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CASE REPORT

Sounds unrealistic: an adolescent girl with anorexia nervosa consumes 19 L of fluid in a few hours: what happens to the physiology?

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Abstract

Background Adolescents with eating disorders (EDs) may present not only with abnormal eating behaviors but also with abnormal drinking behaviors varying widely. These behaviors include water loading to cheat on weight measurements, to feel full and suppress appetite and/or to induce vomiting; as well as restricting fluid intake in addition to food.

Method We present a 16-year-old female adolescent with anorexia nervosa restrictive type and major depressive disorder who was hospitalized due to acute food refusal and developed generalized seizures due to dilutional hyponatremia in consequence of consuming excessive amount of water. Psychiatric diagnoses were made according to 'The Diagnostic and Statistical Manual of Mental Disorders' (5th ed.; DSM–5) criteria.

Results After starting nutritional rehabilitation with a low calorie meal plan to avoid refeeding syndrome, a weight gain of 2 kg was noted in the second day of hospitalization. At the bedside visit, she was observed in a disoriented manner and consecutively in seconds, lost consciousness with a generalized tonic–clonic seizure lasting 2 min. Her serum sodium level was measured as 116 mEq/L, which was normal at the time of admission. It was later learned that she secretly ingested 19 L of water in a short amount of time. She regained consciousness and no further seizures were observed after intravenous sodium deficit correction and fluid restriction therapy. Her serum sodium level was normalized (137 mEq/L) within 12 h.

Conclusion A thorough clinical assessment of hydration and drinking behaviors as well as eating behaviors is essential for patients with EDs to avoid serious medical complications with high mortality and morbidity during follow-up. It is interesting that this amount of fluid consumption in such a short period of time did not present to the clinic with vomiting, gastric dilatation or bowel irrigation symptoms in a case with acute food refusal and restriction for a year, instead absorbed very quickly causing acute and severe symptomatic hyponatremia with generalized seizures.

Keywords Adolescent · Anorexia nervosa · Hyponatremia · Seizure · Water intoxication

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Introduction

Patients with eating disorders (EDs) may present with abnormal drinking behaviors varying widely. Some may increase fluid intake as water loading prior to getting weighed during outpatient or inpatient follow-up to cheat on weight measurements, to feel full and suppress appetite and/or to induce vomiting. Some others may decrease fluid intake to avoid fullness, abdominal discomfort and bloating or some may fear that water will cause them gain weight and restrict fluid intake in addition to food. Thus, an ED needs to be considered as a disorder of fluid intake as much as a disorder of food intake [1].

Abnormalities in fluid consumption behaviors can result in serious medical complications via the impairment of osmoregulation. EDs have been associated with various renal function abnormalities, including a decline in glomerular filtration rate (GFR) which can result in severe kidney disease, impaired water diuresis, a decreased urinary concentrating capacity and various electrolyte abnormalities [2–4]. Polydipsia can often accompany EDs, but it unusually leads to severe complications [5]. Severe hyponatremia and hypo-osmolality, due to excessive consumption of water, causing cerebral edema, and leading to ataxia, seizures, coma, and death were presented among previously published ED cases [5–7]. On the other hand, fluid restriction in EDs for a long period of time is known to be associated with circulatory disorders (hypotension), electrolyte imbalance (hyponatremia or hypernatremia), seizures, permanent brain damage, renal and cardiac complications and/or death [7].

Here, we present a female adolescent with anorexia nervosa restrictive type (AN-RT) who developed generalized seizures due to dilutional hyponatremia as a result of excessive amount of water consumption.

