Influence on Recombinant Human Erythropoietin on Hemorheological Profiles of Hemodialyzed Patients

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Table 1. Influence of rHuEPO on hemorheology

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normal Controls</th>
<th>rHuEPO Treated</th>
<th>rHuEPO Nontreated</th>
</tr>
</thead>
<tbody>
<tr>
<td>RCTT (ms)</td>
<td>200</td>
<td>180</td>
<td>210</td>
</tr>
<tr>
<td>MEA (Myrenne)</td>
<td>0.5</td>
<td>0.4</td>
<td>0.6</td>
</tr>
</tbody>
</table>

Dear Sir,

We have read the paper by Shand et al. [1], published in Nephron and would like to report our own results in a closely related field [2]. We have studied the hemorheological profiles of two groups of hemodialyzed patients in order to investigate whether a hemorheological change could be implicated in some adverse effects of recombinant human erythropoietin (rHuEPO).

Thirty-two hemodialyzed patients were studied. Twenty-one of them were given rHuEPO therapy during 8 months (16 ± 8), the rest were not. A third group consisted of 12 normal age and sex-matched individuals. In every group, hematocrit, RBC filterability (as an indicator of RBC deformability), RBC aggregation and plasma viscosity were determined. Besides this, RBC 2,3-DPG, plasma fibrinogen and haptoglobin were analyzed, due to their influence on those hemorheological parameters. Table 1 shows the results obtained.

We have not found any striking differences between the two groups of hemodialyzed patients. RBC filterability is better in the rHuEPO-treated patients (lower red cell transit time, p < 0.05, but there were no significant differences compared with normal controls. RBC aggregation also decreases with rHuEPO therapy but it remains higher than in normal controls. Perhaps the most puzzling finding is the elevation of fibrinogen observed in both groups of patients. As an acute-phase protein, fibrinogen biosynthesis is regulated by glucocorticoids and cytokines, namely interleukin-6 (IL-6) which stimulates hepatic fibrinogen synthesis [3]. In fact we have observed an increase of IL-6 in hemodialyzed patients [unpubl. results]. This point requires further investigation. On the other hand, it is well known that fibrinogen enhances RBC aggregation. It is surprising that in the rHuEPO-treated group fibrinogen is higher and RBC aggregation lower than in the untreated group. RBC aggregation is due to the interaction of some plasma proteins with RBC surface. It is likely that
rHuEPO acts not only quantitatively, but also qualitatively on erythropoiesis, and thus modifies cell surface. This modification could be advantageous for the hemorheological behavior of the red cell.

References