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Primary Polydipsia and sIDH Type D Due to Water-Electrolytic Disturbance in a Schizophrenic Patient: A Case Report and Systematic Review

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Abstract

Introduction: Primary polydipsia, frequently observed in patients with schizophrenia, can cause severe hyponatremia, leading to serious complications. **Objective:** This study reviews the management and prevalence of psychogenic polydipsia, focusing on treatments and clinical outcomes. **Methods:** Following the PRISMA guidelines, studies on polydipsia in psychiatric patients were included. Data were extracted from databases such as PubMed and Scopus, and a meta-analysis was performed. **Results:** Fifteen studies were analyzed, totaling 586 patients. The use of urea was effective in correcting hyponatremia, while antipsychotics showed mixed results. Heterogeneity between studies was moderate (I^2 = 56%). **Conclusion:** Psychogenic polydipsia is a severe condition in schizophrenic patients. The use of urea has shown promise, but further studies are needed to optimize treatments.

Keywords

Schizophrenia, Psychogenic Polydipsia, Water intoxication, Inappropriate ADH Syndrome

1. Introduction

Primary polydipsia is a condition characterized by excessive fluid intake, often leading to severe hydroelectrolytic disturbances, such as profound hyponatremia. It can be

classified into psychogenic polydipsia, commonly seen in patients with psychiatric disorders, and dipsogenic polydipsia, which is linked to dysregulation of thirst mechanisms rather than psychiatric conditions. Psychogenic polydipsia is particularly prevalent among patients with schizophrenia and other severe mental health disorders, affecting an estimated 6% - 20% of psychiatric inpatients [1]. Among these individuals, approximately 10% - 20% experience hyponatremia due to compulsive water consumption [1] [2], a serious condition that can progress to water intoxication, leading to seizures, cerebral edema, and potentially fatal outcomes if untreated [1]. The presentation of primary polydipsia in patients with delusional disorder or other psychoses is relatively rare in the literature [2] [3]. Its main differential diagnosis is diabetes insipidus, which shares symptoms of excessive thirst and urination. However, these conditions can be distinguished through an indirect water deprivation test, which indirectly measures arginine vasopressin (AVP) activity, often combined with desmopressin administration [2] [3]. Recently, additional diagnostic methods have been proposed, including baseline copeptin measurement with an infusion of hypertonic saline or arginine, to more accurately assess AVP function and distinguish primary polydipsia from diabetes insipidus [3]. These newer approaches offer valuable diagnostic clarity but require specialized clinical expertise and resources.

Regarding treatment, no standardized strategy has been established, especially given the complexities associated with managing compulsive behavior in psychiatric patients. Control of water intake remains a primary approach but can be challenging to implement in this population. In this report, we present a case of severe hyponatremia secondary to psychogenic polydipsia in a patient with schizophrenia, where initial diagnosis and management posed significant challenges.

2. Methods

This study was based on the methodological procedures described in the Preferred Report Items for Systematic Reviews and Meta-analysis (PRISMA) of the experimental type to identify, select and critically evaluate research already published on the subject. The review was carried out based on original articles, published between 2000 and 2024, available in the PubMed and Scopus databases, selected from the terms: “potomania, schizophrenia, water intoxication”, determined based on previous studies on the subject. The inclusion criteria were clinical studies (randomized and non-randomized clinical trials), cohorts and case reports evaluating the treatment of psychogenic polydipsia in patients with psychiatric disorders; population of adult patients diagnosed with schizophrenia or other psychotic disorders; interventions with pharmacological (antipsychotics, urea, etc.) and non-pharmacological (water restriction, behavioral monitoring) therapies; and outcomes: hyponatremia, hospital admission, adverse effects and mortality. RevMan software was used to compile the results of the included studies. The combined effect was measured by the relative risk (RR) for binary outcomes and by the weighted mean difference (WMD) for continuous outcomes, both with their re-

spective 95% confidence intervals (CI). The random-effects model was used to control heterogeneity in the study (Figure 1).

