Psychogenic Polydipsia with Hyponatraemia: A Case Report
Psikojenik Polidipsiye Bağlı Hiponatremi: Olgu Sunumu

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ABSTRACT
Psychogenic polydipsia (PPD) is a rare condition that is characterised by polyuria and polydipsia. Polydipsia with hyponatraemia commonly occurs in chronic psychosis patients. In this case report, we present a 62 year old male patient who was admitted with impaired consciousness. He had been drinking more than 10 litres of water per day for four days.

Keywords: Psychogenic polydipsia, hyponatraemia, emergency department

Introduction
Psychogenic polydipsia (PPD) is a rare condition that is characterised by polyuria and polydipsia (1). Polydipsia with hyponatraemia commonly occurs in chronic psychosis patients (1, 2). Delirium and behavioural changes may be observed in acute hyponatraemic patients, which may mimic psychomotor retardation or agitation (3). Hypotonic encephalopathy may progress to headache, nausea, vomiting, seizures, confusion, lethargy and coma, along with respiratory arrest (3-5). In this case report, we present a patient who has chronic schizophrenia with hyponatremia due to polydipsia.

Case Report
A 62 year old male patient was admitted with impaired consciousness. From the patient’s history over 22 years of follow-up, it was established that he had taken olanzapine for the treatment of schizophrenia. The patient had been drinking more than 10 litres of water per day for four days. On physical examination, his blood pressure, temperature and respiratory rate were 150/90 mm Hg, 36.5°C and 14 breaths/min, respectively. His laboratory findings were as follows: haemoglobin 15.2 g/dL, leucocytes 15300 cells/mm$^3$, haematocrit 46.1%, and platelets 323000 per mm$^3$. At that time, serum electrolytes were as follows: sodium 115 mmol/L, potassium 4.1 mmol/L, chloride 92 mmol/L, antidiuretic hormone (ADH) 6.2 pg/mL, plasma osmolarity 241 mOsm/L, urine osmolarity 180 mOsm/L, and urine density 1005. Renal and liver screening tests, cardiac enzymes (creatine phosphokinase and troponin I) and brain computed tomography were normal.

Hyponatraemia due to psychogenic polydipsia was diagnosed and treatment started. Treatment was initiated with normal saline at a rate of 2000 cc per day, while oral fluids were restricted to 1 litre per day. After three days of treatment his symptoms showed significant improvement. The patient’s symptoms recovered and on the third day, laboratory values were as follows: sodium 134 mmol/L, potassium 4.1 mmol/L, chloride 92 mmol/L, antidiuretic hormone (ADH) 6.2 pg/mL, plasma osmolarity 241 mOsm/L, urine osmolarity 180 mOsm/L, and urine density 1005. Renal and liver screening tests, cardiac enzymes (creatine phosphokinase and troponin I) and brain computed tomography were normal.

The patient was discharged with the recommendation to contact the psychiatric department.

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Table 1. Laboratory parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>1st day</th>
<th>3rd day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na (mEq/L)</td>
<td>132-146</td>
<td>115</td>
</tr>
<tr>
<td>Cl (mEq/L)</td>
<td>99-109</td>
<td>80</td>
</tr>
<tr>
<td>Glucose (mg/dL)</td>
<td>70-100</td>
<td>114</td>
</tr>
<tr>
<td>Urea (mg/dL)</td>
<td>19-48</td>
<td>15</td>
</tr>
<tr>
<td>Urine density (1010-1025)</td>
<td></td>
<td>1005</td>
</tr>
<tr>
<td>Plasma osmolarity (275-295)</td>
<td></td>
<td>241</td>
</tr>
<tr>
<td>Urine osmolarity (300-1000)</td>
<td></td>
<td>180</td>
</tr>
<tr>
<td>ADH (0-8 pg/mL)</td>
<td></td>
<td>6.2</td>
</tr>
</tbody>
</table>

Na: Sodium, Cl: Chloride, ADH: Anti-diuretic hormone

Discussion
Psychogenic polydipsia occurs in 6% to 20% of psychiatric patients and in 25% of the subgroup of patients with schizophrenia (1, 3). In addition, other psychotic conditions, psychotic depression, manic depressive psychosis, personality disorders, autism, and mental retardation have also been described in cases of dementia (3, 6, 7). A significant PPD may even occur in people who have had no previous psychiatric history (7). In this case, the patient had undergone schizophrenia therapy for 22 years.

A well-defined table of clearly understood pathophysiology exists (8). However, the situation is complex and multifactorial; malfunction of the hypothalamic thirst centre is seen as a likely cause (1, 9). PPD is most likely to occur as a result of chronic intake of excess fluid, changing the feedback regulation of the hypothalamic-pituitary axis (9). Inappropriate ADH secretion may also occur in many patients with PPD (10). This may be due to the use of psychiatric drugs. In addition, dopamine hypersensitivity has been reported to be effective in the table (9). Furthermore, atrial natriuretic peptide (ANP) secreted during stress inhibits the intrahypothalamic secretion of vasopressin. In addition, in the absence of the removal of the inhibitory effect on dipsogenic polypeptide angiotensin-2 control of ANP stimulation by serotonin use causes polydipsia (11).

Fluid restriction is sufficient for the treatment of PPD in most cases. In severe cases, however, hypertonic saline solution is recommended in an emergency (1, 3). Clonidine and enalapril are reported to have beneficial effects in terms of serum sodium levels and urine output (12). There are also studies of lithium for preventing demeklosicyan polydipsia (1, 3). In addition, clozapine, low-dose risperidone, and propranolol are proposed therapies, as they are said to be beneficial in preventing the development of pulmonary congestion of furosemide (1, 3, 4, 8, 13). Fluid restriction was sufficient for us to have offered these to our patients. In our case, fluid restriction and treatment with 20 mg olanzapine was continued. Hypertonic saline solution was not given. In our case increased plasma and urine osmolality was observed on the third day of follow up. Request thirst disappeared.

Conclusion
Polyuria, polydipsia and hyponatraemia may occur in patients with psychiatric disorders which can cause morbidity and mortality in severe clinical manifestations such as encephalopathy. Therefore, psychiatric emergency department patients with primary disease symptoms should be questioned and polydipsia and electrolyte imbalances should be ruled out before treatment is continued.

Conflict of interest
No conflict of interest was declared by the authors.

References