CASE SERIES

The Unquenchable Thirst in Patients with Psychiatric Disorders: A Case Series of Psychogenic Polydipsia

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ABSTRACT

Introduction: Psychogenic polydipsia is a prevalent yet often underdiagnosed condition among individuals with psychiatric disorders. Left untreated, it can lead to severe complications, although these are rare. **Case series:** We presented three cases examining the occurrence of psychogenic polydipsia in patients with psychiatric disorders to elucidate its clinical features and therapeutic approaches within this specific population. Addressing the unique challenges it poses in patients with psychiatric disorder necessitates a comprehensive understanding of its manifestations and management. **Conclusion:** The presented cases underscore the urgency for increased awareness among mental health professionals, early identification of polydipsic symptoms, and tailored treatment strategies.
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INTRODUCTION

Primary polydipsia is characterized by excessive fluid intake leading to polyuria and diluted urine of 40–50 ml per kg body weight over a period of time after excluding secondary polydipsia (1). It is often associated with psychiatric conditions like schizophrenia (2), but recent studies suggest an increasing prevalence in the general population. Primary polydipsia is divided into dipsogenic polydipsia and psychogenic polydipsia.

Dipsogenic polydipsia, alternatively known as obligatory water consumption, predominantly manifests in individuals who purposefully consume substantial volumes of water to uphold a state of well-being or in individuals with compromised hypothalamic function. The act of obligatory water consumption is believed to enhance overall health and wellness, a trend that has been increasingly observed recently due to the widespread popularity of lifestyle initiatives (3).

Psychogenic polydipsia (PPD) is frequently observed in patients with psychotic disorders affecting over 20% of individuals, but it is important to note that it also occurs in individuals with other psychiatric conditions such as bipolar disorder, obsessive-compulsive disorder (OCD), anorexia nervosa, depression, anxiety, autism spectrum disorder, dementia and substance-use disorder (such as smoking and alcohol use disorder) (1,4,5). PPD entails polydipsia without physiological stimuli (6), potentially leading to severe complications such as hyponatremia, seizures, and, in extreme cases, death.

Despite being recognized since 1938, the aetiology of PPD is so far not conclusive. Several theories involving psychological and neurobiological components have been put forward to explain this phenomenon. Psychological stress and anxiety can exacerbate the urge to drink water excessively, turning the behaviour into a maladaptive coping strategy for dealing with emotional distress (7). In patients with anorexia nervosa, polydipsia may compensate for the low food intake and relieve the sensation of hunger (8).

From the neurobiological perspective, PPD is hypothesized to be caused by central impairment in thirst regulation, mainly by hypothalamus and anti-diuretic hormone (ADH) control. In patients with acute psychosis, there is an exacerbation in polydipsic behaviour alongside increased ADH levels. It is proposed that the activation of the hypothalamic-pituitary-adrenal axis and ADH secretion during acute psychosis may impact behavioural characteristics, potentially involving the

hippocampus. Notably, individuals with schizophrenia spectrum disorder and PPD have demonstrated reduced hippocampal volume in cranial magnetic resonance imaging (MRI) compared to those without PPD (9).

Besides that, the imbalances in neurotransmitters particularly dopamine may also be implicated in this condition. As dopamine is the primary neurotransmitter within the thirst regulatory centre situated in the lateral hypothalamus, drawing from findings of experiments conducted on animal subjects, one can hypothesize that heightened thirst and escalated fluid consumption are triggered by enhanced dopaminergic operations within the lateral hypothalamus. Conversely, a reduced sensation of thirst is linked to dopaminergic insufficiency. This theoretical framework aligns with the dopamine hypothesis concerning the positive symptoms of schizophrenia and elucidates the correlation between psychosis exacerbation and abnormal escalation in fluid consumption; thus explains the high PPD prevalence in those with psychotic disorder (7). Some psychotropics with anticholinergic effects may also induce sensation of thirst causing increased water intake.

Understanding the clinical presentation, risk factors, and potential treatments for PPD in patients with psychosis is crucial. This case series aims to highlight this condition by detailing the clinical course of three patients with psychiatric disorders experiencing complications due to PPD. Shedding light on this potentially life-threatening condition may aid clinicians in early identification and prevention of complications.