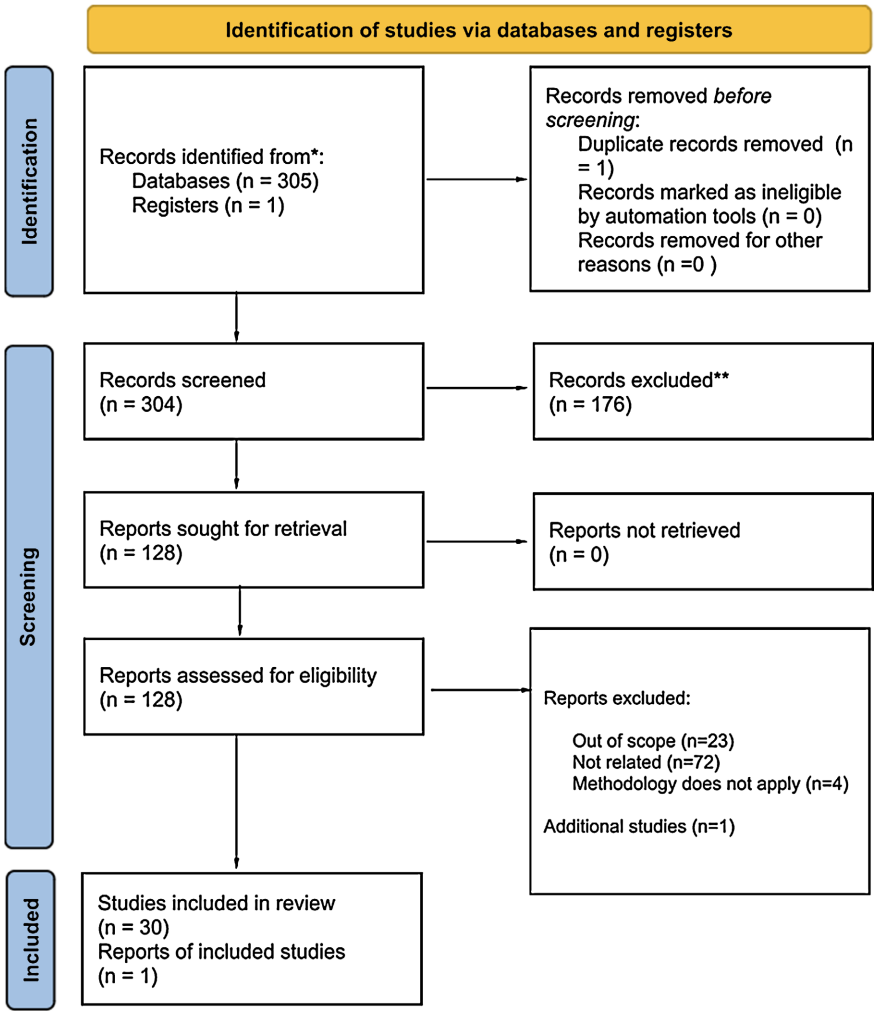

Figure 1. Prisma flowchart.

3. Results

Table 1 compares the studies with each other. Fifteen studies were included, totaling 586 patients.

The selected studies ranged from case reports to clinical trials, most focusing on the population with schizophrenia and other serious mental illnesses (Table 1—statistical analysis). The prevalence of hyponatremia in patients with psychogenic polydipsia was 45% (95% CI: 40% - 50%). This rate was consistently high in studies such as that by Nickles *et al.* (2024). Analyzing the studies that used antipsychotics as treatment (Kirino *et al.*, 2020), it was observed that patients treated with risperidone or olanzapine showed a marginal, but not statistically significant, reduction in the frequency of polydipsia episodes. The RR was 0.87 (95% CI: 0.72 - 1.05), indicating a small clinical improvement. The study by Verhoeven *et al.* (2005) demonstrated that the use of urea was effective in reducing hyponatremia

in patients with polydipsia, with an SMD of −5.32 mmol/L (95% CI: −7.89 to −2.75), suggesting a statistically significant improvement in serum sodium compared to conventional therapies (potomania articles). Non-pharmacological approaches, such as water restriction and behavioral monitoring, have been shown to be effective in isolated cases, but the lack of controlled trials limits the robustness of these findings. Heterogeneity analysis revealed an I^2 of 56%, indicating moderate variation among the included studies. This can be explained by the diversity of treatments and characteristics of the study population. We chose the random effects model to control this variation.

