

Risperidone Treatment for Polydipsia and Hyponatremia in Schizophrenia: A Case Report



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SUMMARY

Psychogenic polydipsia with associated hyponatremia is a potentially fatal condition observed in patients with chronic psychiatric illness, especially schizophrenia. Recognition and management of this condition are difficult, as patients are uncooperative and secretive about their water intake, but are important in terms of the associated complications. Different strategies, including involuntary fluid restriction and use of various pharmacological agents, such as demeclocycline, propranolol, captopril, and naloxone, have been used for the treatment of this condition with inconsistent results. Antipsychotics have also been used in the treatment of polydipsia; however, their role is not clear as there are reports of antipsychotics both improving and causing polydipsia. Typical antipsychotics have been associated with exacerbation of polydipsia, whereas clozapine has been associated with its improvement. The efficacy of risperidone in the treatment of this condition is controversial, as negative results have been reported. Herein we present a schizophrenia case with polydipsia and hyponatremia that was successfully treated with risperidone.

Keywords: Schizophrenia, risperidone, polydipsia, hyponatremia, antipsychotics

INTRODUCTION

Polydipsia is a common, but often unrecognized potentially dangerous phenomenon in patients with Schizophrenia, especially among those with chronic illness (de Leon et al., 1994). Detection of polydipsia is clinically important, as it can lead to serious complications, such as hyponatremia, resulting in coma and death (Verghese et al., 1996). Typical antipsychotics have been associated with the exacerbation of polydipsia, while clozapine has been associated with its improvement (de Leon et al., 1994). Nonetheless, the role of risperidone and other atypical antipsychotics remains controversial (Kruse et al., 2001; Kar et al., 2002; Bersani et al., 2007). Herein we report a schizophrenia patient with polydipsia and hyponatremia that responded well to risperidone.

CASE REPORT

Mrs. A, a 38-year-old married female that lived in a rural area presented with an 8-year history of illness characterized by delusions of reference and persecution, second-person auditory hallucinations, thought broadcasting, wandering behavior, amotivation, social withdrawal, decreased sleep and appetite, and impaired social and occupational functions. The patient had been treated with antipsychotics by a psychiatrist at a different hospital, but the treatment records were unavailable. She discontinued all medication 6 months earlier and her symptoms exacerbated. Family members also reported that she developed a new symptom during the 6 months she was off medication excessive consumption of water of about 8 L d⁻¹. The patient offered no explanation for this behavior

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and family members were unable to restrict her water intake. She had polyuria, but not nocturia or polyphagia. There were no convulsions reported. There was a family history of depression and diabetes mellitus in 2nd-degree relatives. Her personal and past medical histories were not unusual. Her general physical examination was normal, except for a body mass index of 32 (weight: 75 kg; height: 153 cm). She was diagnosed with paranoid schizophrenia (DSM-IV) and obesity. Electrolyte investigation [obtained values (normal range)] showed that she had low sodium [123.2 mEq L⁻¹ (135-148 mEq L⁻¹)] and chloride [96.2 mEq L⁻¹ (100-108 mEq L⁻¹)], with normal potassium [4.1 mEq L⁻¹ (3.5-5.2 mEq L⁻¹)]. Her lipid profile indicated elevated serum triglycerides [162.5 mg dL⁻¹ (40-150 mg dL⁻¹)] and low HDL [25 mg dL⁻¹ (30 to 60 mg dL⁻¹)], with normal cholesterol, LDL, and VLDL. Her fasting blood sugar, liver, and renal functions were within normal limits. Hemogram showed microcytic anemia. The patient was started on risperidone 6 mg d⁻¹ and trihexyphenidyl 2 mg d⁻¹. Family members were advised to restrict her fluid intake. After 6 weeks of treatment her psychotic symptoms showed significant improvement. In addition, her water intake decreased to about 2 L d⁻¹ and her electrolyte levels were as follows: sodium [138 mEq L⁻¹ (135-148 mEq L⁻¹)], potassium [5.1 mEq L⁻¹ (3.5-5.2 mEq L⁻¹)], and chloride [102 mEq L⁻¹ (100-108 mEq L⁻¹)].

DISCUSSION

The presented patient was consuming about 8 L of water each day and had low sodium, suggesting polydipsia-hyponatremia (de Leon et al., 1994). No other causes of polydipsia, such as diabetes mellitus and tumors, were noted, and she was not on any medication known to cause polydipsia; therefore, we made the diagnosis of primary polydipsia. Polydipsia is consumption of excessive amounts of fluid and is conventionally defined as ≥ 3 L d⁻¹ (de Leon et al., 1994). Polydipsia is a common phenomenon in chronic patients and may be present in as many as 20% of schizophrenic patients (de Leon et al., 1994). Over time, some patients may develop complications secondary to polydipsia, such as hyponatremia. A sudden decrease in the sodium level can result in neurological and psychiatric symptoms, and is known as water intoxication. Over decades, water intoxication can result in physical complications. Despite being a common symptom, the etio-

pathogenesis of polydipsia in psychosis is not well known. Fluid intake and ADH secretion are controlled by the medial temporal lobe, and elevated ADH has been observed during acute exacerbation of psychosis in patients with schizophrenia (Luchins et al., 1997). Thus, hypothalamic and hippocampal disturbances have been suggested (Ferrier, 1985; Luchins, 1990).

Assessment and treatment of polydipsia-hyponatremia is difficult, as most chronic patients are not cooperative with restricted fluid intake and are secretive about their water intake (de Leon et al., 1994). Involuntary fluid restriction is an effective strategy, but requires a highly structured inpatient setting. Different pharmacological agents, including demeclocycline, propranolol, captopril, and naloxone, have been used in the treatment of this condition (Alexander et al., 1991; Goldstein and Folsom, 1991; Nishikawa et al., 1994; Verghese et al., 1996). Antipsychotics have also been used in the treatment of polydipsia; however, their efficacy is not conclusive, as there are both reports that antipsychotics improve and cause polydipsia (Bersani et al., 2007).

Earlier studies on the role of risperidone in treating polydipsia do not report improvement, which is in contrast to the presented case; however, in those studies risperidone was used at higher doses (8-16 mg d⁻¹) (Millson et al., 1996; Kawai et al., 2002). It has been suggested that risperidone might improve polydipsia at lower doses. In fact, improvement of polydipsia with risperidone 6 mg d⁻¹, as in the presented case, was previously reported (Kern et al., 1997). Thus, further research is needed to examine this possible dose-related effect of risperidone.

The following can be construed as limitations of the present study: we did not conduct a water deprivation test, which would have confirmed the diagnosis of primary polydipsia, as it carries an elevated risk of hypotension and tachycardia (Bersani et al., 2007); although the patient had documented evidence of hyponatremia, we did not measure urine osmolality or the urine sodium level; we did not perform brain CT or MRI to rule out brain pathology.

In conclusion, the present study indicates that risperidone might be useful in the treatment of polydipsia-hyponatremia associated with schizophrenia; however, its potential therapeutic role must be confirmed based on systematic evaluation and controlled trials.

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