Case Report

Converging Neurobiological Evidence In Primary Polydipsia Resembling Obsessive-Compulsive Disorder

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ABSTRACT

Compulsive water drinking can have phenomenological and pharmacotherapeutic similarities with obsessive-compulsive disorder (OCD). Substantiating neurobiological evidence is lacking for such an association. We report a patient who was referred with a diagnosis of primary polydipsia with no signs of organic pathology in structural neuroimaging. However, positron emission tomography revealed basal ganglia hypometabolism indicating that primary polydipsia with compulsive water drinking is neurobiologically related to OCD. The diagnostic complexities displayed by primary polydipsia and the use of systematic evaluation with supporting neuroimaging evidence in reaching a reliable diagnosis are discussed. The neurobiological evidence will foster the treatment decisions for starting anti-OCD measures when clinicians encounter patients with primary polydipsia exhibiting compulsive patterns of drinking. Nevertheless, such findings need to be replicated in future studies with a larger sample size.

Key words: *Compulsive drinking, neurobiology, neuroimaging, obsessive-compulsive disorder, primary polydipsia*

INTRODUCTION

Primary polydipsia is characterized by excessive thirst and compulsive water drinking in large amounts without any specific identifiable etiology.[1,2] Among psychiatric conditions, schizophrenia is known to have a greater association with polydipsia up to 25%.[1-3] Nevertheless, studies have argued about the behavioral resemblance between the compulsive water drinking of primary polydipsia and compulsive acts of obsessive-compulsive disorder (OCD).[4] Further, primary polydipsia has responded to pharmacological agents such as selective serotonin reuptake inhibitors (SSRIs) indicated in OCD raising questions on a common neurobiological pathogenesis between

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the two conditions.[2,4,5] Many studies regarding the neurobiology of OCD have established that an aberrant cortico-striato-thalamo-cortical circuitry underlies the pathophysiology of OCD apart from the involvement of the cerebellum and parietal cortex.[6,7] Although studies report the phenomenological and pharmacological similarities between primary polydipsia and OCD, there is a lack of understanding of the neurobiological links between these conditions. We report a patient with primary polydipsia who not only had a phenomenological resemblance to the compulsive behaviors of OCD but also had the objective evidence of basal ganglia abnormalities. We also highlight how neurobiological evidence facilitated the diagnostic and therapeutic process leading to adequate symptom control.

CASE REPORT

A 27-year-old married woman with no past medical or psychiatric illness was admitted to the emergency medical services after being rescued following attempted hanging. Her Glasgow Coma Scale was 'four' (E1V1M2). Computerized tomography of the brain showed diffuse cerebral edema suggestive of hypoxic brain injury. In the Intensive Care Unit, she developed ventilator-associated pneumonia followed by delirium which was treated with haloperidol (2.5 mg/day for 10 days). The delirium subsided in 2 weeks. Magnetic resonance imaging (MRI) of the brain revealed no structural abnormalities at that time. After discharge, over 6 months, she developed progressive memory impairment, poor concentration, stuttered speech, impulsivity, emotional lability, and an unstable gait. Further, she exhibited odd behaviors such as consuming frequent gulps of water (12–14 L/day) with consequent increased frequency of micturition (20–25 times in the day; 8–10 times in the night). Initially, her mother thought the behavior was due to increased thirst. However, the acts persisted despite attempts to control. The history was negative for meningitis, raised intracranial pressure, radiation exposure, preexisting diabetes mellitus, and cardiac or renal illnesses. Her family history was nil contributory. Her physician referred her to an endocrinologist for evaluation of polydipsia. The endocrinologist worked up for diabetes insipidus (DI) and primary polydipsia. Her normal renal function tests ruled out nephrogenic causes of DI [Table 1]. Later, the patient was subjected to 7-h "water deprivation test" and was administered vasopressin. The results ruled out pituitary DI corroborated by a repeat MRI of the brain which was normal [Table 2]. Further, an average serum cortisol after corticotrophin stimulation test ruled out hypothalamic-pituitary-adrenal axis dysfunction [Table 1]. Subsequently, the patient was referred to psychiatric services with a diagnosis of psychogenic polydipsia.

Table 1: Blood and urine biochemistry of the patient

Parameter	Patient value	Normal
Hemoglobin (g/dl)	8.0	12-15.5
Total white blood cell count (/μl)	9150	4500-10,000
Mean corpuscular volume (fl/red cell)	68.7	80-96
Erythrocyte sedimentation rate (mm/h)	45	0-29
Peripheral smear	Microcytic hypochromic anemia	
Blood urea (mg/dl)	14	15-40
Serum creatinine (mg/dl)	0.8	0.7-1.2
Serum sodium (mEq/L)	140	135-145
Serum potassium (mEq/L)	4.6	3.5-5
Serum calcium (mg/dl)	9.3	9-11
Serum phosphorous (mg/dl)	3.2	2.5-4.5
Serum magnesium (mg/dl)	2.5	1.8-3
Serum bilirubin	0.7	0.4-1.2
AST (IU/L)	34	0-40
ALT (IU/L)	53	0-45
ALP (IU/L)	306	30-125
Total protein (g/dl)	7.2	6.3-8.3
Serum albumin (g/dl)	4.5	3.5-5.5
Urine routine microscopy (pus cells)	5-10	0-6
Urine culture and sensitivity	Sterile	
Urine specific gravity	1.007	1.005-1.030
Endocrine work-up		
Fasting blood glucose (mg/dl)	84	<108
Postprandial blood glucose (mg/dl)	134	<140
Serum basal cortisol (mcg/dl)	6.67	7-28
Serum cortisol after ACTH stimulation* (synacthen 1 mcg) (mcg/dl)	0 min: 8.33 30 min: 17.50 60 min: 10.63	
Thyroid-stimulating hormone (μIU/ml)	2.38	0.4-4.2
Serum prolactin (ng/ml)	9.37	2-29
Luteinizing hormone (mIU/ml)	9.99	1.9-12.5
Follicle-stimulating hormone (mIU/ml)	6.65	3.1-7.9
Serum testosterone (ng/dl)	35.39	15-70†

