Case Report

A case of Psychogenic Polydipsia

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Abstract

Psychogenic polydipsia (PPD) is characterised by excessive fluid intake in the absence of physiological stimuli to drink. We report a case of a 28 year old male who was admitted with complaints of palpitations and increased urine output for the past 10 months. Earlier investigations at other centres reported a water intake of 13L/day which was not associated with thirst. No significant family and personal history was associated. Although the clinical picture and laboratory tests produce some diagnostic certainty, water deprivation test was done to confirm primary polydipsia. He was diagnosed as having psychogenic polydipsia by exclusion of the other causes and he was managed with fluid restriction, behavioural and pharmacological therapy to prevent the development of hyponatremia. We report this rare case in view of its potential interest and we also review the literature regarding psychogenic polydipsia

Keywords: hyponatremia, polydipsia, water deprivation test

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Introduction

Primary polydipsia is a form of polydipsia which is characterized by excessive fluid intake in the absence of physiological stimuli to drink.[1] This includes psychogenic polydipsia (PPD), which is caused by psychiatric disorders, often schizophrenia, and often accompanied by the sensation of dry mouth.[1]Some forms of primary polydipsia are explicitly non-psychogenic. Primary polydipsia is a diagnosis of exclusion.[1]

Case Presentation

A 28 year old unmarried male electrician came to the outpatient department with complaints of palpitations and increased urine output for the past 10 months. There was no history of thirst. The patient gave history of associated excessive water intake of about 13 L/day. There was no history of head injury, headache, decreased vision, numbness of extremities, drug intake, abdominal pain or chest pain. No significant family and personal history associated.

On examination, patient was conscious, oriented, afebrile and anxious. The findings of general and systemic examinations were normal with blood pressure of 130/80 mm of Hg. The complete blood count showed the following: Total WBC count = 7400/mm,^{3} Hb = 15g/dl, and platelet count = 2.5 lakhs/mm^3 of blood. The liver function tests and thyroid function tests were normal. Although the clinical picture and laboratory tests such as urine osmolality (40 mOsm/L), urine output (12 L/day), electrolytes (sodium = 119 mEq/L, potassium = 4mEq/L) and serum osmolality (260 mOsm/L) showed some diagnostic certainty, water deprivation test was done to confirm primary polydipsia which showed increased urine osmolality after water restriction on adding vasopressin. Ultrasonography of the abdomen and CT brain showed no abnormal

findings. Fasting and postprandial blood sugar values were normal.

The patient underwent a detailed evaluation to rule out associated psychiatric disorders and was managed with fluid restriction, behavioural and pharmacological therapy.

Discussion

This case report briefly highlights the pathophysiology, clinical presentation, difficulty in diagnosis and management of primary polydipsia. The pathophysiology of this syndrome is unclear and multifactorial. Primary polydipsia can be divided into three subcategories.[2] The dipsogenic subtype is characterized by inappropriate thirst and occurs in association with multifocal diseases of the brain such as neurosarcoid, tuberculous meningitis and multiple sclerosis.[2] The psychogenic subtype is not associated with thirst, but is a feature of psychosis or obsessive compulsive disorder.[2] The iatrogenic subtype results from recommended increase in fluid intake for health benefits.[2] Overall, PPD with symptomatic hyponatremia occurs in only 2% to 5% of patients with schizophrenia.[3] PPD has also been associated with affective disorders, anorexia nervosa and personality disorders.[3]
Some studies suggest a higher prevalence of PPD in women. Polydipsia affects all races.

The symptoms and signs of primary polydipsia include excessive thirst, xerostomia, leading to overconsumption of water, hyponatremia and hypervolemia, leading to edema, hypertension and weight gain. Behavioural changes include seeking fluids from any available source.[4] Hyponatremia can progress to water intoxication which presents as cerebral edema, confusion, lethargy, psychosis, central pontine myelinolysis, seizures and death.[4] This syndrome is also called compulsive water drinking, psychosis-intermittent hyponatremia-polydipsia (PIP) syndrome and self induced water intoxication.[5]

The differential diagnoses included primary (psychogenic) polydipsia, diabetes insipidus and osmotic diuresis. The diagnosis of psychogenic polydipsia (PPD) is one of exclusion.[2] It requires a detailed evaluation for other causes of polydipsia, polyuria, hyponatremia, and the syndrome of inappropriate ADH secretion.[2] Tests such as plasma and urine osmolality, complete serum electrolytes, metabolic panel, urea and urine sodium typically show euvolaemic hyponatremia, low serum osmolality and reduced urinary osmolality.[2] Water deprivation test is the definitive test for diagnosis although the clinical picture and laboratory tests produce some diagnostic certainty, often coupled with a test of urine-concentrating ability in response to exogenous vasopressin.[6] In PPD, the urine is very dilute before water is restricted (<100 mOsm/kg H_2O). When vasopressin is administered after water restriction, urine osmolality of >750 mOsm/kg H_2O is diagnostic of PPD.[6] Chronic PPD patients not be able to concentrate urine maximally following the water deprivation test due to medullary gradient washout and down regulation of ADH release.[6] Measurement of plasma ADH is required in case of equivocal results; the plasma ADH is low before water restriction in PPD.[6] Water deprivation test in this case showed increased urine osmolarity after water restriction which excluded Diabetes Insipidus.

Management of PPD includes fluid restriction, behavioural and pharmacological therapy.[7] Serum sodium should be monitored vigilantly and frequently with judicious use of hypertonic saline (3%) to treat symptomatic or severe hyponatremia.[7] Fluid restriction of 1 to 1.5 L/day is started in patients with chronic symptoms and no or mild hyponatremia.[7] Patients should receive education about the free water content of foods and the importance of limiting fluid intake.[7] Non pharmacological behavioral therapy includes behavioral intervention and behavioral modification.[8] The symptoms of PPD are improved by prescribing atypical antipsychotics that are frequently used for schizophrenia, bipolar disorder, major depressive disorders and other psychiatric disorders.[8] Complications such as bladder dilation, rhabdomyolysis which can progress to hydronephrosis, renal failure, enuresis, congestive heart failure and osteoporosis result as a long-term consequence of polydipsia.[9]

Conclusion

This case report highlights the difficulties in diagnosing psychogenic polydipsia, its pathogenesis and the various treatment options available. Though psychogenic polydipsia appears relatively rare, it still needs to be considered in the differential diagnosis of primary polydipsia.

Acknowledgment: Nil

Conflicts of interest: Nil

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