

Fluid Intake and Urinary Tract Infection

Pages with reference to book, From 81 To 81

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Physicians often recommend that patients with UTI increase their fluid intake. This practice has continued for years largely unchallenged because it is so logical: a water diuresis would serve to “flush” the urinary tract of infecting organisms and the increased frequency of voiding would reduce bacterial multiplication in the bladder. However, since dilution of urine affects both the osmolality of the renal medulla and the action of urinary inhibitory substances that participate in natural host defense mechanisms¹, it is of interest to re-examine fairly the evidence supporting this common treatment advice. Andriole, in a controlled animal study showed that enterococcal pyelonephritis in rats could be cured by a sustained water diuresis for 7-14 days without antimicrobial therapy². It was postulated that reduction of hypertonicity of the renal medulla and infiltration of PMNs improved host response to bacterial infection. In 1971, an uncontrolled study of nursing home residents with bacteriuria who were not necessarily symptomatic suggested that hydration and frequent voiding reduced urine bacterial load³. These studies possibly provided the historical rationale for increasing oral fluid intake in patients with TJTI and yet there is an apparent deficiency of additional reports in the literature evaluating the use of hydration alone or as an adjunct to antibiotic therapy in the treatment of TJTI. Although it is clearly important to avoid dehydration, it is essential to consider the potential hazards of forcing fluids, as well as reasons that it may not be necessary. Antibiotherapy is an important component of a treatment regimen for symptomatic UTI. Many antibiotics are satisfactorily concentrated in the urinary tract and, if undisturbed, are effective in eradicating sensitive organisms. Forced-water diuresis could theoretically impair the effect of some antibiotics because their concentration in the urine is substantially reduced with aggressive hydration⁴. Indeed, other treatment protocol such as making the urine alkaline (citrate, bicarbonate) alleviates dysuria and potentiates the activity of sulphonamides⁵ but may significantly reduce the action of most quinolones, currently the preferred urinary drugs, which function only poorly at alkaline pH^{6,7}. Besides, urine with a pH below 5.5, in contrast, tends to be antibacterial and many substances that can acidify urine (ascorbic acid, ammonium chloride, methionine and mandelic acid) can produce antibacterial activity⁸. Also, forcing oral or intravenous fluids can cause clinically significant hyponatremia, especially in the elderly and the chronically ill, because of their diminished renal tubular function and inability to clear free water^{9,10}. In individuals with diabetes, intervention with diuretics to reduce hypertension is associated with excess mortality¹¹. Indeed, the authors have concluded that until there is a clinical trial showing a beneficial effect of diuresis in diabetic patients, there is an urgent need to reconsider its usage in this population. In conclusion, it is considered important to treat symptomatic UTI promptly with appropriate antibiotics or urinary antiseptics and to maintain clinically normal fluid status. Overzealous hydration, especially in the elderly, may not be beneficial and can be harmful, while in the average, patient it may dilute the antibiotic and “hurry” it through the very tract where it was intended to exert its effect.

References

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