*CASE REPORT - OLGU SUNUMU*

# SYMPTOMATIC HYPONATREMIA ASSOCIATED WITH PSYCHOGENIC POLIDIPSIA

# PSİKOJENİK POLİDİPSİYE BAĞLI SEMPTOMATİK HİPONATREMİ

**Gözde BUMİN AYDIN[1], Kadriye KAHVECİ[1], Dilşen ÖRNEK[1], M. A. BİLGİÇ[2] Avni DİNÇ[1], Gülten ÖZGÜN[1]**

**Ministry of Health Etlik Education and Research Hospital [1]Department of Anesthesiology and Reanimation, [2]Department of Internal Diseases Department, Ankara, Turkey**

S.B. Etlik Eğitim ve Araştırma Hastanesi [1]Anestezi ve Reanimasyon Kliniği, [2]Dahiliye Kliniği, Ankara

***SUMMARY***

***Introduction:*** *Psychogenic or primary polidipsia characterized by excessive thirst and compulsive water drinking is a common problem among psychiatric populations, affecting 6% to 20% of patients. Symptoms of hyponatremia may range from increased psychotic symptoms to seizures, coma and even death.*

***Case:*** *A 21 year old male patient referred to emergency unit of our hospital with loss of consciousness. In his labaratory tests showed that he had hyponatremia 117 mmol $L^{-1}$. Medical testing ruled out all medical conditions known to be etiologically related to hyponatremia including pituitary tumor, paraneoplastic syndrome and syndrome of inappropriate antidiuretic hormone. In his psychiatric assesment, he was diagnosed with Psychogenic Polidipsia. After 3 days of hospitalization, he was discharged with serum sodium of 141 mmol $L^{-1}$ with his psychiatric drugs and outpatient behavioral treatment was suggested.*

***Conclusion:*** *Psychogenic polydipsia should be kept in mind in symptomatic hyponatremia patients with a variety of symptoms ranging from headache to encephalopathy or even death. Medical and behavioral treatments both have importance for treatment of this syndrome.*

***KEY WORDS:*** *Hyponatremia; Water Intake; Psychology*

***ÖZET***

***Giriş:*** *Psikojenik veya primer polidipsi aşırı derecede susama ve kompulsif su içme ile giden psikiatrik hastalarda %6'dan %20'ye kadar sıklıkta olan bir problemdir. Hiponatremi semptomları artmış psikotik semptomlardan nöbetlere, koma, hatta ölüme sebebiyet verebilir.*

***Olgu:*** *Yirmibir yaşındaki erkek hasta acil servise bilinç kaybı ile başvurdu. Labaratuvar testlerinde 117 mmol $l^{-1}$ hiponatremisi olduğu ortaya çıktı. Hipofiz tümörü, paraneoplastik sendrom ve uygunsuz ADH sekresyonu gibi hiponatremi oluşturabilecek etyolojik sebepler yapılan testlerle ortadan kaldırıldı. Psikiatrik muayenesinde hastaya psikojenik polidipsi tanısı koyuldu. Hastanın 3 günlük tedavisi sonrası psikiatrik ilaçları ve ayaktan davranışsal tedavi yöntemi önerilerek 141 mmol $l^{-1}$ Na değeri ile taburcu edildi.*

***Sonuç:*** *Semptomatik hiponatremi hastalarında, baş ağrısından ensefalopatiye hatta ölüme kadar semptom verebildiğinden psikojenik polidipsi akılda tutulmalıdır. Medikal ve davranışsal tedavinin bu sendromun tedavisinde önemi büyüktür.*

***ANAHTAR KELİMELER:*** *Hiponatremi; Su Alımı; Psikoloji*

## INTRODUCTION

Psychogenic or primary polidipsia characterized by excessive thirst and compulsive water drinking is a common problem among psychiatric populations, affecting 6% to 20% of patients (1). Excessive oral fluids consumption over a short period of time may lead to hyponatremia, which sometimes causes confusion and seizures (2-3). It was first noted as a complication of severe mental illness in the 1930's (4). This is a frequent phenomenon in chronic psychiatric patients, particulary those with schizophrenia (5). These patients became unable to excrete all of the ingested fluids, leading to dilutional hyponatremia and water intoxication. Symptoms of hyponatremia can range from increased psychotic symptoms to seizures, coma and even death. This state has been termed 'psychosis, intermittant hyponatremia and polydipsia (PIP Syndrome) (6). We have reported a patient who was referred to the emergency unit of our hospital with loss of consciousness, and diagnosed with PIP syndrome.