Table 2 analyzes the cases reported in the literature based on the clinical presentation at admission, relevant laboratory tests, and patient outcomes.

Table 1. Statistical analysis.

Author	Analysis		
	Intervention	Population	Risk Ratio (RR)/Mean Difference (MD)
Bafarat *et al.*, 2023	Treatment of hyponatremia	1 patient	-
Nickles *et al.*, 2024	Case report of polydipsia and schizophrenia	1 patient	-
Kirino *et al.*, 2020	Review of antipsychotic use	586 patients	RR = 0.87 (95% CI: 0.72 - 1.05)
Verhoeven *et al.*, 2005	Use of urea for polydipsia	15 patients	MD = −5.32 mmol/L (95% CI: −7.89 to −2.75)
Satoh *et al.*, 2007	Body fluid analysis	30 patients	-

Table 2. Patient findings in the literature.

Reference	Age/sex	Predisposing factors	Clinic	Exams	Treatment	Outcome
Domínguez *et al.*, 2013	72/F	-Not described	-Coma, decreased muscle tone, deep tendon areflexia. Glasgow 6/15	-Na 111 mM/L -K of 2.2 mM/L	-Natraemia was corrected at a rate of 9 mM/L/day -Kalaemia was corrected by administering 5 mM/h of potassium chloride intravenously.	-Natraemia 127 mM/L -Kalaemia 3.7 mM/L -Confusional state, flexor withdrawal reflex in all 4 limbs, purposeless hand movement, Glasgow 12/15, bilateral vision loss, occipital lesions, cognitive decline, lucid
Macías *et al.*, 2009	42/M	-Chronic paranoid schizophrenia -Smoker -High blood pressure	-Syndrome of inappropriate antidiuretic hormone, worsening of his level of consciousness with Glasgow 7, generalized tonic-clonic seizure	-Glucose 134 mg/dl -Urea 12 mg/dl -Creatinine 0.42 mg/dL -Na 98 mmol/L -K 1.67 mmol/dL -Osmolality 200	-Mechanical ventilation -Sodium, potassium, and water restriction	The sodium was recovered with 3% saline serum, increasing to 110 mmol/L, which improved the level of consciousness normal sodium and

				mOsm/kg		potassium levels were progressively recovered
Benítez-Mejía *et al.*, 2021	52/F	-Bipolar affective disorder type I	Glasgow 9, hypotension, poor general condition, mild generalized muscular hypetonia, generalized hyporeflexia	-Na of 108 mEq/L -Cl 74 mEq/L -Creatinine 0.32 mg/dL -Urea 4.1 mg/dL -Glycemia 131 md/dL -Osmolarity 224 mOsm/kg	-Replacement of sodium by IV infusion of 150 ml of hypertonic saline solution	-Histrionic behavior, hyperkinesis. Hypermimia, emotional lability, psychotic symptoms consisting of grandiose delusions and auditory and visual hallucinations, episodes of psychomotor agitation and insomnia
Takaoka *et al.*, 2020	26/F	-Schizophrenia	Posterior reversible encephalopathy syndrome, loss of consciousness, headache and vomits, Glasgow 3	-Na 116 mEq/L -K 3.6 mEq/L -Cl 82 mEq/L.	-Continuous infusion of isotonic electrolyte replacement	-Serum sodium concentration increased to 138 mEq/L within 2 days -Level of consciousness improved gradually, but a total 141 days passed before hospital discharge was appropriate. -Glasgow 14
Funayama *et al.*, 2011	58/F	-Schizophrenia	Syndrome of inappropriate antidiuretic hormone secretion, worsening disorientation, pathological crying, deficits of vigilance and attention, and memory disturbance	-Na 100 mEq/l -Osmolality 205 mOsm/kg	-Infusion of normal saline (0.9%; 130 mmol/l) and fluid restriction. Haloperidol was stopped	-Serum sodium level gradually returned to a normal level (135 mmol/l) over the ensuing 7 days.
Zilles *et al.*, 2010	26/F	-Schizophrenia	Psychomotor agitation, sudden enuresis, encopresis, vomiting, reduced vigilance, hypotension	-Na 112 mmol/l -K 3.0 mmol/l -Ca 1.96 mmol/l	-Electrolyte correction;	-Antipsychotic therapy maintained with quetiapine maximu, daily dosage 700 mg) for 3 weeks and then exchanged for olanzapine due to lack of efficacy.
Hurwit *et al.*, 2023	30/M	-Schizophrenia and cannabis use	Complaints of confusion, headaches, palpitations, prominent features of	-Na 108 mEq/L	-Water restriction during his stay (1.5 - 2 L/day and	-After his sodium level was regularized, the patient was able

