

# Water Intoxication Due To “Iatrogenic” Polydipsia

## “İatrojenik” Polidipsi Nedenli Su İntoksikasyonu

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### ABSTRACT

Water toxicity is an encephalopathy characterized by low serum sodium levels and serum osmolarity accompanied by a history of excessive water intake. It can rarely be observed in healthy people during clinical examinations and treatments. A 69-year-old male patient was brought to the emergency department with fatigue and dizziness. It was learned from his anamnesis that he had consumed approximately 6 L of water in 4 hours, as had been ordered, to be prepared for the urinary ultrasonography . Serum sodium level was found as 119 mmol/L. Fluid restriction was performed. After a 24-hour follow-up, the patient was discharged when the sodium level was detected as 136 mmol/L. A 52-year-old female patient admitted to the emergency department with nausea. It was learned from her anamnesis that she had consumed about 1.5 L

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of water in 10 minutes and after 1 hour, she had consumed 1 L of water in 10 minutes, as had been ordered, to avoid the side effects of the fundus angiography. Serum sodium level was found as 117 mmol/L. Fluid restriction was performed. After a 48-hour follow-up, the patient was discharged when the sodium level was detected as 134 mmol/L.

Pulmonary edema, brain edema, and even death can be seen. Major factors for the severity of presentation are the degree of hyponatremia and the rate of development. In the present cases, the patients had ingested excessive amount of water in a very short time and acute hyponatremia had developed. We think that individualization of water intake (especially upper limits) recommended by health professionals is important in order to prevent water intoxication.

### ABSTRACT

Su toksisitesi, aşırı su alımı öyküsünün eşlik ettiği düşük serum sodyum seviyeleri ve serum ozmolaritesi ile karakterize bir ensefalopatidir. Sağlıklı kişilerde nadiren de olsa klinik muayene ve tedaviler sırasında görülebilmektedir. 69 yaşında erkek hasta halsizlik ve baş dönmesi şikayeti ile acil servise getirildi. Anamnezinde aynı gün kendisine söylendiği üzere 4 saatte üriner ultrasonografi hazırlığı için yaklaşık 6 L su tükettiği öğrenildi. Acil servis başvurusunda serum sodyum 119 mmol/L bulundu. Sıvı kısıtlaması yapıldı. 24 saatlik izlem sonrasında sodyum 136 mmol/L saptanan hasta taburcu edildi. 52 yaşında kadın hasta bulantı şikayeti ile acil servise başvurdu. Anamnezinde fundus anjiyografi yan etkilerinden korunmak için kendisine

**Anahtar kelimeler:** Ensefalopati, hiponatremi, su toksikasyonu

söylendiği üzere 10 dakikada yaklaşık 1,5 L ve 1 saat sonra 10 dakikada 1 L su tükettiği öğrenildi. Acil servis başvurusunda serum sodyum 117 mmol/L bulundu. Sıvı kısıtlaması yapıldı. 48 saatlik izlem sonrasında sodyum 134 mmol/L saptanan hasta taburcu edildi. Akciğer ödemi, beyin ödemi ve hatta ölüm görülebilir. Başvuruda klinik ciddiyeti için ana faktörler, hiponatreminin gelişim hızı ve derecesidir. Sunulan olgularda hastalar çok kısa sürede aşırı miktarda su almış ve akut hiponatremi gelişmişti. Su intoksikasyonunun önlenmesi için sağlık profesyonelleri tarafından önerilen su alımının (özellikle üst limitlerin) bireyselleştirilmesinin önemli olduğunu düşünmekteyiz.

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## INTRODUCTION

Water toxicity is an encephalopathy characterized by low serum sodium levels and serum osmolality accompanied by a history of excessive water intake (Moritz and Ayus, 2003). It occurs when water intake is rapid and excessive. While it is generally seen as a result of compulsive water consumption in patients with a history of psychiatric illness, cases of water intoxication secondary to excessive water intake have also been reported in the literature in those fed on high amounts of low-solute solutions and due to water intake after heavy exercise (Almond et al., 2005; Hiramatsu et al., 2007; Swanson and Iseri, 1958). However, it can rarely be observed in healthy people during clinical examinations and treatments (Vishwajeet and Aneesh, 2005).