CASE SERIES

Case 1

A 29-year-old Malay single male, unemployed, was diagnosed with schizophrenia and intellectual disability ten years ago. He has been in remission for the past five years with oral olanzapine 15 mg nocte and a 40 mg flupentixol decanoate monthly depot injection. Five years into antipsychotic treatment, he began experiencing generalised tonic-clonic seizures, resulting in status epilepticus which required intubation. Blood parameters revealed severe hyponatremia with sodium levels of 107 mmol/L (normal range: 136–145 mmol/L). Other electrolytes and blood parameters were within normal range. Initial evaluation of hyponatremia was consistent with intravascular volume excess with diluted urine evidenced by urine sodium of less than 20 mmol/L (normal range: 20–40 mmol/L), urine osmolarity of 157 mOsm/kg of water (normal range: 500–800 mOsm/kg), and serum osmolarity of 249 mOsm/kg (normal range: 275–295 mOsm/kg). His inpatient electroencephalogram (EEG) revealed a left temporal focus with secondary generalization.

Further inquiry revealed a significant increase in water intake over several years. He would drink plain water approximately 6-7 litres per day, notably heightened over the past two years,

coinciding with increased cigarette smoking—around two packs daily. Unable to rationalize his water consumption, he justified it with a constant feeling of thirst, particularly after smoking. His urine output measured around 5 litres per day.

He was diagnosed with severe hyponatremia secondary to psychogenic polydipsia, complicated by seizures. Consequently, oral olanzapine was discontinued, and he continued with the monthly 40 mg flupentixol decanoate injection. Olanzapine discontinuation aimed to prevent further hyponatremia and to decrease its anticholinergic effects, which can cause dry mouth after smoking.

After stopping olanzapine, the patient's sodium levels returned to normal. Behavioural modification techniques were implemented to help him measure water intake at home and consume ice when thirsty. Additionally, efforts were made to reduce his smoking consumption. However, due to his intellectual disability, these behavioural modifications were challenging and required monitoring by his sister.

A follow-up a few months later revealed that the patient had successfully controlled his urge to drink water. According to his sister, he adhered to the instructions and limited his intake to only three litres per day, using a specifically labelled jug designated for his use.

Case 2

A 39-year-old Malay single male who was diagnosed with treatment-resistant schizophrenia for the past 20 years and diabetes mellitus for the past two years, was admitted to our

psychiatric ward after having an acute psychotic episode for three weeks following non- adherence to medication. He was previously on clozapine 200 mg nocte plus oral diabetic medications and maintained well for the past ten years. He presented with talking and smiling to himself, disorganized speech about religious topics, and disorganized behaviour characterized by playing with the water tap with full clothes on.

In the ward, he was observed to have frequent urination. Every hour, he would go to the toilet to urinate, including at night. Upon further questioning, this behaviour has been going on for the past four years in which he would drink a lot of water because he felt thirsty and subsequently urinated multiple times per day. We measured the water intake and collected his urine for 24 hours. Within 24 hours, he drank 6.25 litres of water and urinated 5.9 litres of urine.

In the ward, his initial random blood glucose ranged

from 10 to 18 mmol/L with HbA1c of 8.1%. However, after a few days of admission with resumption of his oral hypoglycaemic medications, his blood glucose normalized ranging from 6 to 8 mmol/L. For laboratory investigations, his sodium level was low at 133 mmol/L (normal range: 136–145 mmol/L). Other blood electrolytes, renal function, and liver function were normal. Urine sodium was <20 mmol (normal range: 20–40 mmol/L), urine osmolarity was 41 mOsm/kg (normal range: 500– 800 mOsm/kg), and serum osmolarity was 301 mOsm/kg (normal range: 275–295 mOsm/kg). His thyroid function was normal.

Looking at his low urine osmolarity and urine sodium, his polydipsia and polyuria were concluded to stem from psychogenic polydipsia instead of uncontrolled diabetes mellitus, which will cause osmotic diuresis with high urine osmolarity instead. Throughout his admission for three weeks, he was restricted to only three litres of drinking water in a labelled jug. As his psychosis improved with treatment, there was also a marked reduction in his urination frequency. The volume of urine collected had reduced to less than three litres per day.

Upon discharge, his sodium level had normalized to 141 mmol/L and maintained at 136-140 mmol/L after six months follow-up.

Case 3

A 30-year-old Malay single male, diagnosed with schizophrenia for three years, reported experiencing auditory hallucinations urging him to 'kill' someone unspecified, which he did not act upon. He also suffered from gustatory hallucinations, perceiving a bitter taste in his mouth despite its emptiness. Despite adhering to oral olanzapine 20 mg and oral aripiprazole 15 mg, he continued to experience residual psychotic symptoms. The gustatory hallucinations led to a reduced food intake, causing him to increase his water consumption whenever he was awake to alleviate his hunger. He reported drinking water continuously except when he was asleep, up to 6 litres per day. He also had frequent urination about once or twice in an hour.

