Letter to the Editor

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Inefficacy of Vitamin E Supplementation on Anemia in Hemodialysis Patients

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Dear Sir,

We read with interest the recent report of Ono [1] in this journal on improvement of anemia in 12 of 15 hemodialyzed patients after vitamin E (α-tocopherol) administration on a dosage of 600 mg/day for 30 consecutive days. In the same way, Giardini et al. [2] reported a statistically significant increase of the packed red blood cells (RBC) in 19 uremic patients in chronic hemodialysis after supplementation of tocopherol by intramuscular route on a dosage of 300 mg/day for 15 consecutive days. According to these authors, vitamin E blocks the peroxidation of the polyunsaturated fatty acid constituents of RBC; increased plasma levels of this vitamin provide an excess of α-tocopherol in RBC membrane which may reduce the susceptibility of the cells to osmotic hemolysis.

Methods

10 patients (6 males and 4 females), aged from 20 to 44 years (mean 37.2 ± 4.4), were studied. They had all been dialyzed from 21 to 208 months (mean 89.2 ± 57.4) in 3 × 4–4.5 h weekly hemodialysis sessions using hollow-fiber cuprophan dialyzers. All patients were stable and none had a history of recent acute disease. They were on a normal diet and 3 of them were moderate smokers less than 10 cigarettes/day. Routine medication consisted of folinic acid, CaCO₃, lα-OHDL and antihypertensive agents. None of the patients were receiving oral iron, vitamin A or E, or phosphatebinding agents and none were transfused during the study. Patients received oral supplementation of 600 mg vitamin E daily for 30 consecutive days. Hemoglobin, hematocrit, RBC and reticulocytes were computed using standard methods. Blood samples were taken from the arterial line immediately before dialysis on three occasions, namely 30 days before vitamin E supplementation was started (day -30), just before the study began and then 30 days later (day 0).

Table I. Hematological changes before and after vitamin E (α-tocopherol) supplementation in 10 hemodialyzed patients.

<table>
<thead>
<tr>
<th>Day</th>
<th>30</th>
<th>0</th>
<th>p</th>
<th>30</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin, g/dl</td>
<td>6.63 ± 0.98</td>
<td>6.80 ± 0.60</td>
<td>NS</td>
<td>6.00 ± 0.45</td>
<td>NS</td>
</tr>
<tr>
<td>Hematocrit, %</td>
<td>19.33 ± 2.43</td>
<td>19.50 ± 2.57</td>
<td>NS</td>
<td>18.58 ± 1.10</td>
<td>NS</td>
</tr>
<tr>
<td>RBC, ×10⁶</td>
<td>2.13 ± 0.26</td>
<td>2.28 ± 0.42</td>
<td>NS</td>
<td>2.10 ± 0.22</td>
<td>NS</td>
</tr>
<tr>
<td>Reticulocytes, ‰</td>
<td>17.00 ± 7.10</td>
<td>20.00 ± 5.90</td>
<td>NS</td>
<td>17.70 ± 5.80</td>
<td>NS</td>
</tr>
</tbody>
</table>
treatment (day 0) and 30 days later. Statistical analysis of results was made using the paired t test.

Results and Discussion

The results are shown in table I. No difference was observed in mean values of hemoglobin, hematocrit, RBC or reticulocytes between day -30 and day 0; nevertheless, a moderate decrease of these rates was found between day 0 and day 30 after oral supplementation of vitamin E. These changes are not statistically significant.

Our results confirm the assumption of Heldenberg et al. [3] who found no changes in the fatty acid composition of the erythrocyte membrane phospholipids and normal levels of vitamin E in patients treated by hemodialysis compared to a control group. Since the serum level of vitamin E reflects its concentration within the erythrocyte membrane [4], it is reasonable to assume that an excess of plasmatic levels of tocopherol obtained after oral supplementation do not necessarily increase the antioxidant effect of this vitamin within the erythrocyte membrane and their ability to withstand osmotic stress in this group of patients.

References


Announcement

International Symposium on IgA Nephropathy
Bari, Italy, April 10–11, 1987

The International Symposium on IgA Nephropathy will be held under the auspices of the University of Bari and the Case Western Reserve University of Cleveland, Ohio. Lectures, round tables and poster session will provide the occasion for an exchange of information between experts on recent developments in the pathogenesis of IgA nephropathy, long-term follow-up studies held in different countries and therapeutic approaches aimed at retarding the progression of renal damage. For information, write to Prof. F. Paolo Schena, Chair of Medical Therapy, University of Bari, Polyclinic, 70124 Bari, Italy. Phone: 39–80–27237.

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