patient had a known history of hypertension, ischaemic heart disease (following percutaneous transluminal coronary angioplasty to the right coronary artery three years ago) and benign prostatic hypertrophy, along with a past history of haemorrhoidectomy performed 10 years ago. He had made multiple visits to a dental hospital for dental caries in the past year. His younger brother was diagnosed with major depressive disorder at the age of 35 and had been on medication for the past 30 years. There was no family history of diabetes mellitus. The patient was on the following regular medications: Telmisartan 40 mg, Ecospirin 75 mg, and Atorvastatin 20 mg. He had visited another hospital three months prior for his current symptoms, where he was clinically diagnosed with benign prostatic hypertrophy and started on Tamsulosin 0.4 mg.

On admission, the patient's vital signs were as follows: pulse rate was 80 beats per minute, systolic blood pressure was 120 mmHg, diastolic blood pressure was 70 mmHg, respiratory rate was 20 breaths per minute, and oxygen saturation on room air was 98%. The initial laboratory investigations showed a slightly raised total leukocyte count, with normal haemoglobin and platelet levels, mild hyponatraemia, and normal random blood sugar levels, with an HbA1c of 5.4%. Renal and liver function tests, as well as urine routine microscopy, were normal. Laboratory investigations are mentioned below in [Table/Fig-1].

The patient's water intake and urine output were monitored for the next 48 hours. His total water intake was 4.2 litres on day one and 4.3 litres on day two, with a urine output of 4.5 litres per day on both

Investigation	Result	Normal range
Haemoglobin (g%)	13.5	13.2-16.6
Total leukocyte count (cells/mm <sup>3</sup> )	12200	4000-10000
Platelet count (lacs)	249000	1.5-4
Serum sodium (mmol/L)	131	136-145
Potassium (mmol/L)	4.44	3.5-5.1
Chloride (mmol/L)	99	98-107
Random blood sugar (mg/dL)	102	<200
HbA1c (%)	5.4	<5.7
Liver function tests	Normal	Total bilirubin=0.2-1.2 mg/dL Conjugated bilirubin=<0.5 mg/dL Unconjugated bilirubin=0.1-1 mg/dL
Renal function tests	Normal	Urea=17-49 mg/dL Creatinine= 0.6-1.3 mg/dL
Serum prostate specific antigen	Normal	< 4 ng/mL
Arterial blood gas on room air	Normal	-
Urine routine microscopy	Normal	-

[Table/Fig-1]: Initial laboratory investigations.

days. Thus, the patient exhibited polydipsia and polyuria. Further evaluation revealed the patient's urine sodium was 37 mmol/L, urine potassium was 16.4 mmol/L, and urine chloride was 34 mmol/L, with both urine and serum osmolality remaining normal. Investigations are provided below in [Table/Fig-2] [1,2].

Investigations	Result	Normal range [1,2]
Urine sodium (mmol/L)	37	>20
Urine potassium (mmol/L)	16.4	20-125
Urine chloride (mmol/L)	34	25-260
Urine osmolality (mOsm/Kg)	304	50-1200
Serum osmolality (mOsm/Kg)	275	275-295
Serum cortisol (8 am and 4 pm) (µg/dL)	10.10 and 5.4	5-23 at 8 am 3-13 at 4 pm

[Table/Fig-2]: Shows further investigations done in the patient [1,2].

An Magnetic Resonance Imaging (MRI) of the brain was normal, and a 2D echocardiogram was also normal. Based on the laboratory investigations and neurological imaging, central and nephrogenic diabetes insipidus were ruled out. A water deprivation test was performed to confirm the diagnosis of primary polydipsia. [Table/Fig-3] presents the results of the water deprivation test conducted at various time points (9:00 pm, 12:00 am, 4:00 am, and 10:00 am) to evaluate the patient's ability to concentrate urine and to help differentiate psychogenic polydipsia from other potential causes, such as diabetes insipidus [3,4]. The serum osmolality was stable throughout the test, and there was a gradual increase in urine osmolality, suggesting that the kidneys were capable of concentrating urine, consistent with psychogenic polydipsia rather than diabetes insipidus.

Water deprivation test	9:00 pm	12:00 am	4:00 am	10:00 am
Serum osmolality	278.7	277.7	277.3	273.1
Urine osmolality	304.4	312.4	368.8	407
Urine Na/K/Cl	55/19.4/65	56/9.6/62	108/15.7/119	113/49.7/136
Serum Na/K/Cl	131/4.13/98	132/4.19/98	133/4.21/100	133/4.14/100

[Table/Fig-3]: Shows the results of water deprivation test done at various time points.