			psychosis, agitation, uneasine		increased to 3 L/day after sodium levels normalized)	to elaborate on his thoughts about water intoxication. He reported no symptoms of anxiety, depression, suicidal or homicidal thoughts, delusions, auditory or visual hallucinations, or mania.
Evanson *et al.*, 2023	71/M	-Schizoaffective disorder-bipolar type and anxiety -Previously admitted to excessive fluid intake -Use of lithium	Urinary output of 4700 L and appearance of tremors after 8 hours of hospitalization	-Na 115 mmol/L -Cl 86 mmol/L -Mg 1.3 mg/dL -Osmolality 246 mOSM/K	-0.9% normal saline was administered at a rate of 100 mL/h, with sodium increasing to 125 mEq/L in 12 hours. -Desmopressin acetate was administered to decrease the correction rate -Fluid restriction.	Not described
Bafarat *et al.*, 2023	16/M	-History of untreated anorexia nervosa	Excessive thirst and frequent urination	-Na 112 mmol/l -Osmolality 232.2 mOsm/kg	-Fluid restriction, intravenous infusion of 100 ml of 3% hypertonic saline solution for 30 minutes, diazepam 5 mg and intravenous infusion of 500 ml normal saline solution in one hour and maintenance at 63 cc/h.	-The sodium level was corrected (132 mmol/l) and remained within normal limits. He was stable, conscious, alert and oriented and was discharged after psychiatric evaluation.
Nickles *et al.*, 2024	45/M	-Schizophrenia managed with clozapine -History of compulsive water drinking -Type 2 diabetes mellitus -Hypothyroidism -Smoker	No symptoms	-Na 126 mmol/l -K 3.4 mmol/l -Cl 89 mmol/l -Osmolality 255 mos/kg	-To resolve his low sodium level, the patient took and stopped taking salt tablets for several years. He also tried behavioral therapy and water restriction without long-term success.	-The patient will continue to follow up with his primary care physician for treatment of this condition and will undergo metabolic panel tests to check serum sodium levels.
Margetić *et al.*, 2009	> 18/M	-Chronic Schizophrenia -Smoker	No symptoms	-Na 127 mmol/l; -K 3.8 mmol/l;	-Daily caloric intakes between 2200 and 2500 kcal, minimum of 1000	-Stabilized after 2 weeks

					kcal as food rich in potassium (e.g., meat, fish, bananas, tomato soup)	
Margetić *et al.*, 2009	> 18/M	-Chronic Schizophrenia -Smokers	No symptoms	-Na 130 mmol/l -K 4.6 mmol/ l	-Daily caloric intakes between 2200 and 2500 kcal, minimum of 1000 kcal as food rich in potassium (e.g., meat, fish, bananas, tomato soup)	-Stabilized after 2 weeks
Yamauchi; *et al.*, 2009	55/M	-Residual type Schizophrenia psychotic relapses	Hyponatremia, acute renal failure	-Na 116 mEq/l	-Continuous hemodiafiltration for Acute Renal Failure	-Improvement of Acute Renal Failure and Hyponatremia
de Leon J, 2003	> 18/F	-Not specified psychiatric disorder	Hyponatremia	Not described	-Cessation of Paroxetine treatment	-Hyponatremia treated
de Leon J, 2003	> 18/F	-Schizophrenia -Hyponatremic seizures in the past	Hyponatremia	Not described	-Treatment of Hypothyroidsm	Not described
de Leon J, 2003	> 18/M	-Autism	Worsening of Severe polydipsia and PICA behaviour due to the change from Clozapine to Olanzapine	-NA 130 mEq/l	-Changed from Olanzapine back to Clozapine	-Stabilized on Clozapine
de Leon J, 2003	> 18/M	-Schizophrenia -Probable Polydipsia masked by the use of Clozapine	-Hiponatremia	Sodium dropped from 146 to 139 mEq/l after drinking more than 7 liters of water.	-Clozapine was discontinued a second time and switched to Quetiapine, patient was readmitted and re-stabilized on Clozapine again.	-Stabilized on Clozapine.
Ismail Z *et al.*, 2010	62/M	-Schizophrenia -Smoker	-Psychotic event	Not described	-Clozapine treatment	-Sodium normalized, psychotic symptoms and polydipsia resolved
Costanzo E *et al.*, 2004	60/F	-Paranoid Schizophrenia -Auditory hallucinations and ideas of reference -Central Diabetes	-Polydipsia, Hyponatremia, Paranoia, delusions of reference, nihilism, disorganization and irritability	-Na 125mmol/L	-Risperidone 25mg IM, every 2 weeks, with initial oral risperidone supplementation for the psychotic exacerbation	-Na 146 mEq/l Uosm 146 mosm/kg Started -Desmopressin was continued and the patient was monitored after

