The Beginning of Repeated Hemodialysis Treatment Enhances Some Platelet Functions in Uremic Patients

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Dear Sir,

The thrombotic complications have become the predominant causes of mortality in uremic patients. An essential role in pathogenesis of these complications is played by platelet dysfunction. However, the disturbances of platelet function in uremic patients vary in dialyzed and nondialyzed patients. There is considerable disagreement as to what extent dialysis can reverse these abnormalities. Most investigations on this topic concerned the comparison between these two groups of patients simultaneously, but the patients in the groups were different [1-3]. We studied 10 uremic patients twice: first, in the period of conservative treatment (very-low-protein, low-phosphorus diet) and later on maintenance hemodialysis treatment (thrice weekly hemodialysis program, each for 4 h). We examined 8 males and 2 females, (aged 21-57 years, mean ± SD: 36.6 ± 17.1). Ten healthy blood donors, matched for age and sex were studied as controls. Medication known to affect platelet function as well as erythropoietin was excluded. Blood samples were collected by venipuncture in hemodialysis patients before the dialysis procedure. We studied the platelet count according to the Miesher and Gerarde method [4], bleeding time with the method of Mielke et al. [5], platelet aggregation according to Breddin [6] and, in addition, with the method of Born [7], using ADP as aggregating agent, platelet factor 3 (PF3) availability according to Saleem et al. [8] and platelet factor 4 (PF4) activity according to the method of O’Brien et al. [9]. Statistical analysis was performed using the Student t test.

Table 1. Selected parameters of platelet hemostasis in uremic patients treated conservatively, in the same patients after 3 months of repeated hemodialysis treatment, and in control group (means ± standard deviations)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Conservative Treatment</th>
<th>Hemodialysis</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Platelet Count (x10^9/L)</td>
<td>210±35</td>
<td>250±40</td>
<td>220±30</td>
</tr>
<tr>
<td>Bleeding Time (min)</td>
<td>2.5±0.5</td>
<td>1.5±0.3</td>
<td>1.0±0.2</td>
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<tr>
<td>Platelet Aggregation</td>
<td>0.50±0.15</td>
<td>0.80±0.20</td>
<td>0.60±0.15</td>
</tr>
<tr>
<td>Platelet Factor 3 Availability</td>
<td>0.05±0.01</td>
<td>0.10±0.02</td>
<td>0.03±0.01</td>
</tr>
<tr>
<td>Platelet Factor 4 Activity</td>
<td>2.80±0.5</td>
<td>4.00±0.8</td>
<td>2.00±0.4</td>
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</tbody>
</table>

PF4 activity has been variously assessed, probably according to different measurement procedures [1, 11, 12]. In this study, patients on maintenance hemodialysis presented a higher PF4 activity, compared to the same patients before hemodialysis treatment, which confirms platelet activation. Interestingly, in hemodialysis patients the disturbances of the platelet function appeared before the dialysis procedure; thus it is possible that platelet activation persists in the interdialytic period.
The results obtained are presented in Table 1. The prolonged bleeding time and decreased PF3 availability observed in conservatively treated patients suggest a decreased platelet function, as documented by others [2, 10]. After the beginning of maintenance hemodialysis treatment some platelet functions became significantly enhanced, which suggests platelet activation. This activation, mainly enhanced platelet aggregation, has been previously observed by other investigators [1-3]; increased PF3 availability has been documented in one of our recent stud-

References