After the initial blood, urine and radiological investigations, as well as the water deprivation test, the patient was diagnosed with primary polydipsia. He was started on atypical antipsychotics-Olanzapine and Risperidone. He is also undergoing behavioural therapy and psychotherapy sessions. Currently, the patient has experienced significant symptomatic improvement and better control of his condition.

DISCUSSION

Primary polydipsia, also known as psychogenic polydipsia, is a condition characterised by excessive thirst and excessive fluid intake without an underlying physiological cause [5,6]. It differs from secondary polydipsia, where the excessive thirst results from an underlying medical condition or medication. The precise cause of primary polydipsia remains unclear. Conditions such as schizophrenia and other psychiatric disorders frequently feature primary polydipsia as a symptom [7].

Patients typically present with symptoms of excessive thirst and an insatiable desire to drink large quantities of fluids. This excessive fluid intake can lead to frequent urination (polyuria). Primary polydipsia does not result in significant electrolyte imbalances or dehydration if the intake is well-regulated. However, the overconsumption of fluids can occasionally lead to dilutional hyponatremia (low sodium levels due to excessive fluid intake), which can have serious consequences. Patients may present with vomiting, blurred vision, dizziness, ataxia, tremors, confusion, lethargy and seizures due to dyselectrolytaemia [8,9].

The diagnosis of primary polydipsia is made through a detailed medical history, physical and psychiatric evaluation and laboratory tests to assess for possible secondary causes such as diabetes mellitus or diabetes insipidus [10]. The definitive diagnosis is made by the water deprivation test, which helps differentiate primary polydipsia from central diabetes insipidus and nephrogenic diabetes insipidus. This is a standard physiological test of vasopressin secretion wherein the patient's water intake is restricted overnight, with serial measurements of plasma and urine osmolality, urine volumes and body weight. In primary polydipsia, restricting fluid intake leads to normal or improved urine concentration, whereas in diabetes insipidus, urine remains dilute despite dehydration [Table/Fig-4] [11].

Treatment strategies for psychogenic polydipsia include behavioural therapy, psychiatric treatment with psychotherapy, education

	Normal values	Central diabetes insipidus	Nephrogenic diabetes insipidus	Primary polydipsia
Baseline urine osmolality	>300	<300	<300	300-800
Urine osmolality after water deprivation	800-1200	<300	<300	300-800
Plasma osmolality after water deprivation	Normal	Increases	Increases	Normal
Serum sodium	Normal	May increase (>145)	May increase (145)	Normal
Urine osmolality after administration of desmopressin	-	Increases >50%	Does not increase	-

[Table/Fig-4]: Shows water deprivation test and its normal interpretation.

regarding the risks of excessive fluid intake and fluid restriction to manage excessive intake. In pharmacological management, atypical antipsychotics have been reported to improve symptoms. Clozapine has shown benefit in case reports and prospective trials. Risperidone and olanzapine have also improved polydipsia in case reports, although prospective double-blind studies have not demonstrated any benefit with olanzapine. If not treated adequately, psychogenic polydipsia can lead to significant morbidity and mortality, affecting 6-20% of psychiatric patients [12].

Water Deprivation Test and Its Interpretation [11]

A case report published by Gill M and McCauley M discussed a 43-year-old man who was a known case of bipolar disorder and alcohol dependence. He was admitted with complaints of consuming copious quantities of water and frequent micturition. Initially, his sodium levels were 128 mmol/L, and a Computed Tomography (CT) scan showed no abnormalities. The patient experienced a seizure episode and his condition continued to deteriorate. His repeat sodium levels were 108 mmol/L. He was transferred to the Intensive Care Unit (ICU) following a psychiatric assessment, where he was placed on strict fluid restriction, behavioural therapy and antipsychotics. The patient improved drastically with treatment [13].

Another case report detailed a 25-year-old divorcee who presented with complaints of excessive water intake (12-25 litres per day), occasional urge incontinence and anxiety, which had gradually increased over the past three and a half years. She developed these complaints six months after her separation. There was no history of any psychiatric disease and no significant family history. She reported a marked decrease in appetite and sleep. On admission, her serum sodium was 138 mmol/L, blood glucose levels were normal and the remaining investigations were unremarkable. No diurnal body weight gain was noted. The water restriction test was performed, and the patient was diagnosed with psychogenic polydipsia. She was placed on strict fluid restriction and started treatment with benzodiazepines, antidepressants and supportive psychotherapy. The patient's condition improved drastically following the initiation of treatment [14].

