Blood vitamin status in chronic alcoholics after a single dose of polyvitamin. A preliminary report

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**Summary**

Blood vitamin status (B<sub>1</sub>, B<sub>2</sub> and B<sub>6</sub>) was assessed by erythrocytic enzyme activation tests in 7 male and 1 female chronic alcoholics (mean age (+s.d.), 43·12 (13·7) years; range, 25–64 years) on admission and 6 hr later on the same day after oral administration of a single dose of polyvitamin. Seven out of 8 patients were found to be deficient in vitamin B<sub>1</sub>, 5 in B<sub>2</sub> and none in B<sub>6</sub>. But after single-dose oral therapy, tendency to improvement in blood vitamin status was clearly evident in all patients. It is therefore suggested that a subgroup of chronic alcoholics may benefit from oral supplementation of vitamins. Clinical implications of vitamin deficiency in chronic alcoholics are briefly discussed.

**Introduction**

Multiple vitamin deficiency due to poor dietary intake, impaired absorption, inability metabolically to transform the inactive vitamin (in diet or drugs) to its biologically active metabolite as a result of hepatic hypo-function or impaired tissue storage in chronic alcoholics is well documented (Thomson, 1978; Thomson, Rae and Majumdar, 1980b). Vitamin deficiency may limit repair of ethanol-induced physical damage. It is, therefore, important to ensure that current therapeutic measures effectively replete the deficiencies. Deficiencies are normalized quite well after i.v. polyvitamin therapy (Majumdar, Shaw and Thomson, 1981). The blood vitamin status (B<sub>1</sub>, B<sub>2</sub>, B<sub>6</sub>) was tested in randomly selected chronic alcoholics both before and after a single oral dose of one tablet of polyvitamin in order to make a preliminary assessment of their utilizing capacity of the administered vitamins.

**Patients**

Eight patients (7 male, 1 female; mean age (+s.d.), 43·12 (13·7) years; range 25–64 years) were randomly selected on admission for the study. Their consumption of ethanol was equivalent to more than 80 g/day of absolute ethanol for more than 5 years. They had a few stigmata of hepatic damage (i.e. liver palm, spider naevi, clubbing and hepatomegaly in some patients). There were no clinical features suggesting intestinal malabsorption, diarrhoea or vomiting.

**Treatment**

All patients were given one tablet of Orovit (Bencard) which contains 50 mg thiamine hydrochloride, 5 mg riboflavin, 5 mg pyridoxine hydrochloride, 200 mg nicotinamide and 100 mg ascorbic acid.

**Methods**

Samples of blood were of 4 ml in special vials (containing acid citrate and dextrose as preservative to prevent haemolysis) from each patient on admission before starting treatment and also 6 hr later on the same day. The following erythrocyte enzyme activation tests were used according to methods of Heller, Salkeld and Korner (1974a, b). Erythrocyte transketolase (ETK) for vitamin B<sub>1</sub>; glutathione reductase (EGR) for B<sub>2</sub>; Pyridoxal-5-phosphate (PALP) for vitamin B<sub>6</sub> was directly estimated in red blood cells by methods of Lumeng and Li (1974).

The activation co-efficient is represented by α<sub>ETK</sub>. ETK is a measure of vitamin B<sub>1</sub> status and is the ratio of increased erythrocyte transketolase activity (with thiamine pyrophosphate (TPP)) to the original activity (without TPP). ETK<sub>α</sub> represents the amount of the apoenzyme fraction of the enzyme transketolase without the addition of TPP (normal, >70 μmol/l). Similarly, αEGR is a parameter for riboflavin status and is the ratio of increased
Vitamin deficiency in chronic alcoholics

Table 1. Vitamin utilization status in chronic alcoholics after a single oral dose of Ovorite (one tablet) – 6 hr later