## CASE

A 21 year old male patient was referred to the emergency unit of our hospital with loss of consciousness. He was admitted to intensive care unit. His physical examination revealed with normal vital signs in spite of his agitation. He did not have edema, signs of dehydration or fever. He had neither experienced any head injury nor visited a hospital for this reason earlier. In his labaratory tests showed that he had hyponatremia 117 mmol $L^{-1}$. His blood urea nitrogen was 12 mg $dL^{-1}$ and serum creatinine was 1.1 mg $mL^{-1}$. Serum $K^+$ level was 3.5 mEq $L^{-1}$. No abnormalities was found in cranial region with computed tomography, and no cranial edema was found. In his neurological consultation, he was adviced Nootropil 1 mg (Pirasetam) 3x3 ampules per day. His serum sodium concentration was consistently below the lower limit of the normal range at routine laboratory tests during intensive care unit (117, 120, 123, 124 mmol $L^{-1}$).

Medical testing ruled out all medical condition known to be etiologically related to hyponatremia including pituitary tumor, paraneoplastic syndrome and secretion of inappropriate antidiuretic hormone. When he became conciouss, he admitted excessive water intake and his depression. In his psychiatric assesment, he was diagnosed with Psychogenic Polidipsia. Internal diseases doctor advised fluid restriction so he was supplemented with saline intravenously infused at 60 mL $hr^{-1}$ and his serum concentration returned to 141 mmol $L^{-1}$ on the third day. His medical instructions also included Norodol (haloperidol) 3x5 gutts and Akineton (Biperiden laktat /Abbott) 5 mg (1x1) i.m and Lustral (Sertralin/Pfizer) 100 mg 1x1. He was discharged the third day of treatment with serum sodium of 141 mmol $L^{-1}$ with his psychiatric drugs and outpatient behavioral treatment was advised to the patient.

## DISCUSSION

Hyponatremia develops when free water intake exceeds free water excretion. Excess water intake reduces plasma osmolality and sodium suppressed antidiuretic hormone secretion initiating a cascade of reduced water reabsorbsion in the distal nephron decreasing urine osmolalite to a minimum of 50-60 mOsm $kg^{-1}$ (6). Water intoxication usually happens in patients with psychiatric problem who are subject to compulsive water ingestion. It is seldom observed in ordinary people (7). Our patient also had depression and was using sertralin for this disorder. Water intoxication causes headache, blurred vision, muscle cramps, confusion, letargy, delirium, seizure and coma. Since it may cause brain edema and coma, it can be mortal. When Na concentration gets lower than 125 mmol $L^{-1}$ hipernatremic encephalopathy and neurological symptoms begin (8). It is important to consider water intoxication as a cause of hyponatremia in a patient without signs of dehydration but one should rule out all medical conditions known to be related to hyponatremia.

Some authors think that polydipsia is due to anticholinergic side effect of some psychiatric drugs (9). Some others claim that since excess water intake periods are correlated with psycotic exacerbations, psychosis and polydipsia might have similar physiopathologic mechanisms (9). Skar and Schrier (10) suggest that supraoptic nuclei of hypothalamus which secrets ADH, plays an important role in the ethiology of psychiatric diseases and fluid electrolide balance. Thirst and drinking are controlled by lateral hypothalamus. Dopamin is an important neurotransmitter in this region. In animal experiments it has been shown that the dopaminergic activity is related with polydipsia (11). Lesions created in lateral hypothalamus causes adipsia and afagia. Injections of dopamine to lateral ventricules or apomorfin to hypothalamus causes thirst. Injection of a dopamin antagonist haloperidol to lateral hypothalamus blocades the need for water intake (9). Also haloperidol is used in some pscyhiatric diseases treatment (12). In our patient we used haloperidol to regrese his depression, psychiatric semptoms and his polydipsia and poliuria. This might be related to antidopaminergic effect of haloperidol. We also used sertralin and biperiden for his treatment.

Vieweg et al (6) treated PIP syndrome using lithium and phenytoin, Leon et al (5) and Verghese at al (13) treated a schizophrenia patient with PIP syndrome with clozapine, but in our case we used haloperidol, biperiden, sertralin and behavioral treatment in our treatment modalities.

There are two recorded treatments for PIP syndrome: behavioral management and pharmacological interventions (13). Involuntary fluid restriction has been found to be effective in inpatient settings but it is less feasible in outpatient clinical settings. Behavioral management of fluid intake is an important focus of intervention and there is a report (14) on successful behavioral treatment of an outpatient with psychogenic polydipsia in which there was no attempt to restrict fluid intake.

