Compulsive Water Drinking: A Review with Report of an Additional Case

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There is very little information in the literature or textbooks discussing renal physiology and mechanisms of metabolic derangements in compulsive water drinking. This paper is a review of the literature including clinical as well as basic physiological features of the syndrome. An additional case is presented for illustration.

Compulsive water drinking is an uncommon syndrome, although there are numerous case reports in the literature, one as recently as March 1976.[1] The most comprehensive review to date is by Barlow and Wardener in 1959,[2] and almost every publication regarding compulsive water drinking since then uses that review as a basic reference. The youngest patient encountered in the literature with a compulsive-water-drinking problem was three years of age.[3]

Past medical history is usually significant in compulsive water drinking when an accurate history can be elicited. These patients have often had many vague illnesses with more than the usual number of hospitalizations for psychological and organic illnesses as well as a history of psychological disturbances, usually consisting of neurotic traits. Up to 80 percent are neurotic middle-aged females.[4] Common in the history are frequent job changes and episodes of unexplained absence from the place of employment.[2] An unsatisfactory sex life is common is this disorder.[2]

Compulsive water drinking rarely has a sudden onset but has been reported subsequent to or following an hysterical conversion reaction. Usually compulsive water drinking has been occurring for months or years before the diagnosis is considered or confirmed. In one case reported by Barlow and Wardener, the patient had been a compulsive water drinker for 20 years.[2] The actual onset date may be vague or very specific, usually due to a traumatic event such as the death of a loved one, domestic stress, or other major life stresses.

Signs and symptoms vary greatly. Most patients probably manifest only neurotic or psychotic fixations which lead them to excessive water intake. Early symptoms of compulsive water drinking are headaches, vomiting, agitation, and excessive perspiration.[4] Advanced symptoms include muscle twitching, delirium, coma, and convulsions.[4] The progression of symptoms is correlated with the severity of metabolic derangement associated with the fluid and electrolyte imbalance.

Symptoms of compulsive water drinking are a result of cerebral edema as a direct effect of hypotonicity of both the extracellular and secondarily the intracellular compartments.[5] There is evidence that this state can only exist if there is excessive water intake in the face of an impairment of water excretion at the renal level. This probably accounts for the rarity of the full-blown clinical syndrome and will be discussed in greater detail later in this article.

In symptomatic compulsive water drinking, laboratory tests may be helpful in establishing the diagnosis. The serum sodium is uniformly low and in patients who have seizures and coma, the serum sodium has not been reported above 123 mEq/liter.[6] The physical examination provides limited assistance in diagnosing the problem, and findings are usually completely normal in cases of asymptomatic compulsive water drinking. Patients may show agitation and confusion in moderately severe cases, and in advanced cases, there may be profound coma with positive Babinski sign and total unresponsiveness. Patients may manifest psychological disturbances of hysterical behavior or other neurotic symptoms during the interview and examination.

The treatment of compulsive water drinking depends upon the degree of psychological and physiological disturbance at the time the diagnosis is made.[2-4,6] In compulsive water drinking with little aberration of laboratory values (ie mildly depressed serum sodium), the treatment is focused on the underlying psychological disorder and is closely followed up. In these patients reduction of water intake will correct the metabolic disorder. In symptomatic compulsive water drinking without seizures or coma, fluid restriction will usually suffice. Hospitalization for these patients is best for control of fluid intake and for administration of intravenous saline solution if indicated. The main therapy is water restriction, and, when the condition is stabilized, treatment of the underlying psychological disorder. In severe cases with coma and seizures, hospitalization in the intensive care unit is mandatory. Close monitoring of electrolytes with accurate recording of intake and output is essential; then, use of diuretics along with slow intravenous infusion of hypertonic saline is the most effective method of treatment. Phenobarbital, phenytoin, and diazepam may also be required to control seizures.

Physiology Involved in Compulsive Water Drinking

Fluid intake varies greatly among individuals, for reasons that may be ethnic, personal, social, occupational, habitual, or related to thirst mech-

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anisms. The elaboration of urine in varying concentrations keeps water balance regulated to a fine degree. Simply stated, water is a freely permeable substance and with increased intake, the body osmolality decreases, which results in decreased antidiuretic hormone (ADH) and therefore increased water output by the kidney. Conversely, decreased water intake increases body osmolality, which increases ADH and therefore decreases water output to maintain water balance.[7]

