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Exceptional Case

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The mysterious death of the beer-drinking champ: potential role for hyperacute water loading and acute hyponatremia

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ABSTRACT

Hyponatremia is acute when present for <48 h. Most cases of acute hyponatremia involve both excess free water intake and an at least partial urinary free water excretion defect. By contrast, hyperacute water intoxication may result from a large excess electrolyte-free water intake in such a short time that properly working urinary free water excretion mechanisms cannot cope. A hyperacute decrease in serum sodium may lead to death before medical intervention takes place. Well-documented cases have been published in the military medicine literature. In addition, news reports suggest the existence of cases of voluntary ingestion of excess free water by non-psychiatric individuals usually during 'dare' activities. Education of the public is required to prevent harm from these high-risk activities. Adequate training of emergency medical units may prevent lethal outcomes. Spanish media reported the case of a male who died following his triumph in a 20-min beer drinking contest. 'From a heart attack. Man dies after drinking six litres of beer in a contest' ran the news. We now review the physiology underlying hyperacute water intoxication and discuss the potential contribution of hyperacute water loading and acute hyponatremia to the demise of this patient.

Keywords: beer, drinking contest, HAWLAH, hyperacute, hyponatremia, mortality, potomania, water intoxication

TEACHING POINTS

- Most hyponatremia episodes occur in the setting of urinary dilution defects since physiological urinary dilution mechanisms allow the excretion of 16–20 L of water in 24 h.
- However, deaths from excess water ingestion can occur in apparently healthy individuals in the setting of dare activities or forced electrolyte-free water drinking.
- The general population and many healthcare workers are not sufficiently aware of this danger.
- We propose the term hyperacute water loading and hyponatremia (HAWLAH) to describe potentially lethal hyponatremia occurring in the setting of excess electrolyte-free water drinking that because of its magnitude and timing exceeds the physiological water excretion capacity, even in the absence of urinary dilution defects. Dissemination of this

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concept may create awareness about the dangers of drinking huge amounts of electrolyte-free fluids.

BACKGROUND

Hyponatremia is defined as serum sodium (Na) <135 mmol/L. Since normal kidneys may excrete up to 15–20 L of water/day, the development of hyponatremia usually requires the association of excess water intake with an impaired kidney ability to excrete free water. Inappropriate secretion of antidiuretic hormone, kidney disease, drugs and a low osmolar load may impair free water excretion [1–4].

Hyponatremia usually reflects hypoosmolality and leads to water entry to cells, including brain cells, causing symptoms related to the magnitude or duration of hyponatremia [1, 5]. Acute hyponatremia (developing in <48 h) may cause nausea and malaise that usually appear as plasma sodium concentration decreases to <125 mmol/L, followed by headache, lethargy and obtundation (115–120 mmol/L) and eventually seizures, coma and death (<115 mmol/L) [1].

We now explore the death of a beer-drinking champ who died within 80 min of drinking 6 L of beer. After reviewing the literature, we propose the term hyperacute water loading and hyponatremia (HAWLAH) to be used when the magnitude and timing of a free water load results in severe, potentially lethal hyponatremia, despite normal functioning water excretion mechanism that become overloaded.

CASE REPORT

According to media reports, a 45-year-old man died from cardiac arrest after winning a beer-drinking contest that started at 22:00 h at local festivities in Murcia, Spain [6]. Witnesses said he drank 6–7 L of beer in just 20 min. After raising the trophy, he felt sick and vomited and appeared to lose consciencness. 'For ten minutes I was sitting with him, and he was sleeping, he was snoring' recalled a witness. A call to the '112' emergency phone took place at 23:20 h. The patient arrived in cardiac arrest at the emergency room. There was no information on whether necropsy was performed.

Considering the free water load and timing and assuming a baseline plasma sodium of 140 mmol/L, we estimated a decrease in serum sodium of ∼17–18 mmol/L in 80 min after drinking 6 L of beer, for a final plasma sodium of ∼122 mmol/L (Supplementary data, Tables S1 and S2 and online Appendix) [1, 5, 7–18]. This is a conservative estimate since 6 L were used for calculations. Even more conservative estimates provide a range of 11–18 mmol/L decrease in plasma sodium.

METHODS

A Google search for 'death AND water AND drink' performed on 6 August 2013 disclosed several relevant hits (Table 1). The same search in PubMed yielded only the case of a schizophrenic patient [19]. A PubMed search for 'water intoxication AND death' disclosed psychogenic cases, ecstasy and several cases in which the subject was counselled or forced to drink large amounts of water because of ingestion of toxic compounds or the need to fill the bladder or pee, such as workplace illicit drug testing and bladder echography. Table 2 contains some representative cases where the time course and volume of water ingestion was known and there were no other confounding factors such as sweating or vigorous exercise [20, 21].

