Dear Sir,

In reference to the contribution from the Lambert group [1], we have recently obtained similar data [2,3] also in agreement with Baba et al. [4], but different from those of Mathiesen et al. [5]. In fact, a statistically significant reduction of erythrocyte anion charge (RBCCh) was observed in insulindependent diabetic (IDD) patients with albuminuria of less than 300 mg/day. Although no linear relationship existed between 24-hour albuminuria and RBCCh, we found a good indirect correlation between RBCCh and the albuminuria peak after inhibition of proximal tubular reabsorption of proteins by the lysine test [6] (r =-0.77; p < 0.001).

Studying the RBCCh determinants, we observed that, while the sialic acid content was normal, a significant reduction of the RBC membrane content of glycosaminoglycans (GAGs) was present [2, 3]. We think that this anomaly is related to the generalized derangement of the GAG metabolism in diabetes mellitus, which seems to be crucial for the development of diabetic microangiopathy and particularly diabetic nephropathy [7, 8]. The abnormal urinary excretion of GAGs seems to be another marker of this metabolic disorder [9–11]. However, contrary to what was stated by Bernard et al. [11], we believe that the higher urinary excretion of GAGs in diabetes could be an early marker of abnormal glomerular charge permeaselectivity. Actually, patients in our investigation characterized by the greater reduction of RBCCh (which seems to signify a greater abnormality of glomerular charge permeaselectivity) also had a higher urinary excretion of GAGs (χ² = 9.48; p < 0.005) [unpubl. data]. Moreover, another recent investigation of ours has shown that a lysine provocative test [6] induced a significant pathological urinary excretion of albumin in IDD patients with abnormal urinary GAGs and albuminuria within the normal range, in contrast to IDD subjects with normal urinary GAGs and albumin [12].

In conclusion, we think these observations support the role of GAG metabolism in the pathogenesis of diabetic nephropathy and the value of abnormal urinary excretion of GAGs as an index of glomerular involvement.

References


Bruno Baggio, Istituto di Medicina Interna, Via Giustiniani 2, I-35128 Padova (Italy)


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