*Values within normal range indicate intact hypothalamic-pituitary-adrenal axis; †Normal range for females. ACTH – Adrenocorticotrophin hormone; AST – Aspartate transaminase; ALT – Alanine transaminase; ALP – Alkaline phosphatase

During the detailed evaluation, the patient reported that even without thirst, she had a strong desire to consume water. She could not elaborate on its repetitiveness, irrationality, or ego-dystonicity. There were no mood or psychotic symptoms. However, her mother recalled that she would become restless and fidgety before drinking water and appear relieved after consuming the same. She added that restricting her from doing so would worsen her restlessness or make her angry. No obsessions or compulsions were elicited in the Yale-Brown Obsessive-Compulsive Disorders Scale (Y-BOCS) checklist. A functional neuroimaging was ordered to identify the neural

Table 2: Water deprivation test

Time	Serum osmolality (mOsm/kg)	Urine osmolality (mOsm/kg)	Serum sodium (mEq/L)	Body weight (kg)	Blood pressure (mmHg)
Basal (6 am)	291	261	147	55.9	110/80
The patient underwent 7 h of water deprivation (total urine output during water deprivation was 400 ml; the patient unable to continue fasting beyond 7 h); vasopressin was administered to diagnose either pituitary or nephrogenic DI*					
Before vasopressin 5 IU (1.00 pm)	283	411	148	55.5	108/80
After vasopressin 5 IU (2.00 pm)	278	481	142	55.4	110/80
Result	As there is <50% increase in urine osmolality after vasopressin administration, pituitary DI is ruled out. Possible nephrogenic causes of DI can be considered				

*After administration of vasopressin, an increase of >50% in urine osmolality suggests pituitary DI. Whereas an absent or smaller increase is suggestive of nephrogenic DI. DI – Diabetes insipidus

dysfunction, if any. Fluorodeoxyglucose-positron emission tomography (FDG-PET) of the brain showed global hypometabolism due to ischemic injury especially the basal ganglia and the cerebellum sparing the visual cortex. The basal ganglia dysfunction in FDG-PET pointed toward an OC spectrum disorder possibly due to the hypoxic-ischemic brain injury. After 6 weeks of adequate trial of escitalopram (5–20 mg/day), her compulsive acts improved significantly. The Y-BOCS scale was used to assess the distressful symptoms objectively factoring both the patient and her caregiver's reports. The clinical improvement was reflected in Y-BOCS scores (Baseline = 25 [severe], after 6 weeks = 7 [subclinical]). There were no major exacerbations during the 1-year follow-up period, and the SSRI was continued for prophylaxis. Informed consent was obtained from the patient and her caregiver for the collection of information, treatment procedures, and write-up of this report.

DISCUSSION

Primary polydipsia, when underdiagnosed, can lead to dangerous complications due to water intoxication.[8,9] Amelioration of the distressful behaviors requires establishing a reliable diagnosis and initiating prompt treatment. The treating team experienced significant hardships in diagnosis due to the ambiguity overlying the symptom presentation. A patient's report of intrusiveness, ego-dystonicity, and resistive attempts hold the key to diagnosing OCD as per classificatory systems in psychiatry. Nonreporting of such factors by our patient could be explained by an illness indifference commonly seen in OCD patients with an organic etiology – the hypoxic brain injury in our patient.[10] Concurring reports in the literature have revealed that traumatic brain injury can predispose to polydipsia.[11] Although the compulsive drinking pattern and the associated distress pointed toward an OC spectrum disorder, the FDG-PET revealed convincing evidence of basal ganglia dysfunction which facilitated the diagnostic and therapeutic process. Until recently, studies have quoted phenomenological and pharmacotherapeutic similarities between primary polydipsia and OCD.[2,4,5] There has been limited neurobiological understanding of OC-like features of polydipsia seen in nonpsychotic patients in contrast to the well-replicated findings of increased ventricular volumes of patients with schizophrenia and polydipsia.[12] Our report adds neurobiological evidence of basal ganglia dysfunction in SSRI responsive primary polydipsia similar to that implicated in the pathogenesis of OCD.[6,7,13] To conclude, primary polydipsia cases referred to psychiatric services should undergo systematic evaluation with rational use of neuroimaging exploring the problem behaviors and overcoming diagnostic hurdles. Even in resource-limited settings, when psychiatrists encounter primary polydipsia associated with compulsive symptoms, a trial of SSRI will be worthwhile and can prevent dangerous complications. Nevertheless, long-term, adequately powered studies are required to substantiate the neurobiological observations made in patients with primary polydipsia.

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Conflicts of interest

There are no conflicts of interest.

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