Acute Renal Failure After Binge Drinking and NSAID Use

To the Editor:

In their recent report of a patient who developed acute renal failure after binge drinking and nonsteroidal anti-inflammatory drug (NSAID) use, Johnson and Wen suggest that ethanol may have contributed to volume depletion by having induced a water diuresis (1). However, as patients with diabetes insipidus have taught us, a water diuresis alone is not sufficient to cause volume depletion—there must also be inadequate water replacement. Although the patient described did have nausea and vomiting, I doubt that she had large unreplaced free water losses. Had this been the case, one would have predicted that her serum sodium concentration would have increased. The fact that her serum sodium level was normal on two occasions indicates that her free water losses and gains were equal. Thus, if she had been truly volume depleted, her net losses must have been isotonic, as may occur when hypotonic losses (e.g., through vomiting) are partially replaced by water. It is also surprising that this patient did not have orthostatic tachycardia or hypotension, since her large rapid weight loss suggested marked volume depletion. Although this unusual syndrome of acute renal failure and flank pain after the use of NSAID has now been well described, its pathogenesis and the role of ethanol remain unclear.

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Response:

We agree with Dr. Spital that volume loss in our patient was isotonic, as judged from the normal serum sodium concentration (1). In our report, we did not intend to indicate that alcohol-induced water diuresis was the sole basis for the volume loss. We implied that it can be an initial precipitating factor. More important, binge drinking commonly leads to nausea and vomiting, a state of repeated volume losses via gastric and renal routes with poor replacement, which are probably largely responsible for the subsequent volume loss. In our patient, the volume loss was substantial, as shown by a weight loss of 9 lbs in 6 days, along with the relatively low blood pressure at 110/54 and 104/50 mm Hg in supine and erect positions, respectively, and an initially low fractional excretion of Na of less than 1% in the face of renal failure. We would like to emphasize that nonsteroidal anti-inflammatory drug (NSAID)-induced acute renal failure can occur even with a subtle volume contraction without orthostatic hypotension (2). With milder volume contraction, reversible prerenal failure rather than acute tubular necrosis may occur. We suspect that such subclinical cases are not uncommon among the binge drinkers who use NSAID but may escape clinical attention. Thus, we believe that NSAID-induced renal hemodynamic alterations in the presence of volume loss are the most likely pathogenetic mechanisms of acute renal failure in our patient. However, we do not rule out other unidentified contributory factors that may be associated with the ingestion of a large amount of alcohol.

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