ORIGINAL COMMUNICATIONS

DEHYDRATION IN THE ELDERLY: A SHORT REVIEW

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Dehydration is the most common fluid and electrolyte problem among the elderly. The usual causes of water loss are frequently absent in dehydrated elderly patients. Age-related changes in total body water, thirst perception, renal concentrating ability, and vasopressin effectiveness probably predispose to dehydration. Dehydration related to infection, high-protein tube feedings, cerebral vascular accidents, and medication-related hypodypsia are particularly relevant for elderly patients. Appropriate treatment depends on accurately assessing the water deficit and slowly correcting that deficit.

As the population ages, all physicians, especially those working in urban settings, will be treating an increased number of elderly patients. Of particular importance to black physicians is a well-recognized "crossover" in longevity among black patients. While the life expectancy for blacks of all ages is less than whites, those blacks reaching old age have a higher probability of reaching very old age than whites. Thus, it is important to understand the common medical problems associated with aging.

Dehydration is the most common fluid or electrolyte disturbance among the elderly. While there are still sizable gaps in our knowledge about the incidence, pathophysiology, and treatment of dehydration, recent insights into the pathophysiology, as well as the recognized increase in morbidity and mortality associated with dehydration, make it a subject of interest to all physicians. The term *dehydration* is commonly used to refer to two fluid and electrolyte disorders. The first is water depletion or free water deficit, and the other is sodium depletion, where there is a decrease in sodium and water.[1]

Even though there is very little argument among clinicians that dehydration is a common problem in the elderly, there is little in the literature to support estimates of the incidence of dehydration either as a complication or as a primary disorder. One study based on a retrospective review of a large county hospital's records estimated the incidence of dehydration among noninfected elderly to be 3.21 per 1,000, and among infected elderly to be 11.6 per 1,000.[2] An ongoing study of dehydration among elderly nursing home residents indicates that the incidence is four to five per 1,000 nursing home residents.[3] Apart from being a common problem, dehydration is associated with significant mortality. Two studies of hospitalized patients with dehydration reported mortality rates of 46 to 48 percent.[2,4]

The reasons for elderly people being prone to dehydration are not fully understood. Dehydration is a known complication of disease states associated with salt and water loss, such as vomiting or diarrhea. As the majority of elderly with dehydration do not have such disease states, the incidence of dehydration in the elderly cannot be adequately explained on the basis of water loss.[5] A review of the normal physiology of sodium and water homeostasis, as well as how it

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changes with age, provides some insight into the causes of dehydration among the elderly.

NORMAL PHYSIOLOGY

Total body water and serum osmolality are controlled over a narrow range through mechanisms that regulate thirst and arginine vasopressin (AVP) release. While thirst and AVP respond similarly to changes in plasma volume and serum osmolality, the two have separate osmoreceptors located in the preoptic and anterior nuclei of the hypothalamus. Under conditions of normal hydration the pituitary secretes AVP to maintain the serum osmolality. This corresponds to a measured AVP level of 1.5 to 2.0 μU/mL.[6] When dehydration occurs, the serum osmolality increases. Above an average threshold of 280 mOsm/kg, secretion of AVP increases in a continuous fashion three- to five-fold.[6] Increases in AVP alter the permeability of the renal collecting tubule, thereby increasing the urine to plasma osmolality ratio and conserving free water.

Osmoreceptors for thirst are located near the receptors for AVP. Like the AVP receptors, the thirst receptors are stimulated as water diffuses out of the receptor cell in response to hypertonic extracellular fluid.[7,8] The amount of fluid consumed in response to thirst is related to its severity; however, thirst appears to be relieved before the water deficit has been completely corrected.[9] Satiation of thirst is related to subjective changes in the oropharynx, changes in gastroduodenal distention, as well as mechanical, thermal, and chemical factors.[7]

