

## Psychogenic polydipsia: A case of water intoxication

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### ABSTRACT

Psychogenic polydipsia (PPD), a clinical disorder characterized by polyuria and polydipsia, is a common occurrence in inpatients with psychiatric disorders. Primary polydipsia (PP) is a disorder that is clinically characterized by excessive thirst accompanied by increased fluid intake and subsequent excessive excretion of urine without an obvious cause. PP in adults is due to psychogenic causes, is a rare condition and may be more prevalent than thought. There is some evidence for pharmacological management of this condition, but nonpharmacological management, starting from psycho-education to behavioural modification therapy involving family members, can be a very effective strategy. This case report describes a 35-year-old male with schizophrenia who presented with confusion, seizures, and severe hyponatremia due to compulsive water consumption. Prompt diagnosis and management, including fluid restriction, careful sodium correction, and psychiatric intervention, led to symptom resolution. This case highlights the importance of early recognition and a multidisciplinary approach in preventing life-threatening complications associated with PPD. Further research is needed to establish standardized treatment guidelines.

**Key words:** Acetazolamide, Olanzapine, Polydipsia, Schizophrenia


Polydipsia, conventionally defined as the excessive intake of liquids of more than 3 L/day, is poorly understood and underdiagnosed among chronic psychiatric patients [1]. Psychogenic polydipsia (PPD) is uncommon and can lead to serious complications such as hyponatremia, seizures, or even death. Case reports help clinicians recognize and manage this condition effectively. About three-quarters of a century has passed since Rowntree in 1923, and others began to discuss fluid dysregulation among chronic psychiatric patients. Since the first case report of water intoxication in a schizophrenic patient [2]. The mechanism of altered water metabolism is poorly understood in this case [3]. The prevalence of PPD varies, with studies estimating that it affects 6–20% of psychiatric inpatients. It is particularly common among individuals with schizophrenia [4]. Polydipsia in psychiatric patients is seen in patients with chronic schizophrenia with long-term hospitalization. Other psychiatric diagnoses have rarely been associated with polydipsia, such as affective disorders, psychosis with onset during childhood, mental retardation, personality disorders, and tension/anxiety. Among psychiatric patients, schizophrenia patients are the most likely to develop PPD. In a cross-sectional survey involving 38 polydipsic psychiatric patients, 80% of them had diagnoses of schizophrenia [1].

The illness generally develops in three phases, beginning with polydipsia and polyuria, followed by hyponatremia (water is retained as the kidneys fail to excrete the excess fluid, resulting in low sodium serum values) and finally water intoxication which may manifest as nausea, vomiting, delirium, ataxia, seizures, and coma, and may even be fatal. Though most commonly seen in patients with chronic schizophrenia, other mental illnesses associated with PPD are affective disorders, psychosis with onset during childhood, mental retardation, personality disorders, and tension/anxiety. The pathogenesis of polydipsia is unclear but may involve hypersensitivity to vasopressin, increased dopamine activity, and defects in osmoregulation [5]. The management of hyponatremia and PPD has proven difficult for providers due to the paucity of definitive treatment options. Fluid restriction is the gold standard for this condition, but it usually fails due to a lack of compliance and strong compulsions to drink liquids because of excess thirst [6]. Many medications have been used to manage this condition, including acetazolamide, olanzapine, and clozapine [7].

Therefore, in this brief report, we discuss the case of a 35-year-old male with schizophrenia and PPD, including possible management strategies.

### CASE REPORT

A 35-year-old male patient was admitted to the hospital with complaints of confusion, mild agitation, dizziness, nausea,

Access this article online	
Received - 13 March 2025 Initial Review - 28 March 2025 Accepted - 10 May 2025	Quick Response code 
DOI: 10.32677/ijcr.v11i6.5094	

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excessive water consumption (8–10 L/day), and frequent urination for 1 week. He had no fever, abdominal pain, vomiting, diarrhea, or headache. His appetite remained constant, and no weight changes occurred in the previous months. His past medical history was diagnosed with Schizophrenia 10 years ago, and he is currently on olanzapine. He had a previous psychiatric hospitalization. There was no significant family history of endocrine or renal disorders.

On examination, the mental and hydration status were normal. The blood pressure was 110/76 mmHg, and the pulse rate was 70 beats/min; mild tachycardia was present (heart rate: 98 bpm), and the temperature was normal at 37.2°C. The height was 170 cm (percentile 25) and the weight was 70 kg.

Serum sodium level was 118 mmol/L (severe hyponatremia), and serum osmolality was low (Table 1). Basal hormonal investigations, including adrenocorticotrophic hormone, thyroid-stimulating hormone, thyroxine, insulin-like growth factor, cortisol, and prolactin, were normal. Brain magnetic resonance imaging (normal posterior pituitary and pituitary stalk) and kidney ultrasonography were normal.