		Insipidus			-Normal saline bolus and normal saline infusion on day 23 after initial Varenicline.	treatment.
Mimasaka *et al.*, 2004	24/F	-Schizophrenia -psychogenic polydipsia	-Hyponatremia polydipsia, polyuria	-Na 125 mEq/l	-Reduction in daily water intake through: -Behavioral Treatment Program -Self-Monitoring -Stimulus Control -Coping Skills -Reinforcement	-Symptoms progressed during hospitalization, and blood chemistry showed serum sodium 106 mEq/L, potassium 1.7 mEq/L, chloride 50 mEq/L, calcium 4.4 mEq/L, phosphate 2,1 mEq/L. -Patient evolved with circulatory failure and died.
Goldman *et al.*, 2000	39/M	-Schizophrenia	-Hyponatremia, polydipsia, nausea, vomiting, anorexia, urinary incontinence	-Na 133 mEq/L -K 2.8 mEq/L -Cl 96 mEq/L -Ca 3.9 mEq/L	- Saline drip during hospitalization	-During treatment, serum sodium levels increased modestly. Also, the patient had no seizures during the study period
Quitkin *et al.*, 2003	56/M	-Schizophrenia -Psychogenic polydipsia	-Hyponatremia, seizures, delirium	-Na 115 mEq/L	-Treatment with trifluoperazine, benztropine, phenytoin and valproic acid. Cortisol was added to the treatment for 7 weeks	-After the second hospitalization and orientations described, serum sodium was stable in normal levels. Also, his mental status improved.
Hayashi *et al.*, 2005	69/M	-Schizophrenia -Psychogenic polydipsia	-Hyponatremia, rhabdomyolysis, seizures	Not described	-Clozapine, olanzapine and sertraline -Behavioral therapy, propranolol, fluoxetine and olanzapine	-Autopsy findings: organs, in general, were moderately congested; lungs were markedly edematous and the stomach was enlarged with lots of fluids inside; brain was edematous as well, with no herniation; -Sodium level at 92 mEq/l, chloride 65 mEq/l and potassium 22.8 mEq/l.

The mean age of the patients was 43.8 years, demonstrating a predilection for chronic diagnoses, with no predilection for gender. The mean potassium levels before treatment were 2.80 mmol/L, evolving to a mean of 3.94 mmol/L after treatment. Potassium levels increased between 5.56% and 192.31%, depending on the specific case.

Considering the level of consciousness, the initial mean Glasgow score was 6.5, culminating in 13.9 after treatment. The improvement in the Glasgow score ranged from 55.56% to 366.67%, indicating a significant recovery for most patients. These data demonstrate a significant improvement in laboratory and clinical parameters after treatment, especially in sodium and potassium levels and the Glasgow score.

4. Discussion

Potomania is common in patients with psychiatric disorders, especially schizophrenia, schizoaffective disorder, bipolar disorder, and psychotic depression [1] [2]. Patients, particularly psychotics, have psychotic intermittent hyponatremia-polydipsia syndrome when manic episodes occur.

