PSYCOGENIC POLYDIPSIA AND SEVERE HYPONATREMIA IN BIPOLAR AFFECTIVE DISORDER: CASE REPORT

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ABSTRACT

Psychogenic polydipsia, primary polydipsia or potomania is a disorder of multifactorial etiology which is associated with substantial morbidity and mortality. It occurs frequently in patients with psychiatric diseases, particularly those with schizophrenia, however, it is not exclusive, it has been reported in a lower proportion in patients with anxiety disorders and mood disorders. Although, is still poorly understood and therefore underdiagnosed condition.

It is essential to recognize this clinical entity opportunely, due to its potentially serious complications, such as symptomatic hyponatremia that can lead to coma and death if not detected and managed early. Furthermore, the importance of a correct choice of psychotropic medications should be emphasized, since most of the mood stabilizers and some selective serotonin reuptake inhibitors can cause and / or aggravate the hydroelectrolytic disorder, which implies an additional challenge for the specialist in establishing maintenance therapy.

There are few reports in the literature on documented psychogenic polydipsia in mood disorders, for this purpose, we present the case of a patient with bipolar affective disorder suffering from severe hyponatremia secondary to primary polydipsia.

Key words. Psychogenic polydipsia. Hyponatremia. Bipolar affective disorder.

POLIDIPSIA PSICÓGENA E HIPONATREMIA SEVERA EN TRASTORNO AFECTIVO BIPOLAR: INFORME DE CASO

RESUMEN

La polidipsia psicógena, polidipsia primaria o potomanía es un trastorno de origen multifactorial que se asocia con una morbilidad y mortalidad sustancial. Se presenta frecuentemente en pacientes con enfermedades psiquiátricas, particularmente aquellos con esquizofrenia, sin embargo, no es exclusiva de esta, ya que se ha notificado en menor proporción en pacientes con trastornos de ansiedad y trastornos del estado de ánimo. Peso a todo lo anterior, continúa siendo una afección poco comprendida y por ende subdiagnosticada.

Es fundamental reconocer esta entidad clínica de manera oportuna, debido a sus complicaciones potencialmente graves como la hiponatremia sintomática que puede derivar en coma y muerte si no se detecta y se maneja tempranamente. Además, se debe recalcar la importancia de una elección acertada de los psicofármacos, ya que la mayoría de los estabilizantes del afecto y algunos inhibidores selectivos de la recaptación de serotonina, pueden causar y/o agravar dicho trastorno hidroelectrolítico, lo cual implica un desafío adicional para el especialista al momento de establecer la terapia de mantenimiento.

Son escasos los reportes en la literatura sobre polidipsia psicógena documentada en trastornos del estado de ánimo, con este propósito, exponemos el caso de una paciente con trastorno afectivo bipolar que desarrolló hiponatremia severa secundaria a polidipsia primaria.

Palabras clave. Polidipsia psicógena. Hiponatremia. Trastorno afectivo bipolar.

Dear Editor,

Psychogenic polydipsia (PP), primary polydipsia or potomania is a disorder characterized by compulsive water consumption in the absence of an organic disorder[1], it occurs between 6% to 20% of psychiatric patients[2], being more frequent in schizophrenia and rarely documented in patients with mood disorders[3,4].

It is produced by an alteration in the control of thirst unrelated to the production or release of the antidiuretic hormone; and it generally develops in three phases, beginning with polydipsia and polyuria, followed by hyponatremia and finally a manifestation of symptoms such as nausea, vomiting, seizures, coma, and even death[5]; however, symptomatic hyponatremia occurs in only 2% to 5% of these patients[3]. Given the clinical characteristics of this disorder and the

outcomes it can lead to, the knowledge of it is paramount for a timely diagnosis and treatment; we present an illustrative case of a patient with severe hyponatremia secondary to PP in the context of bipolar affective disorder.

CLINICAL CASE

This is a 52-year-old woman, married, a housewife, with a past history of bipolar affective disorder type I diagnosed at the age of 42 and two episodes of mania with psychotic symptoms, due to auditory and complex visual hallucinations and persecution delusions, each of them requiring hospitalization in a psychiatric unit. In the last year, she was under pharmacological treatment with sertraline 50 mg/day, carbamazepine 600 mg/day, quetiapine 100 mg/day and clonazepam 0.5 mg/day but was poorly adherent to the treatment. She did not have other important medical records nor family history of mental illness. The patient went to the emergency department of a tertiary hospital accompanied by her husband and daughter, who describe a clinical presentation that began 5 hours ago characterized by a distrustful attitude, hyperprosexia, hyperkinesia, anxious and cautious mood, delusions of persecution and complex auditory command hallucinations (The patient expressed: «I have to drink water to cleanse my spirit and to stop evil from persecuting me», «The voice tells me to purify myself»). These symptoms led to an increase in water intake of approximately 4 liters in a short period of time, after which, she expressed social withdrawal and mutism; subsequently, she had multiple emetic episodes of water content and finally generalized tonic-clonic seizures that remitted within 3 minutes.