In Constanza et al's (1) study they treated a schizophrenia patient with PIP syndrome with water restriction and behavioral treatment programme with self monitoring which she could record in a booklet the time, type of fluid, amount and situation or activity for each bevarage consumed which was very usefull, and which also showed us the importance of behavioral treatment that we also used in our treatment modalities.

In Leadbetter et al's (4) study fluid restriction is advised in short term therapy and long term treatment includes a medication management (includes anxiolitic, antipyschotic treatment) which increases patient awareness and self monitoring, but if this treatment is not effective they also add lithium to their treatment modalities. However, in our case haloperidol, sertralin and biperiden were found to be effective for our patient for insetting treatment modalities. In our case we used fluid restriction and medical treatment in hospital and in outpatient settings medical and behavioral treatment was used for the maintanence treatment of the patient.

Psychogenic polydipsia should be kept in mind in symptomatic hyponatremia patients with a variety of semptoms ranging from headache to encephalopathy or even death. Medical and behavioral treatments both have importance for treatment of this syndrome.

**Yazışma Adresi (Correspondence):**
**Dr. Gözde BUMİN AYDIN**
Barış Sitesi 2112. sok No:4 Mustafa Kemal Mahallesi
Ankara /TÜRKİYE
**e-posta (e-mail):** drgozdeaydin@yahoo.com

**KAYNAKLAR**

1. Costanzo ES, Antes LM, Christensen AJ. Behavioral and medical treatment of chronic polydipsia in a patient with schizophrenia and diabetes insipidus. Psychosom Med 2004;66(2):283-286.
2. Hiramatsu R, Takeshita A, Taguchi M, Takeuchi Y. Symptomatic hyponatremia after voluntary excessive water ingestion in a patient without psychiatric problems. Endocr J 2007;54(4):643-645.
3. Vishwajeet S, Aneesh S. Water intoxication leading to hyponatremia and seizures: a rare complication of uroflowmetry. Int Urol Nephrol 2005;37(2):275-276.
4. Leadbetter RA, Shutty MS Jr, Higgins PB, Pavalonis D. Multidisciplinary approach to psychosis, intermittent hyponatremia, and polydipsia. Schizophr Bull 1994;20(2):375-385.
5. de Leon J, Verghese C, Stanilla JK, Lawrence T, Simpson GM. Treatment of polydipsia and hyponatremia in psychiatric patients. Can clozapine be a new option? Neuropsychopharmacology 1995;12(2):133-138.
6. Vieweg WV, Weiss NM, David JJ, Rowe WT, Godleski LS, Spradlin WW. Treatment of psychosis, intermittent hyponatremia, and polydipsia (PIP syndrome) using lithium and phenytoin. Biol Psychiatry 1988;23(1):25-30.
7. Goldman MB, Luchins DJ, Robertson GL. Mechanisms of altered water metabolism in psychotic patients with polydipsia and hyponatremia. N Engl J Med 1988;318(7):397-403.
8. Dirican AC, Altunkaynak Y, Çelik S, et al. Psikojenik polidipsinin neden olduğu bir hiponatremik ensefelapati olgusu. Cerrahpaşa Tıp Dergisi 2005;36(4):221-223.
9. Altındağ A, Yanık M, Nebioğlu M. Bipolar bozuklukta psikojenik polidipsi: Bir olgu sunumu. Klinik Psikofarmakoloji Bülteni 2004;14(2):79-82.
10. Sklar AH, Schrier RW. Central nervous system mediators of vasopressin release. Physiol Rev 1983;63(4):1243-1280.
11. Mittleman G, Rosner AL, Schaub CL. Polydipsia and dopamine: behavioral effects of dopamine D1 and D2 receptor agonists and antagonists. J Pharmacol Exp Ther 1994;271(2):638-650.
12. Gelder M, Gath D, Mayou R, Cowen P. Oxford Textbook of Psyhciatry. 3rd edition. Oxford: Oxford University Pres, 1996.p. 197-245.
13. Verghese C, de Leon J, Josiassen RC. Problems and progress in the diagnosis and treatment of polydipsia and hyponatremia. Schizophr Bull 1996;22(3):455-464.
14. Thoma JL, Howe J, Gaudet A, Brantley PJ. Behavioral treatment of chronic psychogenic polydipsia with hyponatremia: a unique case of polydipsia in a primary care patient with intractable hiccups. J Behav Ther Exp Psychiatry 2001;32(4):241-250.