

CASE REPORT

A Case of Unrecognized Psychogenic Polydipsia

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ABSTRACT

Psychogenic polydipsia is prevalent among people with schizophrenia. Although its pathophysiology is relatively unknown, it causes life threatening complications due to acute or severe hyponatraemia.. This report illustrates a patient with schizophrenia who had unrecognized psychogenic polydipsia and developed severe complication. It also highlights the clinical salience of its management.

Keywords: Psychogenic polydipsia, Schizophrenia, Hyponatraemia

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INTRODUCTION

Psychogenic polydipsia is characterized by a compulsion to seek out or drink water excessively and is most commonly found among patients with chronic schizophrenia (1). Apart from that, it is also associated with mood disorders, intellectual disability, anxiety disorder and personality disorders (1). The prevalence was reported to be as high as 6 to 20% and often resulted in hyponatraemia which subsequently put patients at risk of having seizure or other life threatening complications (2). Many of the cases go unrecognized because it receives less attention during clinical care in comparison to the core symptomatology of the psychiatric illness such as thought or perceptual disturbance.

Various mechanisms have been postulated in the pathophysiology of psychogenic polydipsia, including uncontrolled positive symptoms, compulsive behaviour, stress reduction, anticholinergic effect of psychotropic drugs, neuroleptic-induced dopamine supersensitivity within the hypothalamic-pituitary axis, and even a resetting of the osmoreceptors in the central nervous system due to elevated dopamine levels (3).

Its clinical presentation often starts off with dilution of serum sodium in the body in response to the excessive water intake and results in hyponatraemia. Excessive water intake raises the pressure of the extracellular medium and reduces the antidiuretic hormone (ADH) level. Gradual decline of sodium serum level which usually occurs in chronic hyponatraemia maybe asymptomatic but a rapid decline in an acute case

of hyponatraemia may cause complication such as cerebral oedema. This in turn would be translated into clinical signs and symptoms such as nausea, vomiting, impaired mental function, seizures or coma. Hence, it is important for mental health professionals to be aware of the presence of psychogenic polydipsia among patients with severe mental illness.

CASE REPORT

We report a 49-year-old man who had been treated for schizophrenia for more than 20 years and was a chronic active tobacco smoker. He first presented to psychiatric service at the age of 26 years old with a history of behavioural change, auditory hallucination, delusion of control and subsequently became socially withdrawn and had significant impairment of social functioning. However, there was problem of non-compliance to the medication due to tolerability issues such as somnolence and feeling weak. He was then put on long-acting injectable antipsychotic, but his symptoms persisted hence the possibility of treatment-resistant schizophrenia was entertained. But patient and his family refused the suggestion of clozapine. Therefore, he was just maintained on the depot injections.

While he was treated as such, a year prior to this report, the patient developed a sudden onset of generalized tonic-clonic seizure with disturbed consciousness and post-ictal drowsiness. But he was haemodynamically stable and afebrile. During his initial hospitalization, all other causes of seizure had been excluded except for the biochemical abnormalities. With a baseline of sodium level between 138 to 149 mmol/L, his serum electrolyte investigations showed that there was hyponatraemia (in the range of 117 - 120 mmol/L) and hypochloraemia (in the range of 87 - 91 mmol/L). Further results [obtained values (normal range)] showed low serum osmolality

[268 mOsm/kg (275-295 mOsm/kg)], low urine osmolality [158 mOsm/kg (300-900 mOsm/kg)] and a urine sodium of 13 mmol/L.

Despite having the cause of his seizure identified and the electrolyte imbalance corrected before discharge, unfortunately there was no specific management rendered to explore the contributing factor for the hyponatraemia. This led to the patient suffering from another 3 similar episodes of seizures which required admissions, in every 4 to 5 months since its first onset. During his final hospitalization, a careful corroborative history from his family members finally revealed that he had started to drink water excessively for the last three years prior to the onset of the seizure. He was reported to consume water about 5 to 7.5 litres in a day and he had difficulty to withhold the compulsion to drink excessively. As a result, he was noted to have polyuria, frequent complaints of headache and feel increasingly lethargic each time prior to the onset of the seizures.

Since the identification of psychogenic polydipsia, his family members were advised on measures of monitoring and restricting his excessive water consumption at home. This had prevented future episodes of seizure successfully. It was also worth mentioning that throughout his hospitalizations, while there was a need to correct the severe hyponatraemia, careful measure was taken not to do it aggressively to prevent central pontine myelinolysis, which can result in further mental status changes and would be mistaken for worsening of the psychiatric illness.

DISCUSSION

Polydipsia, a word derived from the Greek (polys - "much, many" and dipsa - "thirst") by its simplest meaning, a symptom of excessive thirst leading one to consume more than usual amount of water (objectively, it is more than 5L per day) (3). It can be due to various causes such as diabetes mellitus, diabetes insipidus, as well as any conditions that create a water deficit state within the body. In the event of psychogenic polydipsia, diagnosis is one of exclusion. Thus, it is of paramount importance to first exclude any of these potential general medical illnesses before coming to the conclusion that it has a psychological basis. This was essentially the first most important step of coming to the diagnosis in our patient who had an underlying psychotic illness.

Psychogenic polydipsia is a recognized condition among psychiatric patients, with the first case report in a patient with schizophrenia since 1938 (4). The condition is prevalent with as high as 20% of psychiatric inpatients, mainly those suffer from schizophrenia, were reported to experience polydipsia (5). Other psychiatric illnesses that were associated with psychogenic polydipsia include major depression or bipolar disorders. There are several risk factors in patients with psychiatric illness that

were reported to be associated with polydipsia which include prolonged hospitalization, lifelong history of excessive drinking habit, side effects of psychotropic medications such as dry mouth or thirst, and heavy tobacco smoking.. We postulated that the latter two factors could have been related to the occurrence of psychogenic polydipsia in our patient, as it involved the ADH-releasing effect of nicotine and compensatory drinking due to the anticholinergic side effects.

In the absence of all other potential causes of excessive thirst, the ultimate key of its diagnosis is by maintaining a high index of suspicion of psychogenic polydipsia whenever there is asymptomatic hyponatraemia. Often, dilutional hyponatraemia has varied clinical presentations ranging from non-specific symptoms such as lassitude, headache, nausea, vomiting to profound neurological symptoms like delirium, convulsion and comatose. It can also lead to unusual or worsening of the psychiatric symptoms, concealing a promptly detection and resulting in delayed treatment.

At present, there are no established clinical guidelines in the management of psychogenic polydipsia. The approach derives from best practice and expert opinions which focus on water restriction via behavioural and / or pharmacological therapy. Nevertheless, restricting excessive water intake can be challenging among schizophrenia patients that are still symptomatic. Hence, the suitable approach requires different strategies involving good family support for constant monitoring, oral sodium supplementation, discontinuation of offending agent, switching to another class of neuroleptic at the minimum effective dose with frequent monitoring of serum electrolytes, and even promptly management of smoking habit to prevent exogenous stimulation of antidiuretic hormone release. Our patient had responded to measure of a more vigilant monitoring of his water consumption at home. This showed that psychogenic polydipsia can be intervened with an early detection.

CONCLUSION

Promoting awareness about psychogenic polydipsia among mental health care professionals is important because it is potentially treatable and reversible.

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