LETTERS TO THE EDITOR

Effect of obesity on PD versus HD survival: Is caloric intake the discriminating factor?

To the Editor: Higher body mass index (BMI) and its positive association with dialysis patients’ survival has become an area of interest [1]. Abbott et al [2] have recently published an article suggesting that any paradoxical survival advantage observed with obesity in hemodialysis (HD) patients may not be seen in patients on peritoneal dialysis (PD). This contradicts an earlier report by Snyder et al [3], but Abbott et al’s finding is probably more reflective of the long-term. If the finding by Abbott et al is to be confirmed (i.e., obesity in PD is not associated with any long-term survival advantage), then one has to ask the interesting question as to why there may be a “paradox within paradox.”

We would like to propose an intriguing and plausible hypothesis that involves difference in the caloric intake. In general, all PD patients, obese or nonobese, employ 1.5% to 4.25% of dextrose in their dialysate, often around the clock, that is estimated to be absorbed at 45% [4]. In contrast, HD patients are exposed to 1% of dextrose in their dialysate during the 4-hour, thrice weekly dialysis. Therefore, the higher caloric intake, rather than obesity, per se, may account for the better survival of dialysis patients, and this may help to explain why nonobese patients on PD may not display any less survival advantage compared with their obese counterparts (Fig. 1). A higher caloric intake by dialysis patients for many conceivable reasons may contribute to longer survival. Anyone for a controlled trial?

SHARIQ AHMAD and ABDULLA K. SALAHUDEEN
Jackson, Mississippi

Correspondence to Abdulla K. Salahudeen, MD, Department of Medicine, University of Mississippi Medical Center, 2500 North State Street, Jackson, MS 39216–4505.
E-mail: asalahudeen@medicine.umsmed.edu

REFERENCES


On the epithelial-mesenchymal transition of mesothelial cells

To the Editor: In a recently published review article, Williams et al [1], on behalf of the Biopsy Registry Study Group, to which some of us belong, raise several criticisms regarding the paper published by our group in the New England Journal of Medicine [2] without first consulting us or taking into account our opinion. In general, these comments are not scientifically based and appear to be personal opinions.

Williams et al remarked only on our data regarding the down-regulation of cytokeratin expression and the increase in vimentin expression by effluent mesothelial cells when compared to “in situ” cells, sentencing that these markers are modulated by the simple “ex vivo” culture of mesothelial cells, questioning the epithelial-mesenchymal transition phenomenon itself. With this simplistic point of view, the authors left out additional and much more important markers, which confirmed the epithelial-mesenchymal transition in our study (i.e., down-regulation of E-cadherin, induction of snail nuclear factor, and changes in integrin expression).

Furthermore, they affirm to be “unable to identify in vivo similar fibroblastic phenotypic changes in the