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Short Communication

Is polyuria related to severity of disease in schizophrenic patients? A short communication

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Abstract

Polyuria is a common finding among psychiatric patients, among all, mostly detected in schizophrenic patients, which could happen due to many reasons including water intake increase, drug side effects and central neurologic or hormonal issues. Some several case reports have proved association of polyuria with schizophrenia, yet not exactly clarified if related to severity of the disorder. Considering the fact that we have so far never had a report of polyuria in schizophrenic monozygotic twins, this report is up to introduce a case with rather new qualities of the disease than has been so far reported.

Keywords: disease, schizophrenic patients

Introduction

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Polyuria is a clinical presentation that usually defined as excessive large production of urine which according to definitions is at least 2.5 to 3 L/24h in adults. Polyuria may be physiologic or pathologic. Physiologic causes include fluid intake elevation , diuretics and pregnancy. Diabetes,glomerulonephritis, congestive heart failure (CHF) , Cushing's syndrome, Addison's disease, Hepatic failure, Chronic Renal Failure (CRF) , lupus (SLE) and other connective tissue diseases, hyperthyroidism, hypopituitarism and psychogenic polyuria are reasons corresponded for pathologic status (1).

Psychogenic polyuria is a common clinical status characterized by both polyuria and polydipsia inpatients with psychiatric disorders. The underlying mechanism which leads to situation is no clear. There is also a high prevalence of excessive drinking in schizophrenic patients which can simply explain polyuria in these patients (2).

Increased water intake and output is more common among psychiatric patients, especially thosewith schizophrenia, than in general population . Polyuria is found consistently with many case reports on psychotic patients (3).

Case presentation

31 years old monozygotic twin sisters with a poor socioeconomic status and low degree of education from a rural living environment, with a history of psychiatric disorders since late childhood,were admitted to our psychiatric hospital with a diagnosis of schizophrenia and a resulted chief complaint of polyuria. Polyuria was associated with other disease symptoms, including auditory hallucinations, paranoid or bizarre delusions, disorganized speech and thinking, social dysfunction, deficit of typical emotional responses and though breakdown.

Our patients' first symptoms of the disease started to appear within years of high school by eratomania which made the first diagnosis to be brain dementia. Since then other symptoms have come to way and our patients have mostly been through a progressively deteriorating state of the disease, resulting in urge to average every 3 months of hospitalization.

Our patients had been put through a therapeutic drug regimen including perphenazine 4mg/ TDS, clozapine 25mg/HS, Na-valproate 200mg/BID, fluoxetine 20mg/daily, biperiden 2mg/BID and six sessions of electro-convulsive therapy (ECT). They were unresponsive to all anti-psychotic therapies and semi-responsive to ECT.

Polyuria and polydipsia within these patients was concurrent with severity of other disease symptoms, improving and deteriorating by progression and regression of disease symptoms. Surprisingly lithium intake did not increase polyuria in our cases.

Although hyponatremia has almost always been associated with water intoxication, lethargy and seizure in many reports, no evidence of water intoxication was found with our patients. Also both urine special gravity and fasting blood sugar have been reported normal during the whole length of disease in both our cases.

Also genetics as one leading factor to polyuria and polydipsia in schizophrenic patients introduced within many former reports seemed to be of no value in our patients because except for the twin sisters, other family members were perfectly healthy.

Discussion

In our study , we introduced a twin monozygotic schizophrenic patients that polyuria and polydipsia were accelerated parallel exacerbation of primary disorder.

Even higher prevalence of polydipsia and polyuria of about 3-17%, has long been recognized and reported within articles in psychiatric patients, even before the era of neuroleptic treatment (4). With antipsychotic therapy it was declined in our cases.

The problem occurs with even a higher frequency in patients with schizophrenia rather than other psychiatric disorders, and in 10% of cases, associated with hyponatremia (5).

The prevalence varies with severity of the problem, from 6.6% with water intoxication and hyponatremia (6,7), to 50-75% with polydipsia (8). Sodium level in plasma in our patient was intact.

Morning urinary specific gravity of1.008 or less, may suggest the presence ofpolyuria in these patients (9).

Mean daily urine volume for patients with schizophrenic disorders was reported as 2,319 ml/h, 1054 ml/h for non-schizophrenicand 1,265 ml/h for normal population (10).

Hyponatremia due to hypothalamic defects, the syndrome of inappropriate secretion of ADH (SIADH) and neuroleptic medications including TCA and Carbamazepine can lead into states of water intoxication associating with other life threatening symptoms of the disease including lethargy, psychosis, seizure and death (11).

Water intoxication can lead to irreversible brain damage and could be the cause of nearly 20% deaths of schizophrenic patients (12).

In our cases, polyuria was respondent to anti-psychotic therapy. The severity of disorder was related in severity of polyuria. Both patients were suffered from this symptom and both patients had similar response to treatment. We suggest the psychiatrists consider water intoxication in these patients . Also, It should been assessed all organic approaches to polyuria and polydipsia.

Conclusion

It appears to us that the disturbances in limbic system associated with psychosis through biochemical pathways bring about changes in supra-optic and para-ventricular nuclei of hypothalamus where thirst andosmotic regulation take place.The management strategy in psychiatric patients should include fluid restriction and behavioral and pharmacologic modalities. Medications including TCA and Carbamazepine might worsen hyponatremic states of patients, so should be considered with sufficient precautions.

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