Thirst and AVP secretion are also stimulated by hypovolemia through one of several baroreceptors. A decrease in plasma volume of 10 percent or more causes an increase in AVP secretion as a result of changes in the frequency of afferent impulses[6] from baroreceptors in the carotid sinus, aortic arch, left atrium, and great pulmonary veins. Similarly, any reduction in arterial or venous pressure due to decreased cardiac output, venous pooling, or positive pressure breathing stimulates thirst and AVP secretion.[6,8] Hypertonicity is a potent stimulator of AVP secretion, evidenced by the fact that a hypertonic solution infused into the carotid artery of a euvolemic or hypervolemic animal will induce AVP secretion.[6,7] Thirst and AVP secretion are also stimulated by high levels of renin and angiotension,[8] increased body temperature,[8,9] physical exercise,[9,10] and prostaglandin.[6] While these stimuli may be secondary to changes in volume and osmolality, they contribute to water homeostasis. A summary of the stimulators and inhibitors of thirst and AVP secretion is listed in Tables 1 and 2.

CHANGES IN PHYSIOLOGY WITH AGING

Aging seems to alter water physiology in four ways. First, the concentrating ability of the aged kidney is decreased. Elderly subjects deprived of water for 12 hours do not significantly decrease their urine flow,[11] and when compared with young subjects, their urine osmolality is substantially lower.[11,12] Because young subjects both decrease their urine flow and increase their osmolality, there is a considerable difference in the maximum urine osmolalities attained by young and old subjects under conditions of water deprivation. While renal failure can lead to a decrease in concentrating ability in any age group, among the elderly, a decreased concentrating ability is not necessarily correlated with diminished creatinine clearance.[11]

The second age-related alteration in water physiology is that AVP is less effective in the elderly. There is controversy concerning the effect age has on the level of AVP. Early studies indicated that AVP levels,[13,14] as well as renin levels,[15] were lower in the elderly. A more recent study using radioimmunoassay to measure AVP indicates that AVP levels are normal to high in the elderly, and that when angiotension II levels between young and old dehydrated subjects are compared, there is no difference.[12] While there is disagreement about the level of AVP, there seems to be agreement that the relative ineffectiveness of AVP is at least partially due to an abnormality of the AVP receptor in the kidney.[10,12,13] The hypothesis that the changes with aging are at the receptor level is supported by studies that measured low urine osmolality in response to vasopressin injection or infusion.[10,13]

Third, total body water decreases with age. Total body water in men aged 23 to 54 years is 54.3 percent ± 1.39, and in women, it is 48.6 percent ± 1.47. With aging, total body water decreases to 50.8 percent ± 1.55 in men aged 61 to 74 years, and to 43.4 percent ± 1.32 in women aged 61 to 74 years.[6] The decrease is distributed between the extracellular and intracellular water, but is more pronounced in the intracel-

TABLE 1. REGULATION OF THIRST AND ARGININE VASSOPRESSIN RELEASE

Stimuli	Inhibitors
Hyperosmolality of extracellular fluid	Hyposmolality of extracellular fluid
Decreased arterial pressure	Increased arterial pressure
Decreased pressure of left atrium or great pulmonary veins	Increased pressure of left atrium or great pulmonary veins
Pain	Emotional stress
Increased body temperature	Decreased body temperature
Drugs (see Table 2)	Drugs Ethanol Phenytoin
Stimulation of renin angiotension system	
Psychosis	

TABLE 2. DRUGS THAT STIMULATE ARGININE VASSOPRESSIN RELEASE

Known Stimulators
Cholinergic agents
Acetylcholine
Methacholine
Barbiturates
Morphine
Nicotine
Possible Stimulators
Vincristine
Chlorpropamide
Prostaglandin E1
Clofibtrate
Tricyclic antidepressants

lular water. Of course, the smaller the amount of total body water, the smaller the amount of water deprivation needed to change osmolality. This may partially explain why elderly subjects have a higher serum osmolality and serum sodium after 24 hours of water deprivation than younger subjects.[12] Nonetheless, most would agree that the documented changes in total body water are less important than the changes in responsiveness to AVP.