The major effect of ADH is to increase the water permeability of the renal distal tubules and collecting ducts. The mechanism by which this is accomplished is not completely understood, but probably occurs via an increase in the adenylcyclase system at the membrane level which increases permeability. By ADH maximizing water permeability, the hypotonic fluid leaving the ascending limb of the Henle loop can approach osmotic equilibrium thereby allowing reabsorption of water. This reabsorbed fluid produces more isotonic fluid delivered through the collecting ducts which causes the urine to become more concentrated as the level of ADH increases.[7]

The physiological regulation of water excretion is maintained by the counter current system which is described in any basic physiology textbook.[8] The mechanism is dependent upon movement of solute across the membrane in the ascending limb of the Henle loop, which is impermeable to water. Whether the final urine is concentrated or diluted, the counter current mechanism is the same. However, the concentration at the medullary level varies greatly from up to 1,200-millosmolars per liter (mOSM/liter) with water deprivation to about 700 mOSM/liter in diuretic conditions.

The main reason for the large variation in medullary concentration is the presence or absence of ADH. The distal tubule and collecting ducts are relatively impermeable to water in the absence of ADH and therefore a dilute urine can be produced. On the other hand, a concentrated urine can be produced with increased ADH.

The counter current system of the medulla is the foundation for excretion of widely varying urine concentrations, but it is ADH that controls the fine adjustments of the mechanism via its effect on the permeability of the distal tubule and collecting ducts.

Factors other than ADH that affect urine concentration are: (1) total nephron population, in which a decrease in population may limit the amount of dilute urine that is formed or the maximum urinary concentration that is achievable; (2) very large solute loads; (3) increased medullary blood flow; (4) enhanced proximal tubular reabsorption, which may impair concentrating ability by decreasing the rate of delivery of the filtrate to the Henle loop and the distal nephron; and (5) other poorly understood intrarenal factors.[7]

ADH release is *stimulated* by decreased extracellular fluid volume, pain, anxiety, and certain central nervous system depressant drugs. ADH release is *inhibited* by ethyl alcohol, expansion of the extracellular fluid compartment, and paroxysmal atrial tachycardia (this is probably a result of the left atrial stretch receptor reflex).

The etiology of disorders of urine-concentrating ability include central diabetes insipidus, both complete and partial, and nephrogenic diabetes insipidus, both congenital and acquired. In compulsive water drinking the normal concentrating ability may be markedly impaired due to the lowering of the medullary concentration profile by chronic "washing out" of the renal medulla.[7]

In compulsive water drinking there is polyuria, polydipsia, and a very low circulating level of ADH; therefore, it can be difficult to differentiate between compulsive water drinking and central diabetes insipidus. However, marked hypo-osmolality is not commonly seen in compulsive water drinking since the rate of fluid intake rarely exceeds the capacity of the kidney to excrete water. In normal renal function, the kidney can excrete 15 to 20 liters of water per day, so a person must exceed this intake in order to retain water. Therefore, many renal physiologists believe that in compulsive water drinking there is no physiological disturbance in the absence of impairment of urinary diluting ability.[7]

Hyponatremia itself usually indicates a disorder of urinary dilution. Retention of excess water will decrease the solute concentration in the body and since sodium is the major solute, its concentration will be decreased. There are a few disorders which can give a factitiously low serum sodium, including hyperglycemia, high serum lipids, and very high serum proteins such as are found in multiple myeloma. When hyponatremia and hypo-osmolality are not factitious, then a cause for impaired urinary diluting capacity should be sought.

Serum osmolality of less than 255 mOSM/liter indicates overhydration and suggests psychogenic water drinking. Serum osmolality of greater than 300 mOSM/liter suggests dehydration, and therefore in the face of polyuria, one must consider central or nephrogenic diabetes insipidus. Intermediate values may be found in psychogenic water drinking or in diabetes insipidus.

With 24-hour water restriction, normal people will lose one to two percent of body weight, urine osmolality will increase to approximately 800 mOSM/liter, and urine flow will decrease to about 0.5 cc per minute. In compulsive water drinking, due to the "washing out" of the renal medulla, the patient may lose up to five percent of body weight and the urine should at least become isotonic. Vasopressin (ADH) administration at the height of the water restriction test will increase urine osmolality in central diabetes insipidus; however, in compulsive water drinking urine concentrations will not increase further. The most sensitive test is radioimmunoassay for urinary ADH with water restriction; however, this is at present a research tool and not readily available to the practicing physician.