Case presentation

Medical history and interviewing the adolescent

A 16-year-old adolescent girl admitted to our clinic with significant weight loss and two missed periods. She was referred by a child and adolescent psychiatrist for medical stabilization and nutritional rehabilitation with an initial diagnosis of AN-RT. Medical history revealed the onset of the ED symptoms dating back 12 months before admission to the hospital when she started dieting after a close relative had told her that she was overweight. She reported that she decided to lose weight because of feeling uncomfortable with her weight and her appearance. Despite the attainment of her initial target weight, she had continued dieting with a progressively more marked restriction of food intake. She confirmed that she lost 35 kg (from 77 kg to 42 kg) and still feeling fat, reporting dissatisfaction with physical appearance and fear of gaining weight. She neither reported excessive exercise nor the use of any laxatives, supplements, diuretics, or dietary pills. A HEEADSSS (home, eating, education/employment, activities, drugs, sexuality, suicide (emotions) and safety) interview [8] with the patient revealed that she was the seventh child out of nine of a low-income and uneducated family. Her father, mother and her older sisters, whom she felt really close and supportive, had to move to another city 2 years ago due to financial problems. She stayed with her grandparents in her hometown to continue her education (9th grade) in the same school she has been attending. Later on because she could not cope with the separation, especially from her sisters she moved back into her parents house 1 month before completing the ninth grade and failed due to non-attendance to classes for that month. She was also unhappy that she had to leave her best friends behind at her hometown. After moving in with her parents, although she was successful at school and determined to pursue her education, she was withdrawn from her new high school against her will by her family and she was enrolled in a distance learning program. She reported to become more isolated and the time she spent at home alone increased. She started restricting her diet just after her relocation and withdrawal from the school and peers.

Physical examination and laboratory evaluation

On her physical examination, she appeared very cachectic. She had non-edematous, cold extremities. Her skin turgor tonus and mucous membranes were normal. The other systems were normal except the increase of the vellus hair on her body. Her weight was 41.7 kg (< 3rd percentile), height was 157 cm (10–25th percentile) and body mass index was 16.9 kg/m^2 (< 3rd percentile) according to 'Centers for Disease Control and Prevention' growth charts [9]. Her body temperature was 36.1 °C and respiratory rate was 18 beats per minute (BPM). Her supine blood pressure and heart rate were 115/70 mmHg and 76 BPM. Her standing blood pressure and heart rate were 110/70 mmHg and 108 BPM. Her ideal weight for height was 46.4 kg (her weight/ideal weight for height percentage was 89%). Target weight was estimated between 48 and 50 kg, which was the weight when she lost her menstrual cycles, approximately 2 months ago. Her laboratory investigations are shown in Table 1.

Table 1 Patient's initial laboratory investigation

Laboratory investigation	Value	Normal range
Hemoglobin (g/dL)	13.4	11.7–15.5
Erythrocyte count	4.7×10^6/mm^3	$3.83–5.08\times10^6$/mm^3
Leukocyte count	6×10^3/mm^3	$4.1–11.2\times10^3$/mm^3
Thrombocyte count	350×10^3/mm^3	$159–388\times10^3$/mm^3
Glucose (mg/dL)	71	60–100
Sodium (mEq/L)	140	136–146
Potassium (mEq/L)	4.08	3.5–5.2
Calcium/phosphorus (mg/dL)	10.7/5.1	8.4–10.2/4–7
Magnesium (mg/dL)	2.32	1.7–2.4
Blood urea nitrogen/creatinine (mg/dL)	7.44/0.7	11–39/0.5–1.2
Total/direct bilirubin (mg/dL)	0.85/0.15	0.3–1.2/0–0.2
ALT (U/L)	9	0–35
TSH (uIU/mL)	1.92	0.27–4.2
Free T3/T4 (pmol/L)	4.05/11.33	3.1–6.7/12–22
Erythrocyte sedimentation rate (mm/h)	4	0–20
Urine pH/urine specific gravity	6/1015	4.5–8.0/1003–1030

Psychiatric assessment

After the initial interview and medical evaluation in the Adolescent Medicine Clinic, she was also evaluated in the Child and Adolescent Psychiatry Clinic. During the psychiatric interview, she described her father as a strict and overprotective parent. She felt that she was not able to express herself well and often repressed her feelings. She was an above average student academically before. She was very sad about not being able to attend regular school anymore with her friends and blamed herself for school failure. She expressed feelings of fatness and fear of fatness, low mood, loss of interest in daily activities, easy fatigability, apathy, decreased concentration and pessimistic ideas about future. No suicidal ideas or unusual perceptual experiences were reported. She denied the serious condition of her physical health and thought that she was eating "just enough". Her sister described that she has been engaged in some classic behaviors, such as food avoidance, hiding food and guilt over eating. She was diagnosed with AN-RT and major depressive disorder according to The Diagnostic and Statistical Manual of Mental Disorders (5th ed.; DSM–5) criteria. Existing medications olanzapine 2.5 mg/day and fluoxetine 20 mg/day were continued.