Upon admission to the emergency department, the following vital signs were recorded: blood pressure 103/68 mmHg, heart rate of 123 beats per minute, respiratory rate of 21 breaths per minute, oxygen saturation of 96% and Glasgow score of 9 (Ocular response = 2, Verbal response = 3, Motor response = 4); she was in poor general condition, drowsy, with mild generalized muscular hypertonia and generalized hyporeflexia, no signs of focal neurological damage were found. Her laboratory tests showed a serum sodium of 108 mEq/L, serum chloride 74 mEq/L, serum creatinine 0.32 mg/dL, blood urea nitrogen 4.1 mg/dL, glycemia 131 mg/dL, serum osmolarity 224 mOsm/kg, urinary osmolarity 90 mOsm/kg, normal thyroid function, and a brain computed tomography scan revealing no significant structural abnormalities.

In this clinical context and given the finding of a serum sodium of 108 mEq/L, the diagnosis of acute severe hyponatremia was established. Furthermore, due to the appearance of seizures, it was defined as severely symptomatic. Additionally, the psychiatric semiology described by the family members about the reason for the acute consumption of large amounts of water was key for establishing PP as a diagnostic hypothesis, which was later on confirmed through a serum osmolarity lower than 280 mOsm/kg and a urinary osmolarity less than 100 mOsm/kg.

Rapid replacement of sodium was started by intravenous infusion of 150 ml of hypertonic saline solution (NaCl 3%) with serum control measurements every 20 minutes after subsequent repetition of such infusions until reaching an increase in serum sodium of 5 mEq/L. A maintenance infusion of 3% NaCl was continued until reaching an increase of no more than 10 mEq/L in the first 24 hours, achieving a safety range for such ion on the third day. Moreover, upon admission, the administration of sertraline, carbamazepine, and quetiapine was suspended.

Once the hydroelectrolyte alteration was resolved during the course of hospitalization, the mental examination revealed histrionic behavior, hyperkinesis, hypermimia, emotional lability and psychotic symptoms consisting of grandiose delusions and auditory and visual hallucinations (The patient expressed «I am the embodiment of good», «Look at that angel smiling at me»); she also presented episodes of psychomotor agitation and insomnia, therefore, risperidone and lorazepam were prescribed at initial doses of 1 mg every night and 4 mg/day respectively, in addition to 5 mg of intramuscular olanzapine in case of psychomotor agitation. For 9 days the titration of risperidone and lorazepam was carried out, until complete remission of psychotic symptoms which was achieved at doses of 3 mg/day and 5 mg/day, respectively. Finally, hospital discharge was given, with an outpatient psychiatric follow-up assessment order, adjustment of psychotropic drugs and complementary psychotherapy (cognitive behavioral therapy) if required.

DISCUSSION

PP is a condition that is poorly known and difficult to manage, being more frequent in patients with schizophrenia and rarely documented in patients with mood disorders[3,4]. Its diagnosis is established by identifying excessive water consumption and ruling out other causes of polydipsia, polyuria, and hyponatremia. The latter, is a finding that occurs in about 10% to 20% of patients, being symptomatic between 2% to 5% of cases1 and its presence in the context of PP is confirmed through a serum sodium measurement less than 135 mEq/L and a serum and urinary osmolarity less than 280 mOsm/kg and 100 mOsm/kg respectively[1,6].

Once PP and hyponatraemia as a secondary cause have been confirmed, it is imperative to identify the presence of

severe or moderately severe symptoms (Table 1)[7], since these warrant rapid intervention through intravenous infusion of hypertonic solutions and strict hemodynamic monitoring, as happened in this clinical case; otherwise, the hydroelectrolytic correction may be less aggressive[1,7].

Regarding the psychiatric management, this represents a challenge for the specialist, since many of the available medications can produce symptoms that simulate hyponatremia, such as lithium. Furthermore, medications such as carbamazepine, oxcarbazepine, and valproic acid can exacerbate hyponatremia[7]. It should be clarified, on the other hand, that atypical antipsychotic agents, in addition to their usual effect, have some success in relieving the symptoms of PP, risperidone and olanzapine, for example, have been assessed for this purpose in some clinical reports1. In our case, risperidone associated with lorazepam was used as a complementary therapy for the management of psychomotor agitation and insomnia, with an adequate clinical response and subsequent referral to an outpatient psychiatric care center. However, more studies are required to assess the safety profile and effectiveness of these drugs as an adjunctive therapy in psychogenic polydipsia associated with severe hyponatremia in the context of mood disorders such as bipolar affective disorder.

CONCLUSION

PP is a clinical entity that, although is more frequent in patients with schizophrenia, should not be ruled out in bipolar affective disorder. It requires a high index of suspicion for its clinical recognition and a rapid treatment is essential since it can lead to severe hyponatremia, a life-threatening complication. Because there are few currently published cases about the entities in question, the ideal therapeutic approach is largely unknown and, therefore, more studies are required to characterize the psychotropic drugs that could be used as adjunctive medications. For the moment, it is necessary for the treating physician to perform an early electrolyte correction and recognize the medications that should be discontinued to avoid major complications.

Conflict of interest. The authors declare that they have no conflict of interest.

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ANNEX

Table 1 Classification of hyponatremia symptoms

Severity	Symptoms
Moderately severe	Moderately severe Confusion Headache
Severe	Vomiting Cardiorespiratory distress Abnormal and profound somnolence Seizures Coma (defined as Glasgow ≤8)