CASE REPORT

Psychogenic polydipsia and hyponatremia – A side effect of psychosis: a review with a case report

Polidipsia psicogênica e hiponatremia – Efeito colateral da psicose: revisão com relato de caso

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ABSTRACT

Primary polydipsia, or psychogenic polydipsia, is a condition that results in considerable morbidity and mortality. In psychiatric patients, psychogenic polydipsia and the syndrome of inappropriate antidiuretic hormone secretion may cause hyponatremia. In the 1970s, it was recognized that antipsychotics such as tiotixene and haloperidol could impair the excretion of a free water load. There are also several case reports of drug-induced hyponatremia in patients using atypical drugs suggesting that these probably can also impair water balance and induce hyponatremia. Case report and review of relevant literature are reported in this article. Psychogenic polydipsia is a common cause of hyponatremia among individuals with chronic mental illness. A case of severe hyponatremia caused by psychogenic polydipsia is described involving a female patient with an adult lifelong history of chronic mental illness diagnosed as schizoaffective disorder. After switching her antipsychotic medication to clozapine water ingestion was normalized as well as sodium levels and her psychotic symptoms improved. Primary polydipsia occurs commonly with schizophrenia and other mental diseases and can cause hyponatremia. PPD may present as an acute psychotic state or as inexplicable emergence of seizures. Appropriate, timely clinical assessment with special attention to thirst, fluid intake, and urine output is essential. Proper treatment may include drug withdrawal and fluid and saline restriction. Once corrected, some pharmacological agents can be tried. The article illustrates the importance of the diagnosis of psychogenic polydipsia given its electrolyte disturbances and life threatening situations.

Keywords
Primary polydipsia, psychogenic polydipsia, hyponatremia, psychotropic medication, antipsychotics.

RESUMO

A polidipsia primária ou polidipsia psicogênica (PDP) é uma condição que se traduz em altas taxas de morbilidade e mortalidade. Nos doentes psiquiátricos, a PDP e a síndrome da secreção inapropriada do hormônio antidiurético (SIADH) podem resultar em hiponatremia. Nos anos 1970, foi reconhecido que alguns antipsicóticos como o tiotixeno e o haloperidol

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podiam comprometer a excreção de água livre. Existem ainda diversos casos reportados de hiponatremia iatrogênica em pacientes medicados com antipsicóticos atípicos sugerindo que essa classe farmacológica pode igualmente resultar em hiponatremia. Um caso clínico e uma revisão sobre o tema são reportados neste artigo. A polidipsia psicogênica é uma causa comum de hiponatremia entre os indivíduos com doença mental crônica. Um caso de hiponatremia grave causada por polidipsia psicogênica é descrito envolvendo uma paciente do sexo feminino com antecedentes de perturbação esquizoafetiva. Após alteração da sua medicação antipsicótica para clozapina, observou-se normalização na ingestão hídrica bem como nos níveis de sódio e melhoria da sintomatologia psicótica. A PDP ocorre comumente em doentes esquizofrênicos ou com outras perturbações psiquiátricas e pode resultar em hiponatremia. Um correto diagnóstico com especial atenção a sede, ingestão hídrica e débito urinário é essencial. O tratamento pode incluir a retirada de fármacos e restrição hídrica e salina. Uma vez corrigida, alguns agentes farmacológicos podem ser utilizados.

Palavras-chave
Polidipsia primária, polidipsia psicogênica, hiponatremia, psicofármacos, antipsicóticos.

INTRODUCTION

Psychogenic polydipsia (PPD) is a condition that results in considerable morbidity and mortality[1].

Hyponatremia is seen in about 4% of chronic schizophrenic patients and sometimes in patients with other psychiatric disorders like psychotic depression, bipolar disorder and mental retardation[2]. It can lead to serious clinical consequences such as delirium, seizures or rhabdomyolysis and is also an important risk factor for neuroleptic malignant syndrome[3].

In psychiatric patients, PPD and the syndrome of inappropriate antidiuretic hormone secretion (SIADH) may cause hyponatremia[2]. PPD is characterized by low plasma sodium in 10% to 20% of those presenting with uncontrollable drinking[1]. Symptoms are uncommon unless patients continue to drink excessively (> 10 L/d) after they reach their limit of urine dilution (100 mOsm/kg with minimum urine osmolality) and full antidiuretic hormone (ADH) suppression[4].

In the 1970s, it was recognized that antipsychotics such as tiotixene and haloperidol could impair the excretion of a free water load. Later, it was revealed that elderly patients treated with phenothiazines had significantly lower serum sodium levels than patients not treated with these drugs. Additionally, it was shown that a greater portion of patients treated with haloperidol had impaired free water excretion and urinary dilution than asymptomatic controls[5,6]. There are also several case reports of drug-induced hyponatremia in patients using atypical antipsychotics suggesting that these might also impair water balance and induce hyponatremia[7].

