Renal Failure

α-Amylase and Isoamylase Levels in Renal Transplant Recipients Compared to Uremic Patients

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CLINICAL STUDY

α-Amylase and Isoamylase Levels in Renal Transplant Recipients Compared to Uremic Patients

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ABSTRACT

Hyperamylasemia is a common finding in chronic renal failure (CRF) patients. The present study was designed to evaluate the frequency, the type, and the hyperamylasemia levels in renal transplant recipients (RTR) compared to patients with renal failure with or without replacement of renal function. One hundred and forty-one subjects (42 with varying degree of renal insufficiency [group A], 74 on hemodialysis [group B], and 25 RTR [group C]) and 47 normal individuals were studied. Total serum α-amylase (Ta) as well as pancreatic (Pa) and salivary (Sa) types of serum isoamylases were elevated in all groups when compared to the levels found in normal subjects. A remarkable proportion of patients belonging to groups A and B had Ta as well as Pa levels over three times the upper normal limits. On the contrary, no RTR had such increased levels of both Ta and isoamylases. A statistically significant correlation was found between Ta, Pa, and Sa and serum creatinine in RTR. However, no statistically significant correlation was found between urine amylases and serum creatinine or between urine and serum levels in all amylases in this group. In conclusion, serum amylase levels are increased in RTR. However, no

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subject in this group had amylase and isoamylase values more than three times
the upper normal limits, which was a common finding in the other groups of
patients.

INTRODUCTION

Studies have demonstrated that serum levels of total α-amylase and isoamylases are
raised in the majority of patients with chronic renal failure (CRF) with or without replace-
ment of renal function (1–3). It has been stated that renal transplant recipients (RTR) also
have increased serum levels of total α-amylase, which was ascribed to drug-induced
subclinical pancreatitis (4–7). However, no detailed description of the values of iso-
amylases or of urine total α-amylase and isoamylases was reported in this group.

The present study was designed to evaluate the frequency, the type, and the hyper-
amylasemia levels in patients with varying degree of CRF as well as in RTR without any
evidence of acute pancreatitis.

MATERIALS AND METHODS

One hundred and forty-one subjects [42 (group A) with varying degree of renal insuffi-
ciency, 74 (group B) on hemodialysis (HD), 25 (group C) RTR], as well as 47 normal
individuals (control group) were studied. None of them had any clinical manifestation of
acute pancreatitis nor was any alcoholic. Subjects with diabetes mellitus were excluded
from the study. Most patients of the groups A and B were taking antiacid tablets and
multivitamin preparations. Antihypertensive drugs, such as α-methyldopa, beta-blockers,
ACE inhibitors, and calcium channel blockers were received by 50% of the patients and
RTR. Ten percent of the group A patients were on small to moderate doses of furosemide.
Prednisone 8–12 mg/day, azathioprine 100–150 mg/day, and cyclosporine A 3–5 mg/kg/
day were administered to group C. The mean (± 1SD) serum creatinine was 5.4 ± 1.2 mg/dL
and 2.2 ± 0.9 mg/dL for the groups A and C, respectively. In all groups serum total
α-amylase (Ta), and pancreatic (Pa) type and salivary (Sa) type of isoamylases, as well as
the urine isoamylases (Tu, Pu, Su, respectively) were measured. In HD patients blood
samples were collected before the beginning of an ordinary hemodialysis session. In order
to measure the urine α-amylases, a 24-h urine specimen mixed with albumin (1 mg/mL of
urine) was used. The blood and urine specimens were stored in −20°C until the determina-
tion day of α-amylases. Measurements of α-amylases were performed by the Phadebas
isoamylase test.

Statistical analysis was done by the analysis of variance method and by Student’s t test.
Linear regression analysis was performed to evaluate the correlation between parameters.

RESULTS

Serum Ta and isoamylase levels are shown in Table 1. One-way analysis of variance
revealed that serum Ta and isoamylases are increased in both patient groups (A and B)
compared to the control population. However, comparison between groups A and B did not
prove any statistically significant difference. Ta and Pa levels of RTR were significantly
Table 1

Serum α-Amylase and Isoamylase Levels

<table>
<thead>
<tr>
<th>Studied Groups</th>
<th>α-Amylases (u/L), ( \bar{x} \pm SD )</th>
<th>Controls ((n = 47))</th>
<th>A ((n = 42))</th>
<th>B ((n = 74))</th>
<th>C ((n = 25))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ta</td>
<td>( \bar{x} \pm 1 SD )</td>
<td>183 ± 54</td>
<td>531 ± 392*</td>
<td>452 ± 180*</td>
<td>333 ± 106*</td>
</tr>
<tr>
<td>Pa</td>
<td>( \bar{x} \pm 1 SD )</td>
<td>84 ± 30</td>
<td>392 ± 268*</td>
<td>306 ± 170*</td>
<td>208 ± 55*</td>
</tr>
<tr>
<td>Range</td>
<td></td>
<td>30–164</td>
<td>120–1383</td>
<td>82–1364</td>
<td>123–293</td>
</tr>
<tr>
<td>Sa</td>
<td>( \bar{x} \pm 1 SD )</td>
<td>98 ± 55</td>
<td>171 ± 164**</td>
<td>138 ± 90**</td>
<td>125 ± 69</td>
</tr>
<tr>
<td>Range</td>
<td></td>
<td>0–239</td>
<td>0–682</td>
<td>0–362</td>
<td>22–309</td>
</tr>
</tbody>
</table>

*p < 0.0001, **p < 0.005 between the groups A, B, C and the normal controls.

Elevated compared to those of normal subjects \((p < 0.0002)\) but statistically significantly lower than those of group A \((p < 0.003, p < 0.006)\) and B \((p < 0.001, p < 0.001)\). However, Sa levels of group C were not statistically different from those of normal subjects or from those of groups A and B.