DISCUSSION

Water homeostasis depends mainly on the regulation of water ingestion by thirst and of renal water excretion by anti-diuretic hormone (ADH). When thirst is overruled by voluntary drinking of free water or by therapeutic administration of free water, only the kidney remains as the last defense against water intoxication. However, the kidney water excretion capacity has limitations, even in healthy individuals. Maximal urine diluting capacity has a lag period of 90 min, since water loading occurs and maximally dilute urine is 50 mOsm/L [1]. Thus, the maximal water excretion rate has an hourly limit that depends on solute excretion (itself dependent on diet), but that may range from 500 to 850 mL for most individuals (Supplementary data, Table S3). A hyperacute water load that exceeds these limits may result in a positive water balance if the water balance was neutral at baseline and in the absence of other water losses. In this regard, it is unusual to drink such large amounts of free water that exceed the maximal water excretion rate of healthy individuals. However, water intoxication may occur within time frames that suggest that maximal urine capacity was not achieved or was not enough to prevent a dangerous decrease in serum sodium, even in individuals without apparent water excretion defects. The two basic scenarios are dare activities, sometimes in public places, such as with the patient under discussion, and compliance with instructions from peers or superiors. Forced intake of excess water is a form of torture [18]. In this regard, the military has enforced rules to prevent hyperacute water intoxication in the setting of public collection of urine and hazing is now forbidden in US universities. However, public and media unawareness of the dangers of hyperacute water intoxication has resulted in a stream of lethal cases that make headlines but are soon forgotten. In all cases, the ingested fluid was water. However, beverages are generally composed mainly of water and may theoretically lead to similar risks. In this regard, the present patient drank a huge volume of beer, an electrolyte-poor beverage.

A PubMed search for 'hyperacute hyponatremia' performed on 6 August 2013 had no hits. However, a repeat search on 22 January 2022 yielded a report from January 2002 in which a decrease in serum sodium from 138 to 119 mmol/L within 7 h was noted after the patient was told to increase water intake and developed stroke-like symptoms [22]. We propose that hyperacute water loading and acute hyponatremia (HAWLAH) is a distinct clinical entity that has been overlooked by the medical community but not by the media. We propose that HAWLAH define symptomatic hyponatremia driven exclusively or mainly by an electrolyte-free water intake overload developing within a time frame that precludes the recruitment of renal adaptation mechanisms, or that vastly exceeds maximally activated urine dilution mechanisms. Naming a problem is the first step towards solving it and we hope that providing a catchy name may help increase public awareness and limit the number of yearly deaths from hyperacute hyponatremia.

A working definition for HAWLAH might be the occurrence of symptomatic hyponatremia within 3 h of the initiation of free water loading. Any time cutoff will be arbitrary for the moment, but after reviewing the literature and considering a lag period from the start of water loading to the absorption of enough water to result in symptoms and the lag period between the initial decrease of sodium concentration and attainment of maximal urinary dilution, a 3-h time frame may provide a working definition that allows further characterization of the syndrome epidemiology and clinical features. HAWLAH would not need inappropriate antidiuresis to result in symptomatic hyponatremia.

Table 1. Representative results from Google searches. A search for 'death AND water AND drink' was combined with additional reports from news sites. Cases 1–5 are most compatible with the concept of HAWLAH

Case	Age (years)/sex	Year	Circumstances	Fluid	Volume drunk (L)/time (h)	Estimated volume drunk in 20 min (L)	Natremia (mmol/L)	Symptoms/outcome
1 (present case) [6]	45/M	2013	Beer drinking competition	Beer	6/0.33	6	Unknown	Vomiting, coma, death within 90 min
2 [23]	5/F	2014	Punishment	Grape soda and water	2.4/1–2	0.4–0.8	Unknown	Vomiting, convulsion, death
3 [23]	12/F	2012	'Water poker' game	Water	6/ND	ND	Unknown	Vomiting, death
4 [25, 26]	28/F	2007	Water-drinking radio station contest	Water	7.6/ > 3	0.85	Unknown	Severe headache/found dead a few hours later
5 [27]	40/F	2008	Dieting[a]	Water	4/2	0.67	Unknown	Headache, death in hospital within 24 h
6 [28]	44/M	2008	Relieve of gum trouble[b]	Water	10/8	0.42	115	Vomiting, unsteady gait, death within 24 h
7 [29, 30]	21/M	2005	University hazing[c]	Water	19/6	1.05	Unknown	Vomiting, seizure, death within 24 h

M: male; F: female; ND: no data.

[a]500 calories a day dieting for 1 week may have decreased urinary solute excretion.

[b]Had been drinking similar daily amounts for the past 48 h, may have decreased urinary solute excretion.

[c]Additional hazing incidents at http://www.scouting.org/scoutsource/HealthandSafety/Alerts/chugging.aspx.

