Plasma Lipoprotein(a) Is Related to Peritoneal Albumin Losses

Reply to the Letter to the Editor by Montenegro et al.

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Dear Sir,

With interest we read the letter by Montenegro et al. concerning the relation between lipoprotein(a) [Lp(a)] levels and peritoneal albumin losses. Their findings of lack of a significant correlation between Lp(a) levels vs. plasma albumin, peritoneal or total albumin losses, are in apparent contradiction to our previous findings, where we reported significant correlations between Lp(a) vs. peritoneal and total clearance as well as excretion for albumin and β 2-microglobulin [1]. In addition, we found a relation between Lp(a) levels and peritoneal glucose absorption [1]. We wish to emphasize that the correlation coefficients of 0.35-0.50 in our study indicate that the above factors only would account for a minor part (10-25%) of the observed variation in Lp(a) levels.

Apart from our study, others have investigated the relation between Lp(a) levels and peritoneal losses. In a study of 64 CAPD patients by Wanner et al. [2] Lp(a) levels correlated significantly to daily protein and albumin losses. Also in hemodialysis, a correlation between serum albumin levels and Lp(a) levels has been reported [3]. It is well established that Lp(a) levels are markedly increased in patients with severe protein losses due to nephrotic syndrome [4]. An interesting recent finding lending further support to a link between protein losses and Lp(a) in CAPD is that partial correction to hypoalbuminemia in CAPD patients by daily albumin infusion markedly decreased Lp(a) levels [5]. Thus, although the underlying mechanism for a link between protein metabolism and Lp(a) in CAPD remains to be elucidated, a number of studies suggest the presence of such an association.

The assay methodology of Lp(a) has not been standardized. This fact, together with the pronounced interindividual variation in Lp(a) levels and apolipoprotein(a) size as well as the impact of ethnicity are obviously important when comparing results between studies. At present it can not be excluded that these factors could contribute to the apparent contradiction between the results in our study [1] and the study by Montenegro et al. For example, the impact of apolipo-protein(a) size has been analyzed only in the study by Wanner et al. [2], and therefore we can not evaluate at present to what extent variations in apolipoprotein(a) polymorphism might contribute to the differing results. The importance of variation in the apolipoprotein(a)

gene for plasma Lp(a) levels was recently underscored by the finding that a polymorphism in the non-coding part of the gene affected plasma Lp(a) levels differently depending on ethnicity [6]. Whether such variations influence plasma Lp(a) levels in CAPD remains to be shown.

In conclusion, a number of studies support a relation between Lp(a) levels and albumin losses in CAPD. We acknowledge, however, that the response in Lp(a) levels in CAPD may be heterogeneous and possibly modulated by genetic factors, and we believe that further studies addressing these mechanisms are warranted.

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