Fourth, the elderly have an altered sensation of thirst.[7,10,12,14] It has been known for some time that lesions of the hypothalamus are associated with decreased thirst. In addition, some elderly patients with nonhypothalamic cerebral vascular accidents who do not have significant language or motor deficits that might limit their ability to get fluid have been found to have repeated episodes of dehydration, which could only be explained by a deficit of thirst perception.[14] Furthermore, a study involving both healthy young and old subjects who were deprived of water for 24 hours and who developed serum hyperosmolality revealed that the elderly had much lower perceptions of thirst than the young.[12] According to a subjective rating scale, some elderly subjects did not feel thirsty at all, while most of the young subjects were very uncomfortable because of thirst. When these same elderly subjects were presented with water, they did not drink, whereas the young subjects drank enough water to correct whatever alterations in serum osmolality had occurred as a result of the water deprivation. These changes in water physiology that occur with aging may be the basis for the characteristically sluggish response to physiologic stress that Leaf has observed in the elderly.[1]

DIAGNOSIS AND TREATMENT

Clinically, it is important to understand the distinction between water depletion vs salt and water depletion because the two show distinct clinical findings; misunderstanding the distinction can lead to delayed recognition of water depletion. Water depletion is characterized by hypernatremia, hyperosmolality, and high urine osmolality, except when the water depletion is secondary to nephrogenic diabetes insipidus. Intravascular volume is preserved (except in cases of very large water deficits) by movement of water from the extracellular space to the intravascular space.[1,16] Similarly, the extracellular space is maintained at the expense of the intracellular space. Therefore, when body water decreases, the intracellular space is affected first and most significantly, and clinical signs of intravascular compromise such as orthostatic blood pressure findings are not common until profound water depletion has occurred.

Sodium and water depletion, on the other hand, is characterized by elevated BUN and BUN/creatinine ratios and hemoconcentration with normal, high, or low serum osmolality and sodium. Because there is intravascular hypovolemia and an overall decrease in extracellular volume, orthostatic blood pressure changes and decreased skin turgor are common physical findings.[1] One must bear in mind, however, that because of decreased subcutaneous fat and skin elasticity, change in skin turgor is often an unreliable finding in the elderly. While the relative frequencies of water depletion and sodium depletion are not well documented in the literature, one study of 56 dehydrated patients found water depletion among 10 percent.[2]

The causes of dehydration, in general, are well known (Table 3), but are not always easy to define in the elderly. Only a few observations about the underlying causes of dehydration in the elderly have been made. Infections of any kind, particularly pneumonia and urinary tract infections, are especially common in elderly patients presenting with dehydration.[2,4,16] It has yet to be determined whether there is a causal relationship. In one study, 82 percent of dehydrated elderly had an infection.[4] The mortality rate of elderly patients with infection and dehydration is high, ranging from 40 to 50 percent.[4,16] As infection may present without fever or leukocytosis in the elderly, recognition and treatment may be delayed, thereby contributing to the high mortality rate. This observation has led one author to recommend empirically treating with antibiotics all dehydrated elderly patients for whom a cause cannot be determined.[4]

Four additional causes of dehydration in the elderly are worthy of mention. First, dehydration as a result of diseases that are predominately seen in the elderly, such as nonketotic hyperosmolar coma or nephrogenic diabetes insipidus secondary to amyloid or multiple myeloma, should be considered. Second, high-protein tube feedings (1 g/kg of body weight per day) when given for more than nine to ten days have been associated with dehydration because of the high nitrogen load and resulting osmotic diuresis.[17] The kidney requires 40 to 60 mL of water for every gram of nitrogenous waste. One gram of protein generates roughly 14 g of nitrogenous waste. Therefore, a person receiving high-protein tube feedings will require additional water above the maintenance volume and careful monitoring of urine output to avoid dehydration. In most of the reported cases of dehydration associated with tube feeding, the patients were getting only 2 L of fluid each day and the onset of dehydration was insidious. Third, recurrent, profound dehydration has been described in six patients with a remote history of cortical cerebral vascular accidents.[14] These patients did not have evidence of any hypothalmic lesions, nor were the causes of their cerebral vascular accidents known. Finally, certain medications that are commonly used among the elderly decrease drinking and may therefore predispose to dehydration. Cardiac glycosides and amphetamines fall into this category.[8] Cardiac glycosides are among the most frequently prescribed drugs for the elderly. Amphetamines, although no longer drugs of choice, are sometimes used for the treatment of refractory depression in the elderly.