This article is designed to present a clinical case of a psychotic patient with psychogenic polydipsia and hyponatremia. A review of the recent literature is presented, based on the best available evidence highlighting the importance of a correct diagnosis and treatment (pharmacologic and behavioral interventions).

CASE REPORT

A 48-year-old female, addressed as Ms. L, presented to our emergency department with altered mental status including disinhibition, dysphoric mood with pressured and coprolalic speech, disaggregated thought and delusional ideas of persecution, increasing in severity over the past 5 days. The patient had a longstanding history of schizoaffective disorder and had been managed with haloperidol 15 mg/day, haldol decanoate 100 mg/month, levomepromazine 150 mg/day, carbamazepine 200 mg ER twice a day, trihexyphenidyl 4 mg/day, lorazepam 10 mg/day. She was also on carvedilol 25 mg/day, irbesartan 300 mg/day, amlodipine 10 mg/day and indapamide ER 1,5 mg/day to control her blood-pressure levels.

A report from the patient's family revealed that she had increased her fluid intake to up to 6-7 L of water daily.

A detailed examination was performed including a blood sample that revealed a serum sodium level of 119 mmol/L, potassium of 3.32mmol/L and chloride level of 79.6 mmol/L. Thyroid hormones, glucose, serum urea and creatinine levels were in the normal range. Urine levels of potassium, sodium and creatinine were also obtained (3.0 mmol/L; 20 mmol/L; < 4.2 mg/dL, respectively) as urine osmolality (43 mOsm/KgH_2O).

She was immediately hospitalized in our psychiatry unit and performed additional analytic and imaging studies including cortisol (8h) and fasting aldosterone levels, both in the normal range, and a CT scan that didn't reveal any morphologic change.

It was diagnosed a normovolemic hyponatremia and a hypertonic saline solution to correct the electrolytic imbalance was prescribed.

Levomepromazine, carbamazepine and indapamide were suspended and haloperidol was reduced to 10 mg/day. Oral fluid restriction of 1.5 L/day was instituted although she complained of intense thirst.

Sodium level normalized in 5 days but she was still delusional and dysphoric with no symptom improvement and always trying to sneak to the bathrooms to drink water. Clozapine was initiated as she had failed several trials of first and second generation antipsychotics. The dose was titrated until 400 mg/day with analytic surveillance, without intercurrences. After three-weeks the sodium levels were at the normal range and she started presenting with less delusional ideas and a more adequate behavior interacting more appropriately with the medical team.

She was discharged one week later with no psychotic symptoms and an adequate water ingestion.

Twelve months after the patient remained free of psychotic or mood symptoms.

DISCUSSION

Hyponatremia is the result of an imbalance in water-electrolyte homeostasis, usually defined as a lowered serum sodium level of < 136 mmol/L[8].

The differential diagnosis of hyponatremia is complex and includes many different etiologies, however it can be simplified into two categories: high (> 295 mOsm/kg) versus low (< 280 mOsm/kg) plasma osmolality. Low plasma osmolality can be further divided into hypo-, hyper-, or normovolemia[8]. Ms. L was included in this last category.

Most psychiatric patients with hyponatremia will fit into the normovolemic category and will have low plasma osmolality, lacking clinical indicators of altered volume status. PPD and SIADH belong to this category[9].

Most cases of PPD with hyponatremia present a very high volume of intake. These cases have very low levels of ADH and urine osmolality is very low (< 100)[9] as seen in the presented case.

Clinical manifestations of hyponatremia are commonly related to the osmotic water shift towards the intracellular space, with subsequent increased intracellular fluid volume that can cause cerebral edema. The symptoms are primarily neurological, with their severity related to the speed of onset and the absolute decrease in the serum sodium concentration. Patients may be initially asymptomatic or can experience nausea, malaise or confusion. As the serum concentration continues to fall, additional symptoms including headache, muscle cramps and lethargy can present, increasing confusion and agitation. Caution must be taken in correction of hyponatraemia as central pontine myelinolysis may occur if correction is overly rapid. A correction rate of less than 10 mmol/L is advisable over the course of 24 hours[10].