## DISCUSSION

PPD is a complicated and frequently overlooked disorder that is more common in people with schizophrenia and other mental illnesses. One example of the difficulties in diagnosing and treating PPD in psychiatric settings is the case of a 35-year-old man who had severe hyponatremia, excessive water intake, and schizophrenia. Patients with chronic schizophrenia, particularly those on long-term antipsychotic therapy, frequently experience the illness, which calls for a thorough, multidisciplinary approach to management that incorporates both medical and behavioral interventions.

The excessive water intake observed in PPD might be related to changes in the hypothalamus and ventromedial nucleus, two parts of the brain that control thirst and satiety. In addition, high dopamine activation, which is a hallmark of schizophrenia, may make the compulsive behaviors linked to PPD worse by intensifying thirst and increasing fluid intake. Electrolyte correction was done by slowly correcting hyponatremia with hypertonic saline to prevent osmotic demyelination. Psychiatric management was done by adjustment of the antipsychotic regimen, and initiating cognitive-behavioral therapy. By doing this, the following outcome includes improvement in cognitive function, reduced water intake over 2 weeks, and normalization of serum sodium levels. Initially, the patient was on treatment with vasopressin receptor antagonist (tolvaptan) 30 mg in divided doses, which was tapered off after sodium levels were stabilized by the physician. The patient showed a significant response to the

acetazolamide. He was started on a daily dose of 250 mg. There was an improvement in hyponatremia and polydipsia, as well as water intoxication.

PPD falls under the classification of primary polydipsia and is found in roughly 20% of patients with psychiatric disorders. The relation to schizophrenia is unknown, but statistics show an increased incidence in this population over schizoaffective disorder and bipolar disorder [8]. The various conditions in which PPD can be seen are: Positive symptoms schizophrenic, compulsive behavior, and stress reduction [4]; PPD can also occur in psychiatric patients in order to reduce the effect of anticholinergic drugs [9].

It is still unclear how PPD develops. Dopamine dysregulation, hypersensitivity to vasopressin, and modified osmoregulatory systems are some of the possibilities. In people with schizophrenia, especially those using neuroleptic drugs (antipsychotics), dopamine, which is essential for controlling thirst, may be dysregulated, which can result in excessive drinking. Antipsychotics, particularly those with anticholinergic effects, may exacerbate PPD by activating the brain's thirst center, according to some research [10]. Fluid restriction is the main strategy for PPD management. Although this method works, it is frequently difficult because of compulsive drinking and non-compliance. Behavioral interventions, like supervised drinking and reinforcement schedules, may assist in lowering water intake and the risk of hyponatremia, according to the research [11].

It is also suggested that elevated levels of dopamine may be stimulating the thirst center, or the super sensitivity of the dopamine receptor may be responsible for the same, as it is usually the chronic schizophrenics with long-term intake of neuroleptics presenting with PPD [4]. This case underscores the challenges in managing PPD, where noncompliance with fluid restriction complicates treatment. While pharmacological approaches, including acetazolamide and vasopressin antagonists, show promise, long-term management should focus on behavioral interventions and close psychiatric follow-up. Another important aspect of treatment is electrolyte correction. Osmotic demyelination, a possible consequence of rapid sodium correction, was avoided by using hypertonic saline to gradually restore sodium levels in light of the patient's severe hyponatremia. Current therapeutic guidelines, which stress the necessity of gradually and carefully correcting hyponatremia in patients suffering from water intoxication, are consistent with this strategy [12].

Vasopressin receptor antagonists, like tolvaptan, are pharmacological treatments that may be used to alleviate abnormal thirst and fluid retention. Tolvaptan was started in this instance, but once the patient's salt levels stabilized, it was tapered off. Vasopressin antagonists have demonstrated potential in the treatment of water intoxication by altering the thirst response and decreasing the kidney's capacity to reabsorb water. Their use, however, needs to be closely monitored because of possible adverse effects, such as liver damage and hypernatremia [13]. Early diagnosis and a multidisciplinary approach are essential to prevent complications such as seizures, coma, or fatal water intoxication.

**Table 1: Laboratory findings of the patient**

Test	Results	Normal ranges
Serum sodium	118 mmol/L	135–145 Mmol/L
Serum osmolality	260 mOsm/kg	275–295 mOsm/kg
Urine osmolality	100 mOsm/kg	>500 mOsm/kg (normal concentrated urine)

## CONCLUSION

This case highlights the importance of recognizing PPD in psychiatric patients. Careful fluid management, electrolyte correction, and psychiatric intervention are key to successful treatment. Acetazolamide may be a promising adjunct therapy for water intoxication in PPD. This case underscores the clinical challenge of PPD leading to severe hyponatremia, a potentially life-threatening condition commonly seen in schizophrenic patients on antipsychotics like olanzapine. The patient's symptoms were due to excessive water intake, causing dilutional hyponatremia and low serum osmolality, confirmed by laboratory findings.

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*Funding: Nil; Conflicts of interest: Nil.*

**How to cite this article:** Rao TR, Sravya G, Kaushik T, Akshaya D. Psychogenic polydipsia: A case of water intoxication. *Indian J Case Reports*. 2025; 11(6):251-253.