Treatment and clinical course

She was admitted to the general pediatric inpatient unit due to acute food refusal where she was followed by a multidisciplinary team consisting of an adolescent medicine specialist, a child and adolescent psychiatrist and a dietician experienced in ED, in addition to a pediatric consultant with three pediatric residents on duty. A 24-h cardiac monitoring was done for the risks of bradycardia and other arrhythmias. Orthostatic changes of blood pressure and pulse were measured every morning and vital signs were continuously evaluated during the day at hourly intervals. Her calorie intake before hospitalization was calculated as 130 kcal. A total of 750 kcal was started with nutritional supplement solutions divided into three meals to avoid refeeding syndrome. Daily calorie intake was planned to be slowly increased according to the daily weight gain. In the second day of hospitalization, a weight gain of 2 kg was noted. At the bedside visit by the adolescent medicine team, it was observed that she was a bit drowsy and answered the questions in a disoriented manner. Suddenly, she threw her body back in the bed creating a misimpression of a conversion action. Consecutively in seconds, she lost her consciousness and developed a generalized tonic–clonic seizure lasting for 2 min accompanied with severe bradycardia (pulse rate = 30 BPM). Her bradycardia improved with the rapid intervention of airway protection and free oxygen administration by nasal cannula. Her laboratory tests revealed that the serum sodium level was 116 mEq/L and urine specific gravity was 1002. Calculated serum osmolality according to the formula of "$(Na^+\times2)+(Glucose/18)+(BUN/2.8)$" was 239.9 mOsm/kg. With the diagnosis of symptomatic acute hyponatremia, rapidly 3% hypertonic saline solution (3 mL/kg) was administered intravenously. Afterwards, it was learned that water intoxication was due to secretly consuming 19 L of bottled water that morning. Her sister noticed that the water bottles that she bought were all empty and the patient confessed consuming large amounts of water because she wanted to be discharged quickly from the hospital with pseudo-weight gain as well as to avoid eating and to suppress her hunger. Fluid restriction and sodium deficit correction therapy (8-h

infusion to rise 6 mEq/L increase in plasma sodium) were initiated. Computed tomography visualization for acute brain pathologies was reported to be normal. She was consulted to the pediatric neurology team. A second seizure was observed within the next hour and it was stopped by intravenous diazepam (0.1 mg/kg/dose) administration. She was transferred into pediatric intensive care unit for close monitoring. Sodium deficit correction was continued with intermittent serum sodium level measurements. Her neurological status progressively got better with the treatment and her serum sodium level was normalized (137 mEq/L) within 12 h. She was transferred back to the pediatric inpatient unit the next day. Her initial and follow-up laboratory values are shown in Table 2.

On the following days, her weight dropped to 40.4 kg because of water diuresis as well as non-compliance with the dietary instructions. Necessary precautions were taken in the pediatric ward to avoid fluid overconsumption. Olanzapine was increased to 7.5 mg/day and fluoxetine to 30 mg/day since she reported suicidal thoughts. Corrected QT, serum lipid profile and prolactin levels were evaluated to screen the side effects of antipsychotic medication. Her diet changed as three main meals and 1–3 snacks on the second week. On the fourth week, olanzapine was lowered to 2.5 mg/day due to increased prolactin levels and reduced depressive symptoms. During her 5-week stay, she gained 5 kg. Therapeutic alliance was established with the solution of returning to school. One year after discharge, our patient reached her ideal weight and she started having regular menstrual cycles. No additional seizures were reported.