Hyperamylasemia \( (> \bar{x} \pm SD) \) was present in 41/42 (98%) patients of group A, 74/74 (100%) of group B, and 23/25 (92%) of group C (Table 2). Moreover, a remarkable proportion of patients belonging to groups A and B had Ta as well as Pa levels over three times the upper normal limits. On the contrary, no RTR had such increased levels of both serum α-amylase and isoamylases.

Urine α-amylase levels are shown in Table 3. Even though Tu, Pu, and Su levels were significantly decreased in the groups A and B compared to the control population, there were no statistically significant differences in the values of urine isoamylases between RTR and control subjects. Moreover, urine amylases of RTR were not different from those of the other groups of patients.

Table 2

Percentages of Subjects With Hyperamylasemia

<table>
<thead>
<tr>
<th>Group</th>
<th>Ta a (%)</th>
<th>c (%)</th>
<th>Pa a (%)</th>
<th>c (%)</th>
<th>Sa a (%)</th>
<th>c (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>98</td>
<td>14.2</td>
<td>98</td>
<td>40</td>
<td>48</td>
<td>9.5</td>
</tr>
<tr>
<td>B</td>
<td>100</td>
<td>14.9</td>
<td>98.3</td>
<td>35</td>
<td>41</td>
<td>0</td>
</tr>
<tr>
<td>C</td>
<td>92</td>
<td>0</td>
<td>100</td>
<td>0</td>
<td>28</td>
<td>0</td>
</tr>
</tbody>
</table>

Note. a = % subjects with values > \( \bar{x} \pm 1 SD \); c = % subjects with values > \((\bar{x} \pm 1 SD) \times 3\).
Table 3

Urine α-Amylases Levels

<table>
<thead>
<tr>
<th>Studied Groups</th>
<th>Controls (n = 47)</th>
<th>A (n = 42)</th>
<th>B (n = 74)</th>
<th>C (n = 25)</th>
</tr>
</thead>
<tbody>
<tr>
<td>α-Amylases (u/L), ( \bar{x} \pm SD )</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tu ( \bar{x} \pm 1 SD )</td>
<td>220 ± 296</td>
<td>102 ± 82***</td>
<td>115 ± 91**</td>
<td>186 ± 162</td>
</tr>
<tr>
<td>Range</td>
<td>24–1206</td>
<td>19–382</td>
<td>157–1508</td>
<td>22–589</td>
</tr>
<tr>
<td>Pu ( \bar{x} \pm 1 SD )</td>
<td>132 ± 186</td>
<td>61 ± 72*</td>
<td>59 ± 60**</td>
<td>134 ± 147</td>
</tr>
<tr>
<td>Range</td>
<td>26–992</td>
<td>9–241</td>
<td>137–1394</td>
<td>5–552</td>
</tr>
<tr>
<td>Su ( \bar{x} \pm 1 SD )</td>
<td>92 ± 152</td>
<td>38 ± 28**</td>
<td>51 ± 56*</td>
<td>53 ± 37</td>
</tr>
<tr>
<td>Range</td>
<td>19–804</td>
<td>0–115</td>
<td>0–432</td>
<td>9–134</td>
</tr>
</tbody>
</table>

*\( p < 0.05 \), **\( p < 0.01 \), ***\( p < 0.005 \) between the groups A, B, C and the normal controls.

A statistically significant correlation was found between serum Ta, Pa, and Sa levels and serum creatinine \( r = 0.65 \) \( (p < 0.003) \), \( r = 0.57 \) \( (p < 0.01) \), and \( r = 0.54 \) \( (p < 0.02) \), respectively) in the RTR group. However, no statistically significant correlation was found between urine amylases and serum creatinine in this group. Moreover, no correlation between urine and serum levels in any amylase was observed.

DISCUSSION

Our study showed that CRF patients with or without renal replacement treatment have increased values of total amylase as well as of isoamylases, thus confirming the results of previous studies (1, 8–10). It has been suggested that if the Ta and, especially, Pa values exceed three times the upper normal limits, then acute pancreatitis can be inferred (3). However, we found that a significant number of our patients had serum amylases more than three times the upper normal limits without exhibiting any clinical sign of acute pancreatitis.

Our RTR have increased serum levels of Ta and Pa, which were intermediate between controls and patients with renal failure (with or without replacement of renal function). The significant correlation between serum creatinine and serum isoamylases observed suggests that renal dysfunction plays a significant role in the pathogenesis of increased serum amylases in this group of patients. However, the absence of correlation between urine amylases and serum creatinine, as well as the absence of correlation between serum and urine values, suggests that the increased amylase levels could not be due to decreased excretion only. A disturbed amylase catabolism or reabsorption could play a significant role in the increased serum amylases. Moreover, the serum activity of Sa is not increased in RTR. It is known that pancreatic-type amylase is cleared by the kidney faster than salivary-type isoamylase (11). Therefore, the serum activity of salivary-type isoamylase might be less sensitive to a decrease of glomerular filtration. This possibility is strengthened by the
finding that salivary-type amylosemia was not correlated to serum creatinine or creatinine clearance.

However, a subclinical drug-induced pancreatitis could not be excluded, as it has been reported that corticosteroids, azathioprine, and cyclosporine cause hyperamylasemia and pancreatitis (6, 7, 12–15). However, none of our patients had any symptoms/signs suggestive of acute pancreatitis. Interestingly, none of the RTR had levels of amylases three times the upper normal limits, in contrast with the other groups of uremic patients.

In conclusion, serum amylase levels are increased in RTR. However, no patient in this group has amylase and isoamylase values more than three times the upper normal limits, unlike the other groups of uremic patients (with or without replacement of renal function).

REFERENCES