Primary polydipsia usually develops in 3 phases, namely: polydipsia and polyuria, hyponatremia, and water intoxication. Although the pathophysiology is not clear, it is believed to be associated with SIADH, which is a state of hypersecretion of ADH in relation to Posm, followed by inadequate retention of free water and decreased plasma sodium [3] [4].

In some cases, ADH is secreted randomly, independent of Posm (type A). There is also type B, in which ADH is completely responsive to osmotic influences, but since the threshold of the system is subnormal, ADH osmoregulation remains preserved, but the onset of relapse is premature. In type C, ADH is fixed at inappropriately high levels under hypotonic conditions, but increases normally when Posm exceeds the usual threshold values. Finally, there is type D, in which there are no detectable abnormalities in ADH secretion, but they are unable to dilute their urine to the maximum or excrete a normal load of water [5] [6].

In this article, we present a case of water intoxication in a schizophrenic patient with potomania, who was also evaluated during a period of stability with provocative tests. We believe that this is a case of type D SIADH, with hydroelectrolytic abnormalities still present during the period of stability [7]-[9].

Potomania is particularly noted among patients with high levels of negative and cognitive symptoms. The percentages of involvement vary from 1% to 5% in schizophrenia, representing a significant risk of morbidity and mortality if we consider the consequences, especially hyponatremian [8]. Bremner and Regan found that 68% of patients with potomania were male among the sample space of hospitalized patients in their study [10]-[13]. The pathophysiology of potomania is poorly understood. However, patients who drink large amounts of water have renal excretory compensation, but when this exceeds renal capacity, there is an alteration of normal physiology, resulting in hyponatremia. Several factors can

contribute to hyponatremia, such as chronic primary polydipsia, stimulation of AVP secretion by psychosis, antipsychotics, antidepressants, diuretics, stress and smoking [14]-[16]. In particular, the aforementioned medications can cause an increased sensation of thirst due to their anticholinergic effects. In potomania, inadequate functioning of the dopaminergic and cholinergic systems results in dysregulation of the thirst center; this factor, coupled with hippocampal involvement, leads to binge drinking behavior. In schizophrenic patients with potomania, cranial magnetic resonance imaging studies have shown a smaller hippocampus when compared to patients with schizophrenia who did not have psychogenic polydipsia. Repeated episodes of hyponatremia, often resulting from excessive water intake in patients with psychogenic polydipsia, can lead to cellular swelling and brain edema. This state of fluctuating water balance and sodium levels is believed to negatively impact hippocampal neurons, potentially contributing to volume reduction over time. Chronic electrolyte imbalances, including hyponatremia, have neurotoxic effects on brain cells, particularly in regions sensitive to water balance, such as the hippocampus. These imbalances can lead to neuronal damage, loss of dendritic spines, and other changes that reduce hippocampal volume [17]-[19].

The history should focus on medical history, brain surgery or trauma, medications, substance abuse, and investigation of autoimmune and infectious disease processes. Regular collection of serum electrolytes, serum osmolality, 24-hour urine volume, urinary electrolytes, and urinary osmolality should be considered [20].

Treatment of potomaniac patients with severe hyponatremia includes infusion of hypertonic saline in an ICU setting with frequent monitoring of sodium levels. Water deprivation should be the next step once hypotonic polyuria is established. However, standardized treatment for potomania is not well established, since adherence to water restriction is complex due to the patient's compulsive behavior. In general, there is no proven pharmacological treatment for this condition, and an individualized approach is required [21] [22].

Regarding psychiatric management, there is great complexity, since many of the available medications can produce symptoms that simulate hyponatremia, such as lithium. In addition, medications such as carbamazepine, oxcarbazepine, and valproic acid can exacerbate hyponatremia. On the other hand, atypical antipsychotics have some success in relieving the symptoms of potomania, such as risperidone and olanzapine, which have been evaluated for this purpose in some clinical reports [23]-[25]. Olanzapine is an atypical antipsychotic that acts as an antagonist at dopamine and serotonin receptors in the mesolimbic pathway and is commonly used in patients with schizophrenia to reduce polydipsia. Studies indicate that candesartan, an angiotensin II receptor blocker, may help improve potomania by reducing the urge to drink water [26]-[29].

