



AMISULPRIDE IN PSYCHOGENIC POLYDIPSIA

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ABSTRACT

Psychogenic Polydipsia which comprises of episodic life-threatening water intoxication, remains an important clinical dilemma for a significant number of patients with schizophrenia. The condition is associated with increased morbidity and mortality from a number of causes. Recognition and management of this condition is difficult, as patients are uncooperative and secretive about their water intake. Nonetheless, it is important and critical to address it in lieu of the associated complications. 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, paradoxically, causing polydipsia in certain patients. Typical antipsychotics have been associated with exacerbation of polydipsia, whereas clozapine has been associated with improvement. Here, we present a case of schizophrenia with psychogenic polydipsia and resultant hyponatremia which did not respond to treatment with clozapine, which is the preferred drug in the given condition, but it improved rather well with the second generation antipsychotic, amisulpride.

KEYWORDS: Schizophrenia, Psychogenic Polydipsia, Hyponatremia, Clozapine, Amisulpride.

INTRODUCTION

Polydipsia is a condition wherein there is intake of more than 5 litres of water per day. The syndrome is sometimes called "compulsive water drinking," "psychogenic polydipsia," and "self-induced water intoxication." Although the underlying pathophysiology of the syndrome is unclear, several factors have been implicated in inducing polydipsia and symptomatic hyponatremia. The illness generally develops in three phases, beginning with polydipsia and polyuria, followed by hyponatremia and finally water intoxication. Hyponatremia occurs due to water retention, as the kidneys fail to excrete the excess fluid, resulting in low sodium serum values. Of those suffering from primary polydipsia, only about one-half of patients have intermittent hyponatremia, others may have water intoxication as a result of temporary retention. Water intoxication may manifest itself by a worsening of psychiatric symptoms, nausea, vomiting, delirium, ataxia, seizures, and coma, and may even be fatal. Polydipsia in psychiatry patients is seen in patients with chronic schizophrenia with long-term hospitalization.^[5]

CASE REPORT

A 38-year-old male, presented in the psychiatry OPD with complaints of withdrawn behaviour, decreased communication, muttering/gesticulating to self, auditory hallucinations, oddities of behaviour in the form of spitting, irritability, agitation for the past 9 years, following a stressor. He was treated, during his multiple hospitalisations, with psychotropics and ECTs, with history of poor compliance. Patient also had history of excessive water consumption (around 10-15 litres/day) since 6 months, with confusion and disorientation since 1 month.

