

My Doctor Said I Should Drink a Lot! Recommendations for Fluid Intake in Patients with Chronic Kidney Disease

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Patients with chronic kidney failure commonly are advised to maintain a generous fluid intake. The reason that physicians tend to recommend such an increased fluid intake is difficult to understand. A scientific basis does not exist. To the modern nephrologist, this element of the therapeutic armamentarium seems long to be outdated; however, it was once recommended by authorities (1) and is still being advocated by primary care physicians.

“My wife forgot to ask whether it is really necessary to keep a fluid intake of 4.5 L daily. This was the advice of her primary care physician. Perhaps 3 L would be better for her BP and sufficient for her kidney.” The husband of the patient raised this question at the end of her consultation visit at our University Hospital Clinic. The patient was seen for a second opinion regarding the management of her chronic kidney disease (CKD) manifested by a serum creatinine of 3.0 mg/dl and non-nephrotic proteinuria. The husband, who is an oncologist, was mainly concerned because his wife was losing sleep at night because of her many nocturnal trips to the bathroom. If the oncologist and his wife were an isolated case, then we would not be writing this article. However, over the years, we have encountered numerous such examples in our nephrology consultative practice. We suggest further that the patients who have CKD and complain about the instruction to drink large amounts of fluid may represent only the “tip of the iceberg.” Likely, there are many more patients who have CKD and whom we nephrologists never hear about because they silently accept their watery fate. Without complaint, they follow the advice of their primary care physician, which is reinforced by the lay press (“drink at least 8 glasses of water a day”) and by well-meaning friends who urge the patient with CKD to “push fluids” to clear the waste from their system. In light of the above, the purpose of this article is to place in modern perspective the widely held but incorrect notion that high fluid intake is a good thing in those with CKD.

Patients with CKD commonly are advised by primary care

physicians or lay people to maintain a generous fluid intake. Moreover, in daily practice, there even seems to exist a direct correlation between serum creatinine and prescribed daily fluid intake. The higher the serum creatinine, the higher the prescribed fluid intake, the upper limit in our experience being approximately 4 L/d. Indeed, two recent authoritative publications recommended “increased” fluid intake in the management of CKD (2,3). The reason that physicians—in almost all cases, non-nephrologists—tend to recommend such an increased fluid intake is difficult to understand. An increased fluid intake is not supported from modern evidenced-based medicine. The origin of this peculiar medical behavior seems to be merely historic. In the early years of renal physiology, it was shown that urinary urea clearance was sharply increased as urine flow rates increased from 1 to 2 ml/min (4). Lower blood urea nitrogen from chronic high fluid intake seems to be the basis for the historic recommendation to maintain urine volumes up to 4 L in chronic kidney insufficiency. Contemporary evidence of a beneficial effect of a high fluid intake in chronic kidney insufficiency is provided only by studies in rats. Increasing fluid intake suppressed maladaptive renal hypertrophy and interstitial fibrosis (5). Studies by Bankir *et al.* (6) in animal models of kidney disease showed that water restriction that results in high antidiuretic hormone levels promotes progression of kidney disease in part because antidiuretic hormone induces glomerular hyperfiltration. However, as mentioned by Bankir herself, marked species differences exist in the osmotic work that is required to concentrate urine. Rats concentrate their urine much more than humans because rats have more nephrons with long loops of Henle than humans. This may explicate why high fluid intake is beneficial in rats, whereas data in humans are lacking (7). What is the treating physician aiming at when he recommends a high fluid intake? Three historic misconceptions can be extracted from daily practice.

Misconception 1. An expanded plasma volume raises renal perfusion, which increases urine production, facilitating enhanced excretion of creatinine and urea. This assumption is valid only in the case of prerenal kidney failure caused by dehydration. Overuse of diuretics, fluid loss as a result of diarrhea or high fever, and inadequate hydration are examples. The decrease in kidney function is functional and fully reversible after administration of fluids. In euvolemic patients, high

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Table 1. Relationship between solute and water intake in those with normal or impaired kidney concentrating ability

	Normal	Impaired
Urine concentrating capacity		
Urine osmolality (mOsmol/kg)	1200	600
Solute excretion (mOsmol)	800	800
Water needed = urine volume (ml)	670	1340
Water balance (food + oxidation – water loss; ml)	200	200
Drinking (ml)	470	1140

fluid intake may lead to volume expansion and arterial hypertension, especially when accompanied by high salt intake.

Misconception 2. The kidney needs a moist environment to work well, and a high urine output indicates a better kidney function. The second misconception engages all patients with CKD. Does a scientific reason exist for this group to improve kidney function by an increased intake of fluid? The kidney needs only a surprisingly small amount of fluid intake to work well. The daily solute load that needs to be excreted by the kidney averages 800 mOsmol. The working range of a healthy kidney is from 50 (maximal diluted urine) to 1200 (maximal concentrated urine) mOsmol/kg. Therefore, the healthy kidney needs a urine output of 670 ml to excrete 800 mOsmol of waste. A kidney with a 50% reduction in the concentrating ability will require a daily urine output of 1340 ml (Table 1). However, not all of this excreted fluid comes from drinking. Our daily fluid balance consists of (1) fluid intake as drink and that contained in food and (2) water generated in the metabolism. In addition to daily urine output, fluid is lost

from the body as insensible losses (skin and airways) and a small amount *via* the stools. Accordingly, the person with the healthy kidney in our example would require a daily fluid intake of only 470 ml to maintain fluid balance (8,9). The patient who has CKD and a maximal concentrating capacity of 600 mOsmol/kg would need only 1140 ml of free fluid drinking (Table 1). However, humans usually drink more than this minimum requirement for social and cultural reasons.