After two years of treatment, he was admitted to the emergency department with multiple episodes of vomiting, severe lethargy, and weakness. All blood investigations were normal except for a sodium level of 110 mmol/L (normal range: 136–145 mmol/L). Urine sodium was 20 mmol (normal range: 20–40 mmol/L), urine osmolarity was 62 mOsm/kg (normal range: 500–800 mOsm/kg), and serum osmolarity was 256 mOsm/kg (normal range: 275–295 mOsm/kg). His thyroid function was normal.

He was diagnosed with symptomatic hyponatremia secondary to psychogenic polydipsia. Oral haloperidol 1.5 mg nocte was added to his regimen, and aripiprazole was discontinued to minimize the combination of antipsychotics. Typical antipsychotics, like haloperidol, are traditionally used for their effectiveness in managing positive symptoms but are associated with a higher risk of extrapyramidal side effects. Subsequently, he reported no further gustatory hallucinations, his appetite improved, and he successfully limited his water intake. He also tolerated haloperidol well without experiencing side effects. During follow-up visits, his sodium levels remained within the normal range.

Table I: Summary of clinical investigations of all patients

Parameter	Case 1	Case 2	Case 3	Reference Values
Serum				
Sodium (mmol/L)	107	133	110	135-145
Potassium (mmol/L)	3.2	3.3	4	3.5-5.0
Urea (mmol/L)	0.7	2.5	1.3	1.7-8.3
Creatinine (nmol/L)	56	79	64	70-130
Osmolarity (mOsm/kg)	249	301	256	275-295
Cortisol (nmol/L)	382.5	Not available	241.2	166-507
T4 (pmol/L)	12.72	19.21	27.71	12-22
TSH (mIU/L)	1.8	0.32	1.49	0.27-4.2
Urine				
Sodium (mmol/L)	<20	<20	<20	20-40
Osmolarity (mOsm/kg)	157	41	62	500-800

*mmol/L = milimoles per litre, nmol/L = nanomoles per litre, pmol/L = picomoles per litre, mOsm/kgH_2O = miliosmoles per kilogram of water, mIU/L = milli-international units per liter

DISCUSSION

In this manuscript, we presented three cases of psychogenic polydipsia (PPD) in patients with chronic psychiatric illnesses. All patients exhibited significant water intoxication symptoms, leading to hyponatremia. Notably, these cases underscore the intricate relationship between psychiatric disorders and dysregulated water intake behaviour.

As depicted in the case studies, the condition developed over the years, consistent with other studies diagnosing the condition in patients with history of 5–15 years of psychosis (6). The clinical mechanism involves a three-stage progression. It begins with simple polydipsia and polyuria. Subsequently, as patients continue excessive water intake, surpassing maximum urine dilution and fully suppressing anti-diuretic hormone (ADH) (10), their kidneys fail to excrete the excess fluid, resulting in water retention in the plasma, leading to water intoxication and subsequent physical complications such as hyponatremia, seizures, and coma (5). Patients often do not complain of polydipsia and polyuria, thus hyponatremia remains undetected until symptoms manifest. Therefore, we propose regular symptom inquiries by clinicians and more frequent electrolyte screenings for early detection.

Hyponatremia is often caused by multiple factors, and those taking psychotropic medications over a long period of time may experience low sodium levels only when additional risk factors are introduced, such as smoking, infections, older age, medical conditions and others (11). Antipsychotics-induced syndrome of inappropriate antidiuretic hormone (SIADH) should be suspected in patients, particularly if hyponatraemia happens within the first two to four weeks of antipsychotics initiation, in the absence of other clinical causes (12). Other than antipsychotics, other psychotropics such as antidepressants and anticonvulsants may also induce SIADH (13). While both conditions result in hyponatremia, PPD is primarily driven by behavioural factors and excessive water intake, whereas drug-induced SIADH involves pharmacological effects on ADH secretion or renal water reabsorption mechanisms. Drug- induced SIADH is primarily caused by the hypersecretion of ADH or the activation of intrarenal mechanisms for water reabsorption, leading to water retention and hyponatremia. In contrast, PPD is characterized by excessive fluid intake leading to hyponatremia. Clinically, SIADH is characterized by urine osmolality >100 mOsm/kg, euvolemia, high urinary sodium (>20 mmol/L), low serum uric acid, normal potassium, and normal acid-base balance, without signs of hypovolemia, hypotension, hypothyroidism, or adrenal insufficiency (14). Therefore, distinguishing between these conditions involves assessing the patient's medication history, psychiatric status, and specific laboratory findings related to sodium and water balance.

