Dear Sir,

Recently, Bommer et al. [1] reported that nephronal obstruction by β2-microglobulin-derived material may be causally related, at least in part, to the genesis of acquired cysts in the end-stage kidney. We have measured serum and cyst fluid β2-microglobulin levels using a radioimmunoassay method in 3 patients with acquired cystic disease of the kidney and 2 patients with simple cyst. As shown in table I, the β2-microglobulin levels in the cyst fluid are very low in acquired cystic disease, as compared with respective sera. We have already found that there is a high ratio of cyst fluid to serum creatinine and that cyst fluid sodium is similar to that of serum [2]. On the other hand, Birenboim et al. [3] reported that β2-microglobulin, creatinine and sodium levels of the proximal cyst in autosomal dominant polycystic kidney disease are similar to those of the respective sera. These results suggest that proximal cysts in acquired cystic disease of the kidney continue to function at a higher level of differentiation than proximal cysts in autosomal dominant polycystic kidney disease. However, the relationship between low β2-microglobulin levels of cyst fluid in acquired cystic disease and the formation of β2-microglobulin-derived material which causes nephronal obstruction in uremic patients remains unknown.

References


Table I. β2-Microglobulin levels in acquired cystic disease and simple cyst