Introduction

Polydipsia, or the excessive intake of liquids conventionally defined as more than 3 litres per day, occurs frequently in patients with schizophrenia and was first noted by Sleeper. It often represents an under-diagnosed disorder that is difficult to recognize and manage within the clinical milieu. The primary condition of polydipsia is associated with two related phenomena of polyuria and water intoxication. Polydipsia and the development of associated medical complications may be characterized as a three-stage process (i) Simple polydipsia with accompanying polyuria (ii) Water intoxication which occurs after 5-10 years of polydipsia (iii) Physical complications, including irreversible brain damage. Typical antipsychotics have exacerbated polydipsia while clozapine has been associated with its improvement whereas use of risperidone and other atypical antipsychotics remains controversial.

We present a case of a 27-year-old male patient with schizophrenia of 10 years who had polydipsia since 2 years with resulting hyponatremia. The patient was started on oral risperidone (4mg/day) and his water drinking reduced to 13 litres of water per day that was needed for the use of the entire family and stored in a container measuring 15 litres. This was distressing to the patients as this water drinking caused a paucity as their village faced water shortage. The patient though, never claimed to have a craving for drinking water. The patient was admitted and thoroughly investigated to rule out medical complications associated with hyponatremia. Serum electrolytes were tested which only revealed hyponatremia (127 meq/l) whereas serum potassium and chloride were normal. Haemogram, renal and liver function tests, fasting and post prandial blood sugars, lipid profile and MRI brain were normal. Despite drinking 13 litres of water/day the patient had no central nervous system sequelae. We present a case of a young patient with schizophrenia with ECT and risperidone -13 litres of water every day, he did not seem to have any central nervous system sequelae and all his other investigations were normal except for hyponatremia. The patient was started on oral risperidone (4mg/day) and his water drinking reduced but aggression increased. He was given a course of 6 ECTs which further reduced his polydipsia and improved his psychotic symptoms.

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The patient was started on 9 salt capsules each containing 1 gram of salt per day in an attempt to raise the serum sodium levels. Further doses of salt capsules were guided by twice a day serum sodium monitoring. Input output charting and diurnal weight monitoring were done. However the patient continued to drink the same amount of water and would become aggressive and assaultive when prevented from drinking water. The patient was started on oral risperidone (4 mg) and tab. trihexyphenidyl (4 mg) both in divided doses. A week later his water consumption decreased to 8 litres though serum electrolyte disturbances persisted (serum sodium- 125meq/l) . However the patient would become very aggressive towards staff and family when prevented from drinking water. Hence for behaviour control, to curtail water consumption and prevent complications of continued hyponatremia electro convulsive therapy (ECT) was started. The patient was given a course of 6 ECTs. We used brief pulse constant current at pulse width of 1, 70 Hz frequency, bilateral bitemporal, 0.8-1.2 seconds duration and seizure duration lasting from 25 to 30 sec for all the ECTs. The first 3 ECTs were given every alternate day. After the first three ECTs the patient’s water consumption reduced to 5 litres per day, with his serum sodium levels rising to 130 meq/l. The next 3 ECTs were given once in 3 days. After 6 ECTs the patient’s water consumption decreased to 3 litres per day, with serum sodium levels of 142 meq/l and a 60% improvement in the psychotic features, at present maintained on the same dose of risperidone on 3 months follow up.

CASE

A 27-year-old unmarried male was brought by his parents with complaints since 5 years of muttering to self, laughing without reason, becoming suspicious that people were against him and talking in reference to him and hearing unknown voices discussing him. His previous treatment papers from a psychiatrist showed that he was on haloperidol but without significant improvement, though the compliance was poor. Over the past two years he had started drinking around 12-13 litres of water per day that was needed for the use of the entire family and stored in a container measuring 15 litres. This was distressing to the parents as this water drinking caused a paucity as their village faced water shortage. The patient though, never claimed to have a craving for drinking water. The patient was started on oral risperidone (4mg/day) and his water drinking reduced but aggression increased. He was given a course of 6 ECTs which further reduced his polydipsia and improved his psychotic symptoms

ABSTRACT

Psychogenic polydipsia occurs frequently among chronic psychiatric patients, particularly those with schizophrenia. It often represents an under-diagnosed disorder that is difficult to recognize and manage within the clinical milieu. The patient was started on oral risperidone (4mg/day) and his water drinking reduced but aggression increased. He was given a course of 6 ECTs which further reduced his polydipsia and improved his psychotic symptoms.

The patient was admitted and thoroughly investigated to rule out medical complications associated with hyponatremia. Serum electrolytes were tested which only revealed hyponatremia (127 meq/l) whereas serum potassium and chloride were normal. Haemogram, renal and liver function tests, fasting and post prandial blood sugars, lipid profile and MRI brain were normal. Despite drinking 13 litres of water/day the patient had no central nervous system sequelae.

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DISCUSSION

The polydipsia-hyponatraemia syndrome (PHS) occurs in about 5 to 10% of institutionalized, chronically psychotic patients.

KEYWORDS

Psychogenic polydipsia, Hyponatremia, ECT, Risperidone, Schizophrenia
80% of whom have schizophrenia. Complications of PHS include delirium, generalised seizures, coma and death. Our patient also had polydipsia with schizophrenia without any known causes for polydipsia. The etiopathogenesis of polydipsia in schizophrenia is unknown. Assessment and treatment of polydipsia & hyponatremia is difficult as patients do not cooperate for restricted fluid intake and are usually secretive about their water intake. Nonpharmacological interventions include fluid restriction, diurnal bodyweight monitoring, behavioural approaches and supplemental oral sodium chloride administration. We tried involuntary fluid restriction in our patient but he turned aggressive.

Several researchers have tried pharmacological agents like propranolol or naloxone for control of polydipsia. Though antipsychotics have also been used, they may cause or improve the polydipsia. It has been hypothesized that chronic administration of conventional neuroleptics produces D2 receptor supersensitivity that may induce polydipsia through an increase of angiotensin II activity in the central nervous system which is the reason we considered risperidone for our patient.

Low dose risperidone probably reduces psychogenic polydipsia due to its D2 receptor sparing profile. Some researchers found improvement in psychosis and reduction in polydipsic behaviour and diurnal weight gain during 12 weeks of risperidone (6 mg/day) treatment whereas when risperidone was used in higher dose (8-16 mg) no improvement was reported. Several reports showed that polydipsia-hyponatremia improved with antipsychotic therapy when the underlying psychosis improved. Therefore, the reduced polydipsic behaviour with risperidone might be secondary to the psychotic improvement.

This is also in keeping with our observations. The benefits of ECT in psychosis are already known and we also found a beneficial effect of ECT on polydipsia. However we did not find literature for benefits of ECT in alleviating psychogenic polydipsia. The improvement in behaviour with ECTs was responsible for curtailing the water intake, normalising the electrolyte balance as well as improving the psychotic symptoms. Thus our case report has significance for directing psychiatrists to deal with the polydipsia-hyponatremia associated with schizophrenia and avert its dreaded complications. Further research is needed to study the effects of ECT and other atypical antipsychotic medications in ameliorating the symptom of polydipsia in schizophrenia.

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REFERENCES