Case report / Olgu sunumu

Co-occurrence of primary polydipsia and bipolar disorder: can it be a sign of HPA axis dysfunction?

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ABSTRACT

Primary polydipsia (PP) is an etiologically unclear condition that characterized by increased thirst and excessive fluid intake with greater than 3L per day and which usually is coincided in patients with psychiatric disorders. The most commonly reported psychiatric disorder is schizophrenia; however, bipolar disorder is also infrequently described in concurrence. It is remarkable to note that PP could be life-threatening owing to hyponatremia which may lead seizures, cerebral edema, cardiac arrest and coma; thereby, diagnosing and treating PP are crucial. We report a case with a diagnosis of bipolar disorder, currently on remission with the treatment of lithium, presented with 5-month-history of increased water intake. A thorough clinical and diagnostic assessment revealed that patient had not any medical condition which could lead to polydipsia. It is argued that dysregulation of hypothalamic-pituitary-adrenal (HPA) axis could be the underlying reason of affective disorders and the mechanism which may lead to PP. Despite the cause of polydipsia remains unclear, we aimed to provide evidence for the associative role of HPA axis dysfunction in PP comorbid bipolar disorder. ***(Anatolian Journal of Psychiatry 2017; 18(Suppl.1):8-10)***

Keywords: *bipolar disorder, HPA axis, polydipsia*

Birincil polidipsi ve bipolar bozukluk birlikteliği: HPA aks disfonksiyonunun işareti olabilir mi?

ÖZ

Birincil polidipsi, artmış susama hissi ve 3 lt/günden fazla sıvı alımı ile karakterize, genellikle psikiyatrik hastalarda rastlanan ve etiyolojisi net olmayan bir durumdur. En fazla eşlik eden psikiyatrik bozukluk şizofreni olarak bildirilmiş olup bipolar bozuklukla birlikteliği nadir olarak gösterilmiştir. Hiponatremiye bağlı konvülziyon, serebral ödem, kardiyak arrest ve koma gibi yaşamsal tehlike oluşturan komplikasyonlara yol açmasından dolayı birincil polidipsinin tanısı ve tedavisi klinik önem taşımaktadır. Bu yazıda, bipolar bozukluğu olan, lityum tedavisi altında halen remisyondaki, beş ay önce başlayan artmış su alımı olan ve tanıya yönelik kapsamlı klinik incelemenin sonucunda polidipsiye yol açabilecek herhangi bir tıbbi durum saptanmayan bir olgu bildirilmiştir. Birincil polidipsinin etiyolojisi belirsizliğini korumakla birlikte, hipotalamik-pitüiter-adrenal (HPA) aks düzensizliğinin bipolar bozukluk ve birincil polidipsiye yol açmasının olası düzenek olabileceği öne sürülmüş ve bu olgu sunumunda iki bozukluğun birlikteliğinde HPA aks düzensizliğinin rol oynayabileceği görüşüne katkı sağlamak amaçlanmış ve tartışılmıştır. ***(Anadolu Psikiyatri Derg 2017; 18(Ek.1):8-10)***

Anahtar sözcükler: *Bipolar bozukluk, HPA aksı, polidipsi*

INTRODUCTION

Primary polydipsia (PP) is a clinical entity, characterized by excessive water intake and frequent occurrence among individuals with mental disorders. Estimated prevalence of PP is 3-17% in psychiatry patients and more likely to be seen in schizophrenia approximately 80%.[1-3] Hyponatremia is the most threatening complication of PP which may lead to coma and death, there-

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fore diagnosing polydipsia is clinically substantial.[4,5] Most cases of PP have been reported in patients with schizophrenia; however, it is rarely reported in those ith affective disorders.[6,7] Here we report an unusual bipolar disorder case with polydipsia which has occured after his last manic episode and organic etiology remained unexplained through all diagnostic examinations.