Table 2. Published reports consistent with HAWLAH. In all cases, patients drank water. No relevant medical history reported except obstructive sleep apnoea for the 2011 case [31]

Year	Age (years)/sex	Circumstances	Total volume (L)/time (h)	Estimated volume drunk in 20 min (L)	Initial natremia (mmol/L)	Clinical symptoms/outcome
1983 [32]	40/F	Drank a small amount of diluted household bleach. Poison unit advised to drink large amounts of fluid	15/2	2.5	Initial: 111 (2 h after onset of symptoms), 24 h later: 129	Early: vomiting, confusion, incoherent speech, convulsion. Late: no response to painful stimuli, pulmonary oedema, cerebral and cerebellar oedema. Outcome: death
1985 [33]	21/F	Ultrasound preparation: 42 glasses	10/min	ND	Initial: 127, later (after treatment): 135	Early: headaches, nausea, vomiting, restlessness, tingling in limbs, fell asleep, mother found her not breathing and unresponsive. Late: confused, bizarre behaviour, non-responding, screaming incoherently. Outcome: recovery
1988 [34]	80/F	Fasting for ultrasound preparation	4/h	ND	Initial: 119, 24 h later: 128	Early: confused and uncooperative. Outcome: recovery
1991 [35]	40/F	Urine drug test according to Federal Aviation Administration rules	3/3	0.33	Initial: 121, 24 h later: 140	Early: vomiting, confusion, slurred speech, unsteady gait. Outcome: recovery
2002 [36]	20/F	Urine drug test while under observation	10–12/2–3	1.1–2	Initial: 123, 5 h later: 126	Early: dizziness, headache, incontinence, jerky movements, lost consciousness. CT: cerebral, cerebellar oedema. Late: no response to deep pain, absent gag/corneal reflexes, fixed, dilated pupils. Chest X-ray: diffuse interstitial oedema, increased cardiac enzymes, echocardiogram showed severe dilated cardiomyopathy, diffuse global hypokinesis. Outcome: death
2011 [31]	37/M	Urine drug test while under observation	14/3	1.5	Initial: 122, 90 min later: 132	Early: abdominal pain, acting confused. Late: restless, inarticulate, ‘dry heaves’, ‘seizure-like activity’. Outcome: recovery
1999 [37]	18/M	Army basic training. Hot day, symptoms confused with dehydration. First drank 8 L in 5 h, then 5 L in 2 h and early symptoms began. Then 2 L in 2 h and 10 L in 90 min[a]	20–25/8–11	0.53	Initial: 121 (4 h after onset of symptoms), ICU: 115, 12 h later: 145	Early: vomiting, dizziness, throbbing headache. Late: gait unsteady, confused, lethargic, loss of consciousness, acute respiratory distress, coma, cardiac arrest, brainstem oedema. Outcome: death

M: male; F: female; ND: no data; ICU: intensive care unit.

[a]This is a complex case and it may be disputed whether it corresponds to HAWLAH. However, the misdiagnosis, administration of 10 L of water in 90 min and natremia values suggest that the final episode was HAWLAH.

The rapid onset may make hyperacute hyponatremia even more dangerous than acute hyponatremia, as indicated by animal experiments and media reports. In this regard, the pathophysiology of acute hyponatremia was frequently explored in animal models of hyperacute hyponatremia. In rabbits, a decrease in serum sodium concentration to ∼120 mmol/L in 120 min was lethal and caused severe cerebral oedema [16]. This mirrors the time frame and severity of hyponatremia of the beer-drinking champ. He was not reported to suffer from seizures, a common symptom in severe hyponatremia. However, the ethanol content of the water load may have increased the seizure threshold [1, 5, 17].

Several conditions limit maximal urine dilution capacity, including inappropriately high ADH levels [2, 3]. In the HAWLAH scenario, water overload is massive and within a time frame that prevents significant water excretion even at maximal urine dilution capacity. Thus we propose that HAWLAH would not encompass conditions associated with decreased water excretion capacity, such as beer-drinker potomania, where chronic ingestion of great amounts of beer without food results in decreased urinary excretion of osmoles and thus in reduced ability to excrete water [38], chronic psychogenic polydipsia, which is associated with urinary dilution defects, or excessive fluid intake during prolonged exertion under heat stress, where multiple physiological abnormalities may impair the ability to dilute urine [37]. While individuals with these conditions would be more prone to harm from excess free water drinking, we would prefer that the term HAWLAH emphasizes the risk of activities involving supraphysiological water intake within short time frames by healthy, non-stressed individuals. Discussion of the concept with hospital-based non-nephrological physicians, residents or medical students usually evidenced unawareness of the potential dangers of excess free water drinking.

Some limitations should be acknowledged. The patient arrived in cardiac arrest at the emergency room and it is unclear whether any serum labs were obtained. It was also not made public whether an autopsy was performed. However, we believe that this does not detract from the estimations made and the need to acknowledge and name the syndrome that may lead to increased awareness and potentially, save lives.

In conclusion, we propose the acronym HAWLAH to define acute symptomatic hyponatremia developing because of massive free water loading within a time frame that precludes the recruitment of a renal adaptation mechanism or overrules normal urine dilution mechanisms. News coverage of similar incidents in recent years suggests that harm from HAWLAH in previously healthy individuals may be more frequent than presented in the medical literature. A formal definition of the syndrome that allows recognition by physicians and the public may help address this problem and promote further research and refinement of the concept.

SUPPLEMENTARY DATA

Supplementary data are available at *ckj* online.

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CONFLICT OF INTEREST STATEMENT

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