Similar to the above case reports, this patient presented with complaints of excessive water intake, frequent micturition, mild hyponatremia and normal radiological investigations. The water deprivation test confirmed the diagnosis of psychogenic polydipsia. Subsequently, the patient was placed on strict fluid restriction, began psychotherapy and was prescribed oral atypical antipsychotics, which resulted in an improvement of symptoms.

CONCLUSION(S)

Psychogenic polydipsia is a rare yet significant clinical entity, particularly in the elderly, where it can be easily overlooked. This case highlights the importance of maintaining a high suspicion for primary polydipsia in elderly patients presenting with chronic excessive thirst

and polyuria, especially in the context of psychological stress or recent emotional trauma. A systematic approach involving a detailed history, clinical examination, appropriate laboratory work-up and a water deprivation test-the gold standard-can help differentiate psychogenic polydipsia from more common causes such as diabetes mellitus or diabetes insipidus.

Timely psychiatric referral, behavioural therapy and appropriate pharmacological management can lead to significant clinical improvement. Physicians should educate patients and caregivers about the risks of excessive water intake and ensure regular follow-up.

REFERENCES

[1]

Marks BE. Initial evaluation of polydipsia and polyuria. *Endocrine Conditions in Pediatrics: A Practical Guide*. 2021:107-11.

[2]

Reddi AS. Interpretation of urine electrolytes and osmolality. In *Fluid, Electrolyte and Acid-Base Disorders: Clinical Evaluation and Management* 2023 May 10 (pp. 15-23). Cham: Springer International Publishing.

[3]

Rowe M, Patel N, Jeffery J, Flanagan D. Use of copeptin in interpretation of the water deprivation test. *Endocrinol Diabetes Metab*. 2023;6(3):e399.

[4]

Akkara Y, Narula K, Lazarus K, Papadopoulou D, Choudhury S, Martin N, et al. Redefining diagnostic cut-offs for the indirect water deprivation test. *Clin Endocrinol*. 2025;102(2):149-55.

[5]

Perestrelo J, Teixeira B. Psychogenic polydipsia and hyponatremia—A side effect of psychosis: A review with a case report. *Jornal Brasileiro de Psiquiatria*. 2016;65(3):300-03.

[6]

Tournikioti K, Voumvourakis K, Moussas G, Plachouras D, Michopoulos I, Douzenis A, et al. Primary polydipsia: A case report. *J Nerv Ment Dis*. 2013;201(8):709-11.

[7]

Ahmadi L, Goldman MB. Primary polydipsia: Update. *Best Pract Res Clin Endocrinol Metab*. 2020;34(5):101469.

[8]

Flippo C, Alter CA, Stratakis CA. Primary polydipsia. *Diabetes insipidus in children: A pocket guide*. 2021:01-08.

[9]

Dundas B, Harris M, Narasimhan M. Psychogenic polydipsia review: Etiology, differential, and treatment. *Current Psychiatry Reports*. 2007;9(3):236-41.

[10]

Filippatos TD, Makri A, Elisaf MS, Liamis G. Hyponatremia in the elderly: Challenges and solutions. *Clin Interv Aging*. 2017;14:1957-65.

[11]

Sailer CO, Winzeler B, Nigro N, Suter-Widmer I, Arici B, Bally M, et al. Characteristics and outcomes of patients with profound hyponatraemia due to primary polydipsia. *Clin Endocrinol*. 2017;87(5):492-99.

[12]

Datta RR, Singla P, Bhargava S, Manocha A, Kankra M. Psychogenic polydipsia: A diagnostic challenge. *Current Medicine Research and Practice*. 2024;14(1):34-36.

[13]

Gill M, McCauley M. Psychogenic polydipsia: The result, or cause of, deteriorating psychotic symptoms? A case report of the consequences of water intoxication. *Case Rep Psychiatry*. 2015;2015(1):846459.

[14]

Kohli A, Verma S Jr, Sharma A Jr. Psychogenic polydipsia. *Indian J Psychiatry*. 2011;53(2):166-67.

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AUTHOR DECLARATION:

- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. NA

PLAGIARISM CHECKING METHODS: [Jain H et al.]

- Plagiarism X-checker: Mar 06, 2025
- Manual Googling: Apr 17, 2025
- iThenticate Software: May 03, 2025 (10%)

ETYMOLOGY: Author Origin

EMENDATIONS: 7

Date of Submission: Feb 27, 2025

Date of Peer Review: Mar 24, 2025

Date of Acceptance: May 06, 2025

Date of Publishing: Jul 01, 2025