Due to the complex underlying mechanism, managing PPD poses a challenge, necessitating a holistic and multidisciplinary approach. Currently, no treatment guidelines exist for this condition. It is imperative to eliminate other causes of polydipsia before attributing it to psychogenic factors. Subsequently, identifying and managing associated risk factors, such as uncontrolled psychiatric symptoms (i.e. psychosis, obsessions), smoking, and psychological stress, is essential. In terms of antipsychotic selection, only clozapine has shown promising results in treating psychogenic polydipsia, while the usage of risperidone and olanzapine remains controversial (15).

Despite typical antipsychotics being associated with exacerbating polydipsia, our third case exhibited contrary results as the patient improved with the addition of haloperidol to control his psychotic symptoms. This is in keeping with a case-control study which showed lower risk of developing hyponatraemia with haloperidol (aOR 5.42, 95% CI 2.97–9.99) compared to other first-generation antipsychotics, while clozapine possessed a higher risk among the second-generation antipsychotics (aOR 1.26, 95% CI 0.98–1.62) (11). Therefore, antipsychotic selection should consider potential causative factors. Non-antipsychotic agents like naltrexone and acetazolamide have shown promise, although the latter lacks confirmation in randomized controlled trials (16). Treatment with selective serotonin reuptake inhibitors (SSRIs) and selective norepinephrine reuptake inhibitors (SNRIs) also aids in alleviating compulsive behaviour in patients with obsessive-compulsive disorder (OCD), as well as addressing symptoms of depression and anxiety, which could be confounding factors (16).

Psychosocial approaches should include patient and caregiver psychoeducation regarding increased water intake effects and the necessity to restrict intake, as fluid restriction remains the gold standard of treatment. Some cases report successful interventions using behavioural therapies such as relaxation techniques, response prevention, and cognitive behavioural therapy (CBT) (17). Calamari et al. reported a behavioural modification strategy involving operant conditioning principles, employing a token economy (18). This strategy, detailed in Fig. 1, effectively managed psychiatric patients with behavioural issues. However, challenges in behavioural modification strategies exist, including patient and caregiver suitability, variability in intervention findings, resource and time requirements, and limitations in outpatient settings.

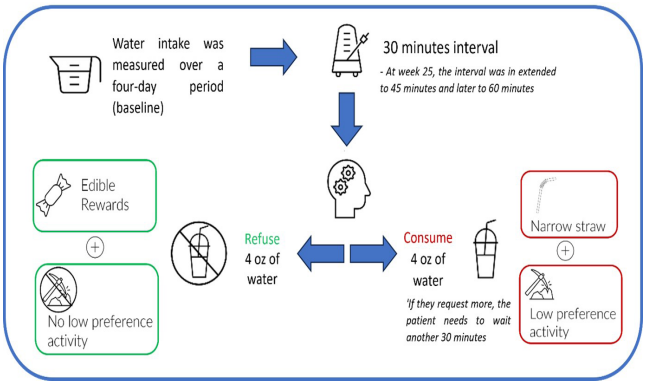

Fig. 1: A behavioural modification strategy for psychogenic polydipsia adapted from McNally et al, 1988 [9]. Baseline Phase: The patient had unrestricted access to water, but intake capped at 96 ounces within 24 hours to prevent potential water intoxication; Treatment Phase: The patient's day was segmented into 30-minute intervals. At the end of each interval, she was offered choices either to consume a specified amount of water or refuse it; Reinforcement Strategy: Water consumption was made less desirable by consumption via a narrow straw, making it more difficult, and followed by less preferred tasks (e.g., vocational work activities). Refusal of water was positively reinforced by edible rewards (initially including juice, gum, and candy) with a period of no required activity provided after refusing water; Interval Extension: The interval lengths was adjusted based on the patient's responses. It was extended from 30 to 45 and later to 60 minutes, aiming to observe and potentially alter water intake behavior based on these adjustments.

CONCLUSION

In conclusion, psychogenic polydipsia is a common yet frequently underdiagnosed condition. It presents a complex clinical challenge requiring a multidisciplinary approach for accurate diagnosis and effective

management. Personalised treatment plans aligned with the underlying psychiatric diagnosis and the patient's individual needs are crucial.

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