Misconception 3. Patients with all forms of kidney stone disease benefit from a high fluid intake. The third point aims at patients with recurrent nephrolithiasis. This is the only group of patients for whom scientific evidence exists that they benefit from ample fluid drinking. In these patients, increasing fluid intake to above 2 L/d will increase the urine flow rate and lower the urine solute concentration, both of which protect against stone formation (10,11). The misconception is that nephrolithiasis and kidney disease are often used synonymously by lay people. This may be another explanation for why the general public believes in the benefits of a high fluid intake for almost everybody with CKD.

There are a few clinical entities that need an increased fluid intake. A list to identify who requires high fluid intake and who does not is shown in Table 2. Patients who need an increased fluid intake can be separated into three categories: (1) Those who need high fluid intake to prevent disease such as nephrolithiasis; (2) those who need a high fluid intake to compensate for a underlying disease, such as renal or central diabetes insipidus or salt-wasting nephropathy; and (3) those who have conditions such as polydipsia or salt gluttony (12), where the underlying disease should be treated.

Table 2. Conditions associated with high water intake: Those in which high water intake is indicated or not indicated^a

Condition	Comment
Conditions for which high fluid intake is indicated to prevent disease uroolithiasis	Generally, fluid intake should achieve a urine volume of 2.0 to 3.0 L/d
Conditions for which high fluid intake is recommended because of underlying disease salt-wasting nephropathy (medullary cystic disease, other causes of chronic interstitial kidney disease)	Very rare disease; high salt intake (e.g., >400 mM/d NaCl) may be required to avoid hypotension; because these patients cannot appropriately concentrate the urine, high fluid intake (e.g., >4 L/d) may be needed
central and nephrogenic diabetes insipidus (genetic or acquired, e.g., lithium nephrotoxicity)	These patients often cannot raise urine to levels isosmotic with plasma; often large water intakes (e.g., >5 L/d) are needed to maintain water balance and a normal plasma osmolality
Conditions for which high fluid intake is not recommended inappropriately high NaCl intake ('salt gluttony') in the patient with CKD	The high NaCl intake "drives" the fluid intake; 24-h urine collection for creatinine (to assess completeness of the collection) and NaCl (to assess NaCl intake) will detect this condition; the appropriate management is to decrease salt intake
primary polydipsia because of the mistaken belief that high fluid intake is good for the kidney	This practice should be discouraged

^aCKD, chronic kidney disease.

So far we have dealt with the fact that increasing the fluid intake does not increase kidney function, but is there evidence that an increased fluid intake may even harm the kidney? In humans, there has been no prospective study of the effect of fluid intake on kidney disease progression. However, a retrospective analysis of the Modification of Diet in Renal Disease Study disclosed a significant association between 24-h urine volume and decline in GFR. It was shown that kidney patients with a high daily fluid intake (urine volume 2.4 L/d) have an accelerated loss of kidney function compared with patients with a lower fluid intake (1.4 L/d). This loss of kidney function was independent of other risk factors (13,14). It is noteworthy that there were no signs of renal salt or water wasting in those patients with the highest urine volume. Thus, the patients with the highest fluid intake were “pushing fluids.” It was concluded that in CKD, there was no evidence of benefit of a high fluid intake. Indeed, although a cause-and-effect relationship between high fluid intake and faster GFR decline could not be established, by these association studies, high fluid intake was shown to be an independent risk factor. A hypothesis that could explain how high urine volume might cause faster kidney disease progression is that high urine volume increases intratubular volume and pressure, and these stretch forces could induce fibrogenic mechanisms (14). Another hypothesis that has been put forward is that an increased fluid intake leads to intravascular volume expansion and eventually to an increase in BP, which is one of the major factors of kidney disease progression (15,16).

This does not mean that it is beneficial for the kidney to restrict fluid intake. However, we advocate that there is no advantage to increasing the daily fluid intake above what the thirst sensation tells you. The thirst mechanism is one of the most delicately regulated body systems and works very predictably. However, it should be noted that age-related changes in thirst sensation increase the susceptibility for dehydration in the elderly (17), particularly in the female elderly (18).

In this context, it is of interest that it was reported recently that excessive consumption of fluids can cause hyponatremia in marathon runners (19). In fact, many organizations are beginning to revise their recommendations that fixed, large volumes of dilute fluids be consumed during athletic competition. It is now suggested that athletes use thirst as their guide for fluid replacement. This is a major change in guidelines (from “stay ahead of your thirst” to “replace sweat loss”) (20).

Conclusion

Patients with CKD should not “push fluids.” Normal thirst-guided intake should determine water intake, unless there is a specific reason to increase fluid intake (Table 2). There is no evidence of a beneficial effect of a high fluid intake. Indeed, there is evidence that it could cause harm by promoting progression of kidney disease.

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