Increased Serum Angiotensin-Converting Enzyme in End-Stage Renal Disease

KW. Rumpf
A. Brat
V. Armstrong
F. Scheler

Department of Nephrology, University Hospital, Göttingen, FRG

K.W. Rumpf, MD, Department of Nephrology, University Hospital, Robert-Koch-Strasse 40, D-3400 Göttingen (FRG)

Dear Sir,

We read with interest the article by Silverstein et al. [1] concerning serum angiotensin-converting enzyme (SACE) activities in patients with renal disease. Contradictory results have been published on this topic in the past [2–5].

We would like to communicate our own data relating to SACE in 34 patients with end-stage renal failure on maintenance hemodialysis treatment. These data partially confirm the results of Silverstein et al. [1]. Moreover, they may give some insight into the mechanisms underlying the increase of SACE of this patient population.

Our results are given in table I. It may be seen that in the patient group as a whole SACE [6,7] was significantly (t = 3.0898; p < 0.005) elevated as compared to normal controls. This is consistent with the findings of Silverstein et al. [1] and earlier results of Patel and Ansari [485x379]↓[491x379]↑[494x379].

Our group of 34 hemodialysis patients had been especially selected to comprise 20 patients with consistent hypotension (predialysis systolic blood pressure value consistently < 100 mm Hg). Further analysis of our data showed that SACE was significantly (t = 3.4539; p < 0.005) elevated in the hypotensive patient group only, whereas in the hypertensive/normotensive patient group only a slight elevation of SACE of borderline significance (t = 2.0367; p = 0.055) was detected. This finding is similar but not identical to the results of Silverstein et al. [1], who found elevated SACE activities in patients with low postdialysis blood pressure. Since some of our patients had evidence of liver disease (serological evidence of hepatitis B, alcoholic liver disease, or slightly elevated transaminase activities of unknown origin) – a situation in which increased SACE activities have been described

[8] – we further analyzed our patient data as to the presence of liver disease. If these patients were excluded from the calculations, SACE was still elevated significantly (t = 2.2637; p < 0.05) in the remaining patients. However, patients with liver disease exhibited the highest SACE values, which differed highly significantly (t = 5.3650; p < 0.001) from controls and also from patients without liver disease (t = 2.4486; p < 0.025). When patients with liver disease were excluded from both the hypertensive/ normotensive and the hypotensive patient group, patients

Table I. Serum angiotensin-converting enzyme (SACE) activity in patients with end-stage renal failure on maintenance hemodialysis and in normal control (mean ± SD)

<table>
<thead>
<tr>
<th></th>
<th>Rumpf</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Brat</td>
</tr>
<tr>
<td></td>
<td>Armstrong</td>
</tr>
<tr>
<td></td>
<td>Scheler</td>
</tr>
</tbody>
</table>

[1]Supported by Deutsche Forschungsgemeinschaft (SFB-89-Kardiologie).
with hypotension still had significantly (t = 2.6585; p < 0.02) increased SACE values, whereas in patients with normal or high blood pressure values SACE showed only a borderline, nonsignificant (t = 1.1851; p < 0.3) increase.

Increased Serum Angiotensin-Converting Enzyme in End-Stage Renal Disease


We conclude from our data that elevated SACE values will be frequently found in patients with endstage renal failure on maintenance hemodialysis treatment. However, this may only partially be due to kidney failure per se as has been proposed by Silverstein et al. [1] and by Patel and Ansari [2]. Our data presented above suggest 4 that both liver disease and hypotension, which are often found in the dialysis patient population, may be implicated in the development of high SACE values. The association of high SACE and hypotension is of special interest.

Although this association may be far from causal 6 and rather circumstantial, the mechanism underlying this association merits further elucidation. In summary, our results may explain, why some authors [1,2] found elevated SACE activity in patients with renal failure, whereas others [3] did not. They do not explain, however, why two other groups of investigators [4, 5] found decreased SACE activities in patients with renal disease.