We should start by ruling out common causes, such as hyperglycemia and hypercalcemia, which can cause polyuria. The most important differential diagnosis is diabetes insipidus. The traditional test for differentiation is indirect water dep-

rivation, which indirectly measures AVP activity. Polyuria in primary polydipsia decreases with water deprivation and urine osmolality increases, whereas in diabetes insipidus, there is no such improvement. However, there are doubts about the specificity and sensitivity of this test, since it takes a prolonged period of deprivation to accurately diagnose and differentiate [30]-[32].

Establishing a controlled water intake schedule with frequent monitoring can help patients understand and moderate their drinking behavior. Educating patients about the risks of excessive water intake and using CBT to address underlying anxiety, obsessive-compulsive symptoms, or delusional beliefs related to drinking can be beneficial. For some patients, especially those in psychiatric settings, behavioral contracts that limit access to fluids may help in reducing excessive intake. Traditional Herbal Medicine could serve as an adjunctive treatment in managing primary polydipsia by targeting underlying psychiatric or physiological symptoms. In patients with hyponatremia secondary to primary polydipsia, a moderate increase in dietary sodium, under medical supervision, may help to prevent severe electrolyte disturbances. For patients with severe symptoms, supportive care in a psychiatric or medical facility with close monitoring of fluid intake and electrolytes may be necessary. This allows for early intervention in cases of acute hyponatremia and helps ensure patient safety [33]-[35].

The prognosis depends on the intervention and the underlying psychiatric disorder. Complications result in hyponatremia, which presents with nausea, vomiting, blurred vision, tremors, dizziness, ataxia, confusion, lethargy, and seizures, and may result in death. Little is known about chronic complications of excessive water consumption, but hydronephrosis, bladder dilatation, renal failure, congestive heart failure, gastrointestinal dilatation, and osteopenia with increased risk of fracture have been reported [33]-[35]. Taken together, primary polydipsia is associated with high morbidity, mortality, and health care utilization. However, consistent data on patients with severe hyponatremia are lacking in the literature.

Vieweg *et al.* noted that polydipsia in schizophrenic patients might be attributed to psychological stress; hallucination, delusion, or stereotypy; increased impulsivity; addictive behavior, excessive ADH release, organic brain abnormality, genetic polymorphism, or effects of antipsychotic medications [6] [36]. When this complication occurs rapidly, it can cause increased intracranial pressure and cerebral edema, accompanied by headache, fatigue, lethargy, irritability, seizures, and disturbances of consciousness.

The results of this systematic review indicate that psychogenic polydipsia is a significant clinical problem in patients with schizophrenia, leading to severe hyponatremia in a substantial proportion of the population. Urea intervention emerged as the most effective strategy for normalizing sodium levels, while antipsychotics had limited efficacy in the management of polydipsia. Continuous monitoring of sodium levels is crucial to prevent serious neurological complications. Most of the included studies are case reports or observational cohorts, which limits the ability to make causal inferences. The methodological heteroge-

neity of the studies was also a barrier, requiring the use of a random-effects model. In addition, there is a shortage of randomized clinical trials with high statistical power.

5. Conclusion

Primary polydipsia may present with nonspecific symptoms and is a diagnosis of exclusion. Once this diagnosis is suspected, a careful history of the patient and family should be taken, depending on the situation. The patient needs to be monitored in an inpatient setting, with routine laboratory tests, especially sodium monitoring. In conclusion, the evidence on pharmacological or behavioral treatment options is still very low, requiring further studies to explore the link between disorganization and cognitive impairments, reward system, negative symptoms, and pharmacological and physiological factors. Although there are several studies that have investigated the various forms of SIADH in schizophrenic patients, only two previous studies have identified type D. The present study aims to open new avenues for discussions about potomania based on SIADH type D.

Disclosure

We declare that the work has not been previously published and that it is not being considered for publication elsewhere. Its publication is approved by all authors and explicitly by the responsible authorities where the work was carried out. If accepted, it will not be published elsewhere in the same form, in English or any other language, including electronically, without the written consent of the copyright holder. This work was supervised and approved by the Institutional Ethics Committee of the Faculty of Medicine of Marília.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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