pole, and contribute to crescent formation. Recent studies support the second possibility [1–6]. It was reported that chemokines produced by proximal tubular cells promoted the infiltration [3,4]. Proximal tubular epithelial cells activate urinary complement proteins in situ and contribute to the mediation of tubulointerstitial injury [6]. The tubular epithelial cell is the major site of M-CSF production within the injured kidney; macrophage accumulation and local proliferation can occur in the tubulointerstitium in the absence of glomerular inflammation [2]. Proximal tubular cells also promote fibrogenesis by transforming growth factor-β1-mediated induction of peritubular myofibroblasts [1]. Most important is that recent studies performed on cultured cells and experimental nephropathies suggest the possibility of epithelial–mesenchymal transition of tubular epithelial cells, i.e. transdifferentiation. One study, done on a human renal biopsy, also suggested such a transdifferentiation [7]. Finally, is it possible that proximal tubular cells transdifferentiate and migrate towards the glomerular urinary pole and contribute to crescent formation?

Conflict of interest statement. None declared.

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Ladislava Grcevska1
Croatia is not spared from diabetic nephropathy

Sir,
In the article by Rutkowski [1], Croatia was pointed out as having a peculiarly low proportion of patients with diabetes
Adult polycystic kidney disease in patients on haemodialysis in the south of Brazil

Sir,

About 5–10% of chronic dialysis patients have adult dominant polycystic kidney disease (ADPKD). Few epidemiological data on this disease are available in Brazil. The purpose of our investigation was to study the prevalence of ADPKD in Porto Alegre, a city in the south of Brazil.

Case. We studied patients in 15 haemodialysis centers, searching in particular for patients who had a family history and imaging findings compatible with the diagnosis of ADPKD. The control group was composed of patients who were also on dialysis but did not have evidence of this hereditary disease.

Of the 975 adult patients that composed the study population, 74 had ADPKD as the primary cause of chronic renal failure, corresponding to 7.6% of the total dialysis patient population in Porto Alegre (Table 1).

Comment. In Brazil, epidemiological data about ADPKD have only been collected for the state of São Paulo, showing a prevalence rate of 3% in dialysis patients [1]. This prevalence is well below that found in our study and also those in American and European studies. This may be a consequence of either incomplete assessment or a different ethnic composition of the population in São Paulo.

Similar findings to ours have been published in other countries. In the USA, 8–10% of dialysis patients have this diagnosis [2]. In European studies, the prevalence of this pathology is about 10% [3]. As the majority of the Porto Alegre population is of European descent, we can state our data are compatible with data from other white populations. A lower prevalence is found in Asia, where only 2.5–3.2% of dialysis patients have ADPKD [4].

ADPKD affects both sexes similarly [4]. In our sample, there were more men (53%) than women (47%). This difference, though not significant, may be partially explained by the higher number of men than women in chronic dialysis programmes.

Table 1. General sample characteristics

<table>
<thead>
<tr>
<th></th>
<th>ADPKD patients (n = 74)</th>
<th>Controls (n = 901)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>53.8 ± 11.0</td>
<td>53.2 ± 16.1</td>
<td>0.77</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male (%)</td>
<td>39 (53)</td>
<td>517 (57.8)</td>
<td>0.44</td>
</tr>
<tr>
<td>Female (%)</td>
<td>35 (47)</td>
<td>384 (42.2)</td>
<td>0.44</td>
</tr>
<tr>
<td>Ethnic background</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White (%)</td>
<td>51 (85.7)</td>
<td>578 (74.3)</td>
<td>0.44</td>
</tr>
<tr>
<td>Black (%)</td>
<td>23 (14.3)</td>
<td>320 (25.4)</td>
<td>0.44</td>
</tr>
</tbody>
</table>

Conflict of interest statement. None declared.

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