Although PPD is multifactorial, malfunction of the hypothalamic thirst center is seen as a possible cause. Chronic intake of excess fluid may change feedback regulation of the hypothalamic-pituitary axis, leading to dysregulation of antidiuretic hormone (ADH) secretion[11]. Autoimmune illnesses may also lead to hypothalamic lesions, causing increased thirst. Another theory proposes that patients with PPD present with polydipsia even when ADH is fully suppressed[11]. Patients exhibit symptomatic hyponatremia when the kidney's short-term capacity to excrete urine (10 to 15 L/d) is overwhelmed.

There are various other theories concerning the etiology of PPD including schizophrenia positive symptoms, compulsive behavior, stress reduction and efforts to counteract the anticholinergic medication side effects[1]. It was proposed that the increased prevalence of ventricular enlargement in schizophrenic patients may lead to structural impairment of the lateral hypothalamus and disturbances in thirst and osmolality. The polydipsic theory of dopamine supersensitivity is another explanation for the association of polydipsia and psychosis. Dopamine is an important neurotransmitter in the area of the thirst center in the lateral hypothalamus. In animals, a hyperdopaminergic state is associated with increased fluid intake, which can be reversed by dopamine antagonists. High dopamine levels are hypothesized to stimulate thirst centers and dopamine receptor hypersensitivity might explain why PPD often occurs late in the course of schizophrenia, after years of exposure to typical neuroleptics[11,12].

PPD may present as an acute psychotic state or as unexplained emergence of seizures. Some studies show a temporal relation between exacerbations of psychosis and psychogenic polydipsia and water intoxication in psychiatric patients[10].

Most inpatients with PPD require evaluation and treatment of their hyponatremia. Symptoms usually occur over 48 hours due to intake far greater than normal levels and resulting cerebral edema. Often with chronic hyponatremia, central nervous cells have adjusted to the chronic state thus, low sodium is asymptomatic. The main goals are diagnosis and treatment of the primary condition and monitoring of electrolytes. Therapeutic fluid restriction is a low-cost form of treatment but given the low insight and noncompliance in psychiatric patients, it may take several days for an effect to be seen[12].

Atypical antipsychotic agents have been shown in case reports to have some success in alleviating symptoms of PPD. Low-dose risperidone and olanzapine improved polydipsia in a case report[1]. Clozapine has effects in managing PPD but its efficacy remains unproven in large trials[13,14]. Although its doubtful efficacy, the patient presented in the clinical case was medicated with clozapine showing great improvements. Beta blockers such as propranolol have been found to be effective[8]. Another treatment for patients with chronic hyponatremia is demeclocycline, 600 to 1200 mg/d, that directly inhibits ADH action at the distal renal tubules

and reduces urine concentration[8]. Lithium, which works as a direct competitive antagonist of ADH action, is seldom used as it may be potentially nephrotoxic and thyrotoxic.

Another study[15] looked at the use of clonidine and enalapril and found improvement in fluid consumption concluding that medications that affect body water balance might decrease excess fluid intake in some patients with history of water abuse. Irbesartan, an angiotensin receptor blocker, was found to be an effective adjunct in the treatment of PPD in a case report[1]. Lately, newer agents named "aquaretics" that antagonize ADH receptors have been found to increase free water clearance without directly affecting tubular sodium. Conivaptan belongs to this class and has been recently approved by the US Food and Drug Administration for the treatment of euvolemic hyponatremia in inpatients[8].

This case emphasizes the potentially life-threatening effects of psychogenic polydipsia. The phenomenon is not a new one, but this case highlights a different viewpoint questioning whether hyponatremia is an independent result of psychogenic polydipsia, or whether it leads to a vicious circle, resulting in hyponatremia that may cause psychiatric disturbances.

CONCLUSION

Primary polydipsia occurs commonly with schizophrenia and other mental diseases and can cause hyponatremia, with acute cerebral edema, coma, and even death. PPD may present as an acute psychotic state or as inexplicable emergence of seizures.

Appropriate, timely clinical assessment with special attention to thirst, fluid intake, and urine output is essential. Proper treatment may include drug withdrawal, fluid and saline restriction which reverse the electrolyte disturbance. Once corrected, some pharmacological agents can be tried. The current literature is not clear as to which treatment is superior, and numerous agents that have been shown some success treating this disturbance are available. In this patient there was a complete recovery after switching her antipsychotic to clozapine. Importantly, at 12-month follow-up the patient has been free of psychotic and affective symptoms. A combination of behavioral treatments and drugs might be the best approach and has been shown to be effective in the long term, but PPD, has a relapsing course and may reoccur without clinician vigilance and appropriate management.

INDIVIDUAL CONTRIBUTIONS

João Perestrelo – Conception, design and drafting of the article.

Bruno Teixeira – Critical revision of the article and final approval of the version.

CONFLICTS OF INTEREST

No author has a conflict of interest.

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