Dear Sir,

Acquired cystic disease of the kidney is a disease first identified in the process of the remarkable advance in the treatment of chronic renal failure [1, 2] and has recently been delineated as a pathological and clinical entity [3]. Cyst fluid chemistries in acquired cystic disease of the kidney have not been reported so far. The analysis of cyst fluid chemistries may contribute to knowledge of the pathogenesis of acquired cysts, i.e. whether acquired cystic disease is related to simple cysts or adult-type polycystic kidney disease, or whether it is related to neither of them.

We analyzed 17 acquired cysts in 2 autopsy cases (47 and 58 years of age, males) who had been on hemodialysis for more than 10 years (10 and 13 years) due to chronic glomerulonephritis. Sodium, potassium, chloride, urea nitrogen, creatinine, and total protein were measured in cyst fluid and serum by automatic analysis (Smac®; Tech-nicon). The serum levels were obtained at steady state (prior to dialysis) and agonal state or 30 min after death. The cyst fluid was obtained at autopsy, which was performed within 30 min or 2 h of the patients’ demise. Three patients with simple cysts and 1 patient with polycystic kidney disease were also evaluated as controls.

The results are listed in table I. Sodium, potassium, chloride and urea nitrogen of the cyst fluid in acquired cysts were similar to those of the respective sera. The sodium of cyst fluid in acquired cysts was 138–160 (146.1 ± 6.0 mEq/l; mean ± SD). The ratios of cyst fluid to serum creatinine were high, 7.1 and 5.4, respectively. The creatinine levels of cyst fluid in acquired cysts were

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Number of patients analyzed</th>
<th>Number of cysts analyzed</th>
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<tbody>
<tr>
<td>Simple cyst</td>
<td></td>
<td></td>
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<tr>
<td>Polycystic kidney disease on hemodialysis</td>
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</tbody>
</table>
Acquired cystic disease of end-stage kidney

\[ 7_{10} \]

\[ 7_{10} \]

\[ 1.0 \]

\[ 1.1 \pm 0.04a \]

\[ 1.1 \pm 0.03 \]

\[ 1.2 \pm 0.04\text{c} 0.9 \pm 0.02 \]

\[ 2.2 \]

\[ 1.5 \pm 0.22 \]

\[ 0.5 \pm 0.05 1.5 \pm 0.22 \]

\[ 1.2 \]

\[ 1.3+0.12 \]

\[ 1.1 \pm 0.04 \]

\[ 0.8 \pm 0.07 \]

\[ 1.2 \pm 0.04 \]

\[ 1.1 \pm 1.31 5.4+ 1.50 \]

\[ \pm 0.96 5.8+ 1.61 \]

\[ 0.7 \]

\[ 0.1 \pm 0.08 0.2 \pm 0.36h \]

\[ 0.1 \pm 0.09 0.2 \pm 0.54h \]

Serum value at steady state was used.
Including 2 hemorrhagic cysts.
Serum value at agonal state or at autopsy.

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28.5–77.0 mg/dl (57.5 ± 15.7 mg/dl). The origin of cysts in polycystic kidney disease was defined by the ratio of cyst fluid to serum sodium according to Huseman et al. [4], i.e. the ratio of cysts fluid to serum sodium of a proximal cyst is 0.8–1.2 and that of a distal cyst less than 0.4. Our results of cyst fluid composition were in accord with those obtained in proximal cysts in polycystic kidney disease [4–6] except for the creatinine level. All 17 cysts showed sodium concentrations similar to those of serum, and there were no cysts with low sodium concentration compatible with distal cysts in polycystic kidney disease. The high creatinine level in an acquired cyst was rather similar to that of a distal cyst in polycystic kidney disease [4]. Our results differ from the cyst composition of simple cysts [7] because of a high ratio of cyst fluid to serum creatinine. These findings might be used for differential diagnosis between acquired and simple cysts. However, any other specific site of the nephron [8] other than the proximal tubule was not indicated from our results. Our results suggest that an acquired cyst is a focal dilatation of the functioning proximal tubule, including an altered hyper-plastic epithelium, with nonionic simple diffusion and ion trapping [9] or a similar mechanism for creatinine. Another speculation is that creatinine in the cyst fluid is only derived from glomerular filtration, and some specific concentration mechanism for creatinine is present in proximal cysts [4]. Communication to the glomerulus [9] was suggested in the majority of acquired cystic disease cases from this high creatinine concentration of cyst fluid. Therefore, this reasonably explains why a significant
number of acquired renal cysts in dialyzed patients regress rapidly after successful renal transplantation [3]. In conclusion, acquired cystic disease of the kidney is not multiple simple cysts, and all 17 acquired cysts analyzed in this study showed a high ratio of cyst fluid to serum creatinine with the character of a proximal cyst.

References


Addendum

I have now observed a third patient with acquired cystic disease of the kidney who also showed the high ratio of cyst fluid to serum creatinine and cyst fluid sodium similar to that of serum.