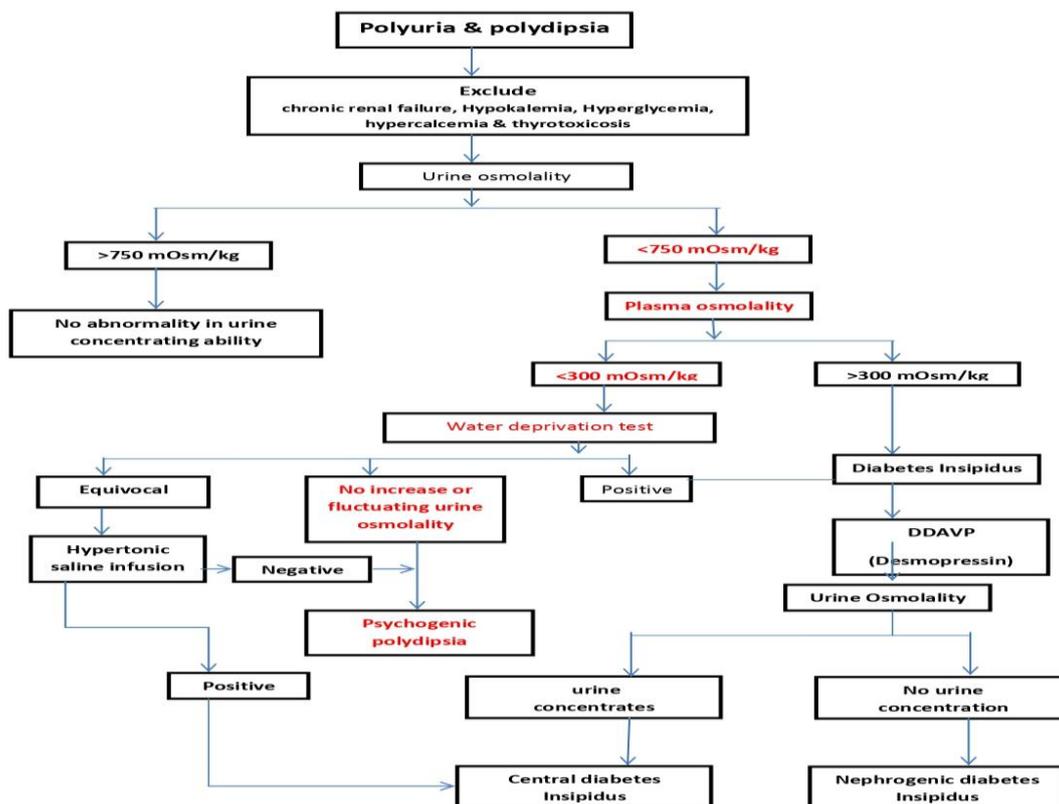
On detailed investigation, value of serum sodium was **126** (Normal value = 135-145mEq/L), serum osmolality was **277** (285-295mmol/kg) and urine osmolality **74** (300-900mOsm/kg). His other investigations, such as complete hemogram, thyroid function tests, liver function and renal function tests, potassium, chloride, calcium, magnesium, ammonia, blood sugar, ADH, Aldosterone were found to be normal. All his radiological investigations like CT scan brain, MRI brain (coned down view of pituitary) USG abdomen, were also normal. These findings rule out the possibility of

organicity and patient was managed on the line of psychogenic polydipsia.

Subsequently, with restriction of water, the patient's confusion resolved, and all his investigations turned out normal except urine osmolality which was persistently low. Patient was started on clozapine 12.5mg along with risperidone, and his fluid intake was restricted to 2-3 litres/day. But with increment of dose of clozapine, patient again started manifesting confusion. Clozapine had been tried twice, but on each occasion, the patient developed confusion. Hence, clozapine was discontinued and the patient was started on amisulpride (50mg), as patient also had predominant negative symptoms. The dose of amisulpride was gradually increased to 150mg and patient showed noticeable improvement. Behaviour therapy in the form of restricted water drinking with half litre bottle every 2 hours was planned. His father was advised to keep a record of intake and output of fluids. The family members as well as the patient were psycho-educated regarding effects of increased water intake and the need to restrict water intake. His water consumption was maintained at 4-5 litres/day from 10-15 litres/day.

DISCUSSION

On account of the diverse manifestations of psychogenic polydipsia, establishing its clinical diagnosis can become a challenging task. The differentials include diabetes insipidus, which could be central and nephrogenic. Central Diabetes Insipidus occurs when there is not enough AVP (Arginine Vasopressin) in the body to regulate urine production whereas in Nephrogenic Diabetes Insipidus, AVP level is adequate but kidneys fail to respond to it.^[3,5] Apart from an elaborate and detailed history, clinical investigations can aid in establishing diagnosis. Urine osmolality plays an important role here. Other investigations include water deprivation test (no increase or fluctuating urine osmolality indicates psychogenic polydipsia) and desmopressin test (differentiate types of diabetes insipidus on the basis of ability of urine concentration). Approach to a case of polydipsia consists of the following.^[8,9]



Antipsychotics have been routinely used in the treatment of polydipsia; however, their role is not very clear, as there are conflicting reports of antipsychotics both improving and causing polydipsia.^[2,4] Clozapine has a corrective and stabilizing effect on psychogenic polydipsia with hyponatremia in chronic schizophrenia, but it may not be effective for all the patients. In such circumstances, other second generation antipsychotic

like Amisulpride are associated with diminution of symptoms and improvement of patient, though the exact mechanism is still unclear.

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