## CASE PRESENTATION

### Case-1

A 69-year-old male patient was brought to the emergency department with the complaints of fatigue and dizziness. The patient had diagnoses of type 2 diabetes mellitus, hypertension, and benign prostatic hypertrophy. His regular medications were metformin, acetylsalicylic acid, and perindopril/indapamide. In his anamnesis, it was learned that he applied to the urology outpatient clinic on the same day for urinary ultrasonography, and as he was ordered, he consumed about 6 L of water in 4 hours for the preparation of the procedure. The patient's current complaints started approximately 5 hours after the procedure. The patient said that after fluid consumption, he could not urinate for about 2 hours, and then urine output started. Vital signs and electrocardiography (ECG) were normal. In the systemic physical examination, no edema, signs of dehydration, neurological deficit, or pathology that

required a psychiatrist's opinion were found. Serum sodium (119 mmol/L) and glucose (113 mg/dL) were found in the emergency room admission. Liver, thyroid, and kidney function tests were also normal. Plasma osmolality was 247 mosm/kg, and urine density was 1005, urine osmolality was 555 mosm/kg. Serum sodium was observed at 135 mmol/L in his application to the urology outpatient clinic the day before and it was evaluated as acute hyponatremia. Since he was symptomatic, 300 ml of 3% NaCl was administered intravenous (IV), and control sodium was 124 mmol/L, plasma osmolality 259 mosm/kg, and urine osmolality 589 mosm/kg. Due to the regression of the patient's complaints and urine output, hydration was interrupted and fluid restriction was performed. After a 24-hour follow-up, the patient was discharged when the sodium level was detected as 136 mmol/L.

### Case-2

A 52-year-old female patient was admitted to the emergency department with a complaint of nausea. The patient had diagnoses of type 2 diabetes mellitus and hypertension. Her regular medications were metformin, acetylsalicylic acid, insuline detemir, insuline aspart and ramipril. In her anamnesis, it was learned that she applied to the ophthalmology outpatient clinic on the same day for fundus fluorescein angiography, and as she was ordered, she consumed about 1.5 L of water in 10 minutes and after 1 hour, she consumed 1 L of water in 10 minutes to avoid the side effects of the procedure. The patient's current complaints started approximately 3 hours after the procedure. Vital signs and ECG were normal. In the systemic physical examination, no edema, signs of dehydration, neurological deficit, or pathology that required a psychiatrist's opinion were found. Serum sodium (117 mmol/L) and glucose (210 mg/dl) were found in the emergency room admission.

Liver, thyroid, kidney function tests were also normal. Plasma osmolarity was 250 mosm/kg, and urine density was 1008. Two weeks ago, serum sodium was observed at 136 mmol/L in her application to the internal medicine outpatient clinic and it was evaluated as acute hyponatremia. Since she was symptomatic, 300 ml of 3% NaCl was administered IV, and control sodium was 122 mmol/L, plasma osmolarity (262 mosm/kg). Due to the regression of the patient's complaints hydration was interrupted and fluid restriction was performed. After a 48-hour follow-up, the patient was discharged when the sodium level was detected as 134 mmol/L.

## DISCUSSION

The diagnosis was made in these cases according to the history and excluding organic-psychiatric causes. Early symptoms are usually mild and making a diagnosis is difficult in chronic cases (Moritz and Ayus, 2003). However, the clinical presentation may be drastic in acute hyponatremia. Pulmonary edema, brain edema, and even death can be seen (Hiramatsu et al, 2007). Major factors for the severity of presentation are the rate of its development and the degree of hyponatremia (Swanson and Iseri, 1958). In the present cases, a high volume of water intake has been ordered by the healthcare staff to be able to optimize the pelvic USG evaluation and avoid the side effects of the angiography. However, patients ingested excessive amount of water in a very short time and acute hyponatremia developed. Although the kidneys try to dilute the urine at a physiologically significant level after excessive water intake, their capacity is limited even if they have normal function (Hiramatsu et al, 2007). In addition, it is well known that the excretion of water further decreases with the presence of kidney disease and senility. Generally, severe neurological

symptoms do not occur unless the serum sodium level falls below 125 mmol/L. However, it has been reported that encephalopathy is seen at higher levels, especially in women who are in the hormonally active period. In premenopausal women, the risk of residual neurological damage following symptomatic hyponatremia is 25 times greater than in men (Weiss, 2004). Early diagnosis and treatment are very important in terms of reducing mortality. In our cases, acute hyponatremia was diagnosed before the unconsciousness progressed, treated appropriately, and clinically stabilized. Most of the time, fluid restriction is sufficient in the treatment of mild cases. But emergent IV administration of hypertonic NaCl may be required in some cases that have encephalopathy, like the present cases.

## CONCLUSION

The increase in the elderly population worldwide, the high prevalence of kidney disease, the frequent use of imaging/endoscopic procedures in the diagnosis of diseases, and the incomplete and incorrect information given to patients by health personnel about water consumption are risk factors for the development of "iatrogenic" water toxicity, as in these cases. We think that it is important to individualize the water intake (especially the upper limits) recommended by health professionals in order to prevent this complication.

### Author Contributions

Concept: İK, FK

Design: Bİ, ST, SÜ

Data collection and/or processing: Bİ, ST, NG

Writing manuscript: FK, NG

Critical review: İK, SÜ

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