Discussion

Our patient had acute, symptomatic, severe, dilutional hyponatremia. EDs can be complicated by various electrolyte abnormalities especially when purging accompanies and/or during the refeeding period [10]. Hyponatremia in EDs can be a result of different factors and if the underlying etiology is unknown, urine sodium and urine specific gravity tests can be helpful to identify the cause. While hyponatremia secondary to purging is mainly accompanied by dehydration, in restrictive disorders, hyponatremia might be a result of either dehydration (fluid restriction) or overhydration (water loading). Hyponatremia is the most common consequence of overhydration and defined as plasma sodium being lower than 135 mEq/L [11]. Serum sodium levels below 125 mEq/L are considered as severe hyponatremia and time of development before 48 h is accepted as acute onset.

The initial diagnostic approach to hyponatremia is to differentiate hypotonic from nonhypotonic hyponatremia [12]. Our patient's low serum osmolality indicated hypotonic hyponatremia. Once hypotonic hyponatremia diagnosis was established, the volume status of the patient should be evaluated [13]. Since there was an acute onset of severe symptoms in our case, immediate treatment with hypertonic saline was done, and urine osmolality and urine sodium levels could not be assessed. However, the diagnosis of dilutional hyponatremia was made through the history of overconsumption of fluids and physical examination findings. It is essential to quickly identify and treat the underlying cause of an acute symptomatic seizure, as they have higher mortality rates compared to unprovoked seizures [14]. Electrolyte abnormalities should be among the primary differential diagnoses when a patient with ED develops seizures.

Water balance is regulated by multiple mechanisms to sustain extracellular fluid osmolality around 300 mOsmol/kgH2O, mainly through the vasopressin-stimulated aquaporin 2 water channels in the kidney collecting duct which controls water reabsorption [15]. When excessive amounts of fluid intake occur, osmoreceptors within the brain inhibit both the vasopressin secretion and osmotically driven thirst mechanism of the body. Receptors throughout the gastrointestinal system also help in the termination of drinking behavior. According to the functional magnetic resonance imaging (MRI) studies, over-drinking is perceived as unpleasant by the body [16]. On the other hand, psychogenic

Table 2 Selected laboratory values and body weight follow-up of the patient

	First admission Day #1	Day #2	Day #3	Discharge at Week #5	After discharge Week #1
Body weight (kg)	41	43	41.9	45	45.9
Density of urine	1015	1002	1006	1013	1008
Sodium (mEq/L)	140	116	137	141	138
Potassium (mEq/L)	4.08	3.6	3.52	4.43	3.87
Glucose (mg/dL)	71	102	76	89	86
Blood urea nitrogen (mg/dL)	7.44	10.7	5.81	9.26	14.74
Creatinine (mg/dL)	0.7	0.52	0.47	0.5	0.51
Calcium (mg/dL)	10.7	8.64	8.99	9.42	8.9
Phosphorus (mg/dL)	5.1	3.5	4.2	4	3.81

polydipsia is associated with reward seeking, anxiety relieving, coping mechanisms in psychiatric patients [15].

Chronic overhydration leads to the downregulation of aquaporin 2 water channels resulting in an adaptation mechanism of urinary free water excretion [17]. However, acute overconsumption of water (approximately 3–4 L of fluid in less than an hour) or when the consumption exceeds the capacity of water excretion (approximately 10 L per day), water intoxication (dilutional hyponatremia) might occur [18, 19]. Our patient's history of consuming 19 L of bottled water in a very short amount of time like a few hours explains the development of severe hyponatremia and related generalized seizures. There are limited number of case reports in the literature describing acute onset seizures in patients with AN of which most were related with hyponatremia after water intoxication [5, 20]. After correction of the electrolyte disturbances, no further seizures were observed [21].