Behavioral Perspective

From the behavioral perspective the very name of this syndrome indicates an emotional problem. The term "compulsive" almost immediately triggers the cognition of "compulsive personality," a psychological state describing an individual who attempts through some fantasy or realistic device to maintain an illusion of control over his or her functioning. This is done by avoiding commitments and decisions and never exposing oneself to the possibility of failure, thus avoiding the awareness of imperfection, fallibility, and humanness.[9]

Also, "compulsive personality" is frequently used as a synonym for

"obsessive personality." The obsessional person's way of life is largely organized to meet the dilemma created by the hypocrisy and ambiguity characteristic of an early familial life situation. The self-system of the obsessional person develops in a climate of hostility, rejection, and power struggles hidden beneath a facade of loving care and concern.[10]

This developmental background leads to the passive, inhibited nature of these patients or to their occasional need for compulsive acting out.

The obsessive-compulsive personality may also indicate persons who need to satisfy or placate the needs of others in order to feel secure; they may unconsciously resent the demands they believe others place upon them.[11]

One can easily be taken in by the use of the word "compulsive" in compulsive water drinking. In reality the psychopathology reported in the literature covers the entire spectrum of disorders from acute, hospitalized schizophrenics to mildly neurotic, depressed housewives.

It is the latter condition on this continuum that would seem to characterize the patient whose case history follows. However, this case may be an example of "complaint" water drinking as opposed to compulsive water drinking because the background and personality of this patient seem strong and free of psychological problems. She had been told years ago that drinking large quantities of water was the only way she could combat a chronic condition of constipation. Her resulting assumption that an improvement in her condition required consumption of more water led to her eventual chemical imbalance.

Case Presentation

A 74-year-old wife of a physician was admitted to the medical intensive care unit comatose. She had vomited prior to admission. She showed no response to painful stimuli and had a left Babinski sign and ankle clonus bilaterally. Reflexes were hyperactive but the remainder of the physical examination was unremarkable and vital signs were stable. Her past history was significant in that she had been hospitalized six months earlier after experiencing a grand mal seizure and was treated with phenytoin and phenobarbital and had experienced no further seizures. Electroencephalogram, brain scan, and skull films were normal at that recent admission. She also had undergone a radical mastectomy for breast carcinoma 14 years earlier and had no evidence of recurrence or metastasis since the operation.

At this admission an ECG was immediately performed, as was a chest x-ray, urinalysis, complete blood-count, and SMA-18. All were essentially normal with the exception of serum sodium of 114 mEq/liter and chloride of 80 mEq/liter. Urine specific gravity was 1.002. With this data the possibility of metabolic coma was considered and she was treated with intravenous normal saline. She began to awaken over the next 12 hours and was completely bright and alert in 18 to 24 hours. Intravenous fluids were discontinued when the patient began eating solid food, and over the next 48 hours all the electrolyte studies were normal and the patient was completely asymptomatic with the disappearance of the ankle clonus and Babinski sign.

Nephrological and neurological consultations were obtained and the patient underwent studies to confirm compulsive water drinking as the etiology of her disorder. A random urine osmolality was 12 mOSM/liter (normal 390-1,090 mOSM/liter), and five days later it was 548 mOSM/liter with a normal water intake. A urine concentration test was performed which proved that with water restriction she could concentrate her urine. A serum osmolality was not measured until she was recovering and it was within the low range of normal at 282 mOSM/liter (normal 275-300 mOSM/liter). At the same time, the urine osmolality was still low at 281 mOSM/liter (normal 290 to 1,090 mOSM/liter).

After she returned to full alertness, she admitted to drinking 10 to 14 glasses of water a day to "soften her stool." She also drank coffee and tea in addition to the water. She had no other neurotic traits and was completely in touch with reality.

One year later, she is and has been asymptomatic. She responded to patient education about her disorder, and random serum sodiums drawn in the office have been consistently in the 140 mEq/liter range. This indicates her acceptance and understanding of this self-induced problem.

Summary

Symptomatic compulsive water drinking is uncommon. The pathophysiology of compulsive water drinking is essentially that of an acute organic brain syndrome from cerebral edema.

The syndrome does not occur clinically unless there is an impairment of water excretion, since normal renal function allows up to 20 liters per day excess intake without accumulation.

It has been reported that about 80 percent of cases are neurotic middle-aged females. In almost all cases there is at least a history of neurotic symptoms or maladaptive symptoms if the history is probed. In most reported cases the patients were psychotic.

When symptoms and laboratory aberration are mild, the sole treatment is water restriction. In moderate but very symptomatic cases, treatment includes both intravenous saline and water restriction. In severe cases treatment includes water restriction, intravenous hypertonic saline, diuretics, and anticonvulsants. After the acute phase, patient education may suffice as in the above case, but if psychosis is present, appropriate therapy is indicated accompanied by frequent checks of serum electrolytes to detect early aberration.

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