CASE

Our patient, 28-year-old single and engineer male, with the three-year diagnosis of bipolar disorder, presented with 5-month-history of increased water intake, around 12-15 L/day. He had a history of three episodes of mania and two of depression in the past. His last manic episode was registered five months ago. Although remission was supplied with lithium 2100 mg/day and paliperidone 6 mg/day treatment, excessive water intake has occurred nearly five months ago and proceeded. He admitted our outpatient clinic for a follow-up and on his psychiatric examination he appeared his age and self-care was normal. Cooperation and orientation were intact. Affect was appropriate while mood was euthymic. Psychomotor activity was normal as speech output was coherent and relevant. Psychotic or obsessive compulsive symptoms were not determined. There was no history of substance or alcohol use. His current Young Mania Rating Scale and Hamilton Rating Scale for Depression scores were 2 and 1 respectively. His antipsychotic treatment with paliperidone has tapered off and he was on remission currently with treatment of lithium 2100 mg daily. Beside use of lithium, the patient did not give any reason for the polydipsia. He had not have any medical condition. He had polyuria but not nocturia or convulsions. There were no signs of altered sensorium. His vital signs were stable. He did not show significant decrease in sodium levels (139 mEq/L). Renal function tests was in normal range (BUN: 10 mg/dl and creatinine: 0.71). His liver and thyroid functions were normal while no electrolyte imbalance was detected. Diurnal urine density screening did not support water intoxication. Water deprivation test and intranasal 20 mcg desmopressin were performed and did not confirmed central or nephrogenic diabetes insipidus. Magnetic resonance imaging of the brain did not show any significant structural abnormalities. We diagnosed him remitted bipolar disorder with comorbid psychogenic polydipsia. Lithium regime was not considered to change due to his clinical remission and irrelevant for PP etiology in our patient. We planned to add quetiapine 150 mg/day and aimed to potentiate to 300 mg/day on his treatment. Furthermore, close follow-ups with endocrinology and nephrology consultations were offered.

DISCUSSION

The diagnosis of PP requires the presence of a psychiatric disorder and the exclusion of all known causes of polydipsia. A thorough physical investigation did not confirm any known somatic disease. Central diabetes insipidus and nephrogenic diabetes were actually excluded through laboratory findings after water deprivation test and intranasal administration of desmopressin, hence lithium-induced nephropathy is also casted out. Furthermore, no autoimmune or neurological disorder that could affect the hypothalamus was detected. The etiology of PP is poorly understood and likely multifactorial.[1,4] Malfunction of the hypothalamic thirst center, in supraoptic nucleus, is seen as a likely cause.[8] Excessive intake of fluid was postulated to altered feedback regulation of the HPA axis.[4] Watson et al. asserted that HPA axis function is abnormal in patients with bipolar disorder, while the degree of abnormality is equal in remitted and non-remitted patients.[9] It is theoretically argued that dysregulation of the HPA axis via reduced glucocorticoid receptor expression lead to increased dopamine neurotransmission through hypersecretion of corticosteroids[10] (Image 1). Moreover, it is suggested that disturbance of dopamine neurotransmission mechanism may be underlying reason of manic episode.[11] Dopaminergic hyperactivation has been registered in some trials and elevated dopamine levels may cause stimulation of thirst center while induce a manic episode.[12,13] Therefore, dopaminergic receptor blockage with antipsychotics such as quetiapine could be efficient in treatment of PP.[6,7] We planned to add-on quetiapine to lithium treatment and determined to benefit from its blockage effect on D2 receptor consequently.

In PP, as our patient is, hyponatremia often is mild and asymptomatic unless there is another contributing comorbidity. Anyhow, it would be better to be cautious in treating polydipsia comorbid with bipolar disorder in view of potential drug adverse effects. We planned more frequent follow-ups in order to monitor the clinical progression of PP while switching mood stabilizor. This report may provide evidence to the literature about the role of hypotalamic dysfunction in bipo-

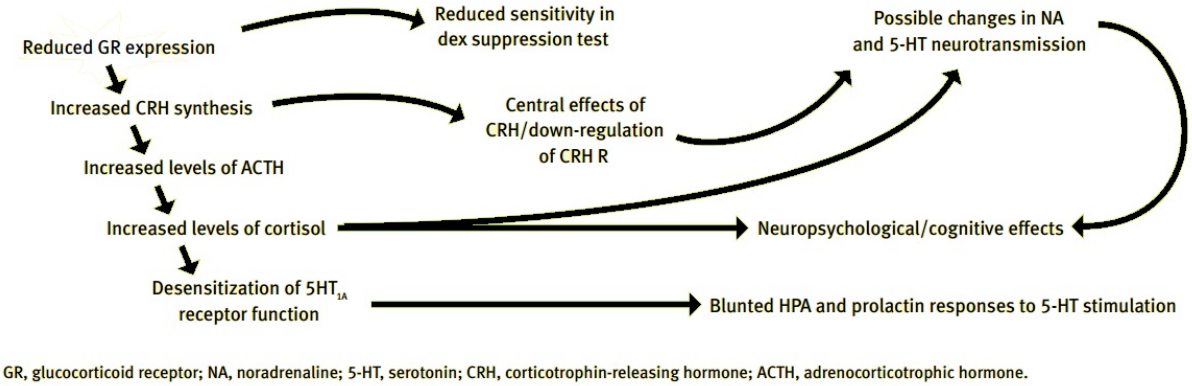

Image 1. The glucocorticoid receptor theory of mood disorders[10]

lar disorder as well as in PP.

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