Since the two subsequent seizures in our patient happened as a consequence of an acute electrolyte imbalance, they were diagnosed as acute symptomatic seizures and unprovoked or epileptic seizures were not among the initial diagnoses [22]. According to the findings of large case series, seizures caused by reversible metabolic or toxic disturbances are associated with a minor risk of epilepsy [23]. Acute symptomatic seizures caused by electrolyte disturbances are not often reoccur, unless the underlying etiology recurs [22]. In the adolescent age group, the electroencephalography (EEG) and MRI are usually recommended in the presence of an apparent unprovoked and/or focal seizure, a cognitive or motor impairment with unknown etiology and unexplained neurologic examination abnormalities to identify the cause [24]. Hyponatremia usually produces nonspecific EEG slowing; however, full seizure activity is very rare [22]. Since our patient had a generalized tonic–clonic seizure with an identifiable cause and her neurological status got progressively better after the initiation of sodium deficit correction therapy, no further neurodiagnostic tests were applied. During her follow-up over 1 year, no additional seizure activity was recorded.

Several studies investigated the neurological complications of EDs. Structural and the neurochemical alterations in the brain and the cognitive manifestations of these abnormalities in AN patients are among the most common findings. Reductions in gray and white matter volume, ventriculomegaly, enlarged cortical sulci and interhemispheric fissure, cerebellar atrophy, increased cerebrospinal fluid volume and neural response aberrations have all been reported in patients with AN. Further evidence has shown that not all of these changes are completely reversible [25, 26]. These structural and neuronal changes might worsen the neurological consequences of dilutional hyponatremia in patients with AN. Also, AN has been associated with abnormal osmoregulation and impaired urinary concentrating capacity, which might increase the risk of water imbalance. Hypothalamic dysfunction, intrinsic renal failure, antidepressant medications and the duration of AN or the combination of these factors could be the causes of the defective osmoregulation in AN [3]. The hypothesis of an abnormal osmoregulation of antidiuretic hormone (ADH) of central nervous system origin in patients with AN was supported by the studies of Gold et al. [27]. Also severely malnourished states are further complicated by the low solute state presented to the kidney's tubules, resulting from few ingested calories, which leads to inability to excrete free water and thus results in hyponatremia [10]. Our patient's electrolyte imbalance might have been worsened by the existing impaired mechanisms.

Depression was thought to be one of the underlying causes of our patient's ED. Negative affect has been a relatively consistent predictor of ED symptoms but there is also evidence that EDs can result in depression [28]. Two psychosocial stressors for our patient may have contributed to the development of AN; being overweight prior to and being in a deadlock in her education process. Some adolescents who were overweight or obese previously can develop a full ED. Initial attempts to lose weight by eating in a healthy manner may progress to severe dietary restriction [29]. Our patient's illness was triggered by the humiliating words about her weight from a close relative.

We presented this case, because of the amount of water consumed and the extra-ordinary clinical presentation. It may sound unrealistic that an individual consumes 19 L of fluid in a few hours. Depending on our past clinical experiences, it would be expected that this amount of fluid consumption in such a short time period in an adolescent with AN may present to the clinic with vomiting or gastric dilatation. In this present case, we later learned that she had progressively ingested larger amounts of water, but never had that much before. It is interesting that the gastrointestinal system had tolerated this much without vomiting in a case who was expected to have a decreased gastric motility after losing almost half of her body weight with food restriction continuing for a year and later progressing to acute food refusal. In addition, 19 L of water did not work as bowel irrigation, instead absorbed very quickly causing acute and severe hyponatremia leading to central nervous system symptoms and generalized seizures.

In conclusion, any abnormal fluid intake may result in severe clinical complications in patients with EDs. This case confirms that a thorough clinical assessment of hydration and drinking behaviors is essential during the management of EDs and electrolyte abnormalities should be among the initial pre-diagnoses when patients with EDs develop seizures.

Compliance with ethical standards

Conflict of interest On behalf of all authors, the corresponding author states that there is no conflict of interest.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee (Human Studies Subcommittee at the VA Connecticut Healthcare System) and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. This article does not contain any studies with animals performed by any of the authors.

Informed consent Informed consent was obtained from the patient and her parents in this case report.

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