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Age</th>
<th>Sex</th>
<th>Vitamin B₁</th>
<th>Vitamin B₂</th>
<th>Vitamin B₆</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>ETK₀</td>
<td>αETK</td>
<td>αEAR</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Before</td>
<td>After</td>
<td>Before</td>
</tr>
<tr>
<td>1</td>
<td>53</td>
<td>M</td>
<td>78-6</td>
<td>79-5</td>
<td>1-02</td>
</tr>
<tr>
<td>2</td>
<td>25</td>
<td>M</td>
<td>66-5</td>
<td>67-9</td>
<td>1-05</td>
</tr>
<tr>
<td>3</td>
<td>34</td>
<td>F</td>
<td>63-4</td>
<td>86-3</td>
<td>1-12</td>
</tr>
<tr>
<td>4</td>
<td>43</td>
<td>M</td>
<td>47-3</td>
<td>53-6</td>
<td>1-21</td>
</tr>
<tr>
<td>5</td>
<td>55</td>
<td>M</td>
<td>62-4</td>
<td>62-7</td>
<td>1-16</td>
</tr>
<tr>
<td>6</td>
<td>64</td>
<td>M</td>
<td>42-8</td>
<td>50-4</td>
<td>1-16</td>
</tr>
<tr>
<td>7</td>
<td>28</td>
<td>M</td>
<td>55-5</td>
<td>63-0</td>
<td>1-16</td>
</tr>
<tr>
<td>8</td>
<td>43</td>
<td>M</td>
<td>34-2</td>
<td>40-6</td>
<td>1-34</td>
</tr>
<tr>
<td>Mean ± s.d.</td>
<td></td>
<td></td>
<td>43-12</td>
<td>63-0</td>
<td>1-14</td>
</tr>
<tr>
<td>Normal values</td>
<td>&gt;70 μmol/l</td>
<td>&lt;1-15</td>
<td>&lt;1-19</td>
<td>&gt;5 (ng/ml)</td>
<td></td>
</tr>
</tbody>
</table>

ETK₀, erythrocyte transketolase; EGR, glutathione reductase; PALP, pyridoxal-5-phosphate.

erthrocyte glutathione reductase activity (with flavin adenine dinucleotide (FAD)) to the basal activity (without FAD). Pyridoxine is inactive as such and is converted in the body to its biologically active metabolite – pyridoxal-5-phosphate (PALP) with the help of adenosine triphosphate (ATP) and PALP is measured directly in the erythrocytes.

Results

Results (± s.d.) are given in Table 1.

Vitamin B₁

Seven out of 8 were found to be deficient in ETK₀ (mean (± s.d.), 56-33 (14-34)), it improved in all patients but normalized only in one and the mean still remained below normal (mean (± s.d.), 63-0 (15-06)). αETK was lower than normal in 4 out of 8 but returned to normal in all cases except one (patient no. 8).

Vitamin B₂

αEGR was within normal limits on admission and it improved after therapy.

Vitamin B₆

PALP was lower than normal in 5 out of 8 patients and it was considerably improved after treatment.

Discussion

Post-treatment blood vitamin status improved in all patients and a tendency to further improvement on continued oral treatment was clearly evident in all of them.

Vitamin deficiency (B₁, B₂ and B₆) in chronic alcoholics may be partially attributed to low dietary intake, impaired absorption, deficient metabolic transformation to their active metabolites due to hepatic hypofunction and impaired tissue storage (Thomson, 1978; Thomson et al., 1980b). All these vitamins are converted in the body, especially in the liver, and erythrocytes are converted to their biologically active metabolites with the help of ATP which then act as essential co-enzymes in vital functions, i.e. thiamine to TPP, pyridoxine to PALP, and FAD, which is the main co-enzyme form of riboflavin.

The biologically active co-enzyme form of vitamin B₆ (PALP) in erythrocytes and plasma has been shown to reflect the nutritional status (Rossouw et al., 1977). αETK and αEGR also reflect the vitamin status for thiamine and riboflavin respectively (Heller et al., 1974a, b) and hence the nutritional state as well. The present observations indicate the need for relevant vitamin supplementation in chronic alcoholics and also supports the inclusion of vitamins in a conventional detoxification regime for chronic alcoholics.

Vitamin deficiency frequently accompanies alcoholism in the U.K. (Thomson, 1978; Thomson et al., 1980b) and also in the U.S.A. (Leevy and Baker, 1970; Levey, Thomson and Baker, 1970) and may limit repair of physical damage induced by chronic ethanol ingestion.

The provision of nutrients which can be utilized at the subcellular level may therefore limit the degree of permanent brain damage and enhance tissue repair (Thomson et al., 1980a). The findings reported in this paper suggest that polyvitamin therapy may be of value but the response to sustained- as opposed to single-dose administration has yet to be evaluated.
Acknowledgment

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References


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