Assessing acid retention

Troels Ring

1Department of Nephrology, Aalborg Hospital, Aalborg; and 2The Water and Salt Research Center, Aarhus University, Aarhus, Denmark

TO THE EDITOR: Wesson and coworkers (4) make a commendable attempt at explaining the interesting beneficial effect of alkali administration to ameliorate progressive renal failure. They conclude that patients with moderately reduced renal function already have proton retention, which may have clinical consequences. They demonstrate an alkali-sensitive mechanism in terms of endothelin and aldosterone, which, based on their previous work, could possibly be involved.

The strategy of the authors to infer acid-base status based on the dynamic response to alkali load is very attractive. The authors find that patients with two degrees of renal failure have similar renal net acid excretion (NAE) and also similar “acid intake.” The latter is not measured in the classic way (2) but indirectly. They do not measure (or comment on) endogenous acid production (1), yet from their findings they conclude that the more severely afflicted stage 2 chronic kidney disease (CKD2) patients do have acid retention. Despite similar blood values of pH, Pceo, and total CO2, and despite similar load and excretion of acid, they measure it. The method employed by the authors to reveal H+ retention in CKD2 compared with CKD1 consisted of demonstrating greater NAE after similar loads of HCO3 in the latter compared with the former. In CKD1, the acute alkali load (0.5 meq/kg) lowered NAE from 24.7 ± 2.9 to 9.5 ± 3.3, while in CKD2 the change was much smaller, from 24.6 ± 5.0 to 18.2 ± 5.1. Then, following 30 days of oral alkali (0.5 meq/kg), and after the same acute alkali load again, NAE in CKD1 was unchanged at 9.8 ± 3.4, while in CKD2 it declined compared with the acute load without pretreatment to 13 ± 4.8.

We are not told what NAE was after 30 days of oral alkali but without the acute load, but since in CKD1 the NAE is unchanged after the acute load it appears that 30 days of alkali did not change NAE in CKD1. Since blood values were also reported to be unchanged, the physiology of the response noticed during the acute load appears problematic. Insofar as the response was related to acid-base physiology, it seems an anomaly that it only occurs in the acute setting. NAE was measured in the classic way (2) over an 8-h period, but it was not specified which component(s) of NAE caused the difference between the groups or over what time period. Also, since it has previously been pointed out (3) that NAE may be influenced by mechanisms felt to be fundamentally indifferent to the control of pH, the argument for acid retention in CKD2 might have been more plausible if there was a relationship between baseline NAE and the suggested acid retention across the spectrum of estimated glomerular filtration rate.

In conclusion the strategy to study acid retention in CKD in the paper by Wesson et al. (4) is very interesting. How to model the findings in an optimal way is still a question.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author.

REFERENCES