TABLE 3. CAUSES OF DEHYDRATION

Sodium and Water Depletion
Diuretics
Adrenal insufficiency
Renal salt wastage
Vomiting and/or diarrhea
Alkalosis with bicarbonaturia
Excessive sweating
Burns
Water Depletion
Fever
Central diabetes insipidus
Nephrogenic diabetes insipidus
Essential hypernatremia
Hypodipsia with partial diabetes insipidus
Osmotic diuresis

The complications of dehydration and its treatment are not trivial. Hypernatremia is associated with mental status changes ranging from lethargy to coma.[16] The mechanism is thought to be that extracellular hyperosmolality inhibits acetylcholine, consequently blocking neural transmission.[16] In extreme cases, a rapid rise in serum sodium concentration can cause petechial hemorrhages and thrombotic occlusion throughout the cortex and white matter. The osmotic gradient causes rapid movement of water from brain cells to the extracellular space and the resulting shrinkage of the brain puts stress on veins, potentiating tears and hemorrhage.[18] The elderly suffer mental status changes associated with electrolyte abnormalities that younger people tolerate quite well.[1] Another complication of dehydration is irritation of muscles and the central nervous system, leading to seizures.[19] The decrease in extracellular and intravascular volume that can occur with dehydration alters drug effects and predisposes to acute renal failure and to cardiovascular collapse.[16-18,20]

TABLE 4. TREATMENT OF DEHYDRATION IN THE ELDERLY

Replace one half of water deficit in the first 24 hours, and the remainder over the next 48 hours
Dextrose 5% and $\frac{1}{2}$ normal saline is the preferred solution in most cases
Use normal saline for severe intravascular compromise
Avoid dextrose 5% in water
Monitor daily weights
Adjust renally excreted medications

In the absence of diabetes insipidus, the treatment of dehydration in the elderly is conceptually no different from treatment in younger patients (Table 4). However, it should be done more slowly to prevent the most serious complications of treatment, cerebral edema and congestive heart failure. Diabetes insipidus is rare, requires special therapeutic consideration, and will not be discussed here. Fatal cases of cerebral edema are more likely when dehydration is corrected within 24 hours.[18] The total water deficit is best corrected over two to three days. In cases of hypernatremia, a hyponatremic solution should be used. Five percent dextrose in water (D5W) is the least favored, as diffuse extravascular edema is more common when water deficits are corrected with D5W. In cases of salt and water depletion, (D5W1/2NS) 5 percent dextrose and 1/2 normal saline should be used except in cases of severe intravascular collapse, when normal saline is the preferred solution. The total body water deficit should be calculated. To calculate the water deficit, calculate the current volume and normal volume:

Current volume

$$= \frac{140 \times 0.5 \times \text{current body weight (kg)}}{\text{measured serum Na (mg/dL)}}$$

Normal volume

$$= 0.6 \times \text{normal body weight (kg) in the young}$$

but

$$0.45\text{–}0.50 \times \text{normal body weight in the elderly}$$

$$\text{Water deficit} = \text{normal volume} - \text{current volume}$$

The usual recommendation is that no more than one half of the deficit should be corrected in the first 24 hours, and the remainder over the next 48 hours. This may be too rapid, however, for very frail elderly patients or for those with severe hypernatremic dehydration. Weighing patients daily is an inexpensive, easily accessible way to follow the progress of therapy. Testing for serum electrolyte levels daily is essential during repletion, but should not be overutilized once the patient is stable.

Prevention, of course, is preferable. Physicians should be aware of the increased potential for dehydration in the elderly. They should encourage fluid intake among all elderly who do not require fluid restriction, monitor fluid intake of elderly patients with episodic illnesses, prescribe tube feedings with an appropriate amount of fluid, and, of course, initiate rehydration early when dehydration does occur.

In conclusion, dehydration is common among the elderly, and is associated with significant morbidity and mortality. Aging may decrease thirst, change the effectiveness of AVP, reduce renal concentrating ability, and decrease total body water, all of which probably contribute to the elderly's propensity to become dehydrated. Neurologic complications are the most dreaded. Fluid replacement should be slow (48 to 72 hours) and D5W1/2 normal saline is the preferred solution in most cases.

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