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Effect of Tolvaptan treatment on acid-base homeostasis in ADPKD patients

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To the editor:

We read with great interest the recent report of Heida et al[1] on the impact of tolvaptan on acid-base status in ADPKD patients. The authors hypothesized that V2 receptor antagonism may induce a state of metabolic alkalosis by activation of V1 receptors through increased circulating vasopressin, which in turn would stimulate renal net acid excretion (NAE). The authors found no changes in plasma bicarbonate after tolvaptan administration and therefore concluded that tolvaptan does not affect renal NAE.

Recent studies[2,3] indicate, however, that urinary citrate is superior to plasma bicarbonate for the detection of subtle acid-base alterations. We previously observed that ADPKD patients treated with tolvaptan exhibit a significant reduction in renal net acid excretion (NAE), with an increase in urinary citrate[4]. These changes were associated with increased net gastrointestinal alkali absorption (NGIA; a marker of alkali intake) but unaltered urinary sulfate (a marker of acid intake), suggesting that lower NAE may be due to higher alkali intake or gut alkali absorption in patients taking tolvaptan. Thus, our data suggest that tolvaptan treatment is associated with changes in renal NAE and hence acid-base homeostasis in ADPKD patients. The underlying mechanisms, however, remain to be elucidated.

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