EFFECT OF PYRIDOXINE ON PLASMA LEVELS OF HGH, PRL, AND TSH IN NORMAL WOMEN

By

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ABSTRACT

The effect of chronic administration of pyridoxine (vitamin B6) on HGH, basal and TRH stimulated PRL and TSH levels was studied in seven healthy female volunteers. HGH and TSH basal levels remained unaffected. Basal PRL and TRH stimulated PRL and TSH levels were slightly reduced without statistical significance ($P > 0.05$). The results indicate, that stimulation of the hypothalamic dopaminergic pathway by pyridoxine is highly variable and unpredictable. Therefore it seems meaningless to recommend vitamin B6 for the treatment of hyperprolactinaemia.

Reports on the effect of pyridoxine (vitamin B6) on PRL secretion are still controversial. Foukas (1973) claimed pyridoxine to be effective in suppressing puerperal lactation. Subsequent studies of McIntosh (1976) and Delitala et al. (1976) also indicated PRL inhibiting properties of pyridoxine in the galactorrhoea-amennorhoea syndrome and in normal volunteers. However, these findings could not be confirmed by other investigators (del Pozo et al. 1975; McDonald et al. 1976; Tolis et al. 1977; Spiegel et al. 1978).

The present study was designed to evaluate a possible dopaminergic influence of pyridoxine on the functional reserve of the pituitary. Plasma levels of PRL, TSH and HGH were followed in healthy females after TRH stimulation with and without pyridoxine treatment.

MATERIAL AND METHODS

Seven normally cycling women 19–31 years of age volunteered for the study. All volunteers were fully informed and gave their written consent. The participants received daily 600 mg pyridoxine orally from day 26 of the cycle to day 5 of the
following cycle. Blood was drawn before and on the last day of treatment. After one hour of resting, blood was obtained for HGH determination via an indwelling catheter. Subsequently 0.2 mg TRH was injected. Blood samples were taken before and after the injection at stated intervals (−20, 0, 10, 20, 30, 45, 60 and 90 min) for the determination of TSH and PRL. The maximal responses were compared with the basal levels.

HGH and TSH were measured by radioimmunoassay kits purchased from CIS-Isotopen Dienst-West, Dreieich/Frankfurt, F.R.G. Accuracy and specificity fulfilled the generally accepted criteria. HGH standard: I.IRP (MRC). Sensitivity: 0.02 ng. Precision: within-assay 8.8 %, between-assay 12.8 %. Normal range: 0–6.5 ng/ml. TSH standard: MRC 68/38. Sensitivity: 0.1 ng. Precision: within-assay 7.5 %, between-assay 14 %. Normal range: 2–9 µg/ml. PRL was determined by radioimmunoassay employing the NIAMDD kit (V.L.S.3). Standard: MRC 71/222. Sensitivity: 0.5 ng. Precision: within-assay 5.2 %, between-assay 11.1 %. Accuracy and specificity satisfied the generally accepted validity criteria. Normal range: below 25 ng/ml. Mean values and standard deviation (sd) were calculated. Statistical significance of the differences was assessed by Student’s t-test for paired data.

R E S U L T S

Basal levels of HGH before treatment averaged 1.25 ± 0.05 ng/ml and during pyridoxine administration 1.57 ± 0.54 ng/ml (P > 0.05) as shown in Fig. 1.

Maximal levels of PRL and TSH were recorded 10–30 min after the injection of 0.2 mg TRH and are also presented in Fig. 1.

![Fig. 1.](image-url)

Mean values and standard deviations of basal HGH, basal and TRH stimulated PRL and TSH levels in plasma prior to and during pyridoxine treatment in 7 normally cycling healthy female volunteers.

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Individual basal and maximal TRH stimulated PRL and TSH levels in plasma prior to and after the administration of 600 mg pyridoxine daily for 8 days in 7 normally cycling healthy female volunteers.

<table>
<thead>
<tr>
<th>volunteers</th>
<th>PRL (ng/ml)</th>
<th>TSH (μU/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>basal</td>
<td>TRH-stimulated</td>
</tr>
<tr>
<td>M.M</td>
<td>18</td>
<td>71</td>
</tr>
<tr>
<td>C.B</td>
<td>2.5</td>
<td>37</td>
</tr>
<tr>
<td>S.U.</td>
<td>15</td>
<td>62</td>
</tr>
<tr>
<td>U.F.</td>
<td>8</td>
<td>51</td>
</tr>
<tr>
<td>U.W.</td>
<td>10</td>
<td>89</td>
</tr>
<tr>
<td>A.D.</td>
<td>21</td>
<td>96</td>
</tr>
<tr>
<td>K.W.</td>
<td>13</td>
<td>69</td>
</tr>
</tbody>
</table>

The slight reduction of the basal and stimulated PRL levels is statistically insignificant ($P > 0.05$).

TSH secretion in response to TRH prior to and during pyridoxine treatment remained unaffected ($P > 0.05$). The individual data obtained are listed in Table 1, indicating a slight reduction under B6-treatment especially in basal PRL and TRH stimulated TSH levels. Because of the wide variation in the individual values and consequently the large standard deviations no statistical significance could be calculated.

**DISCUSSION**

Pyridoxine is a precursor of pyridoxal-5-phosphate, serving as a coenzyme for decarboxylation and transamination of amino acids. In particular pyridoxine is required for the decarboxylation of both L-dopa to dopamine and 5-hydroxytryptophan to serotonin.

Recent findings have demonstrated an inhibiting effect of pyridoxine on PRL secretion and a stimulating effect on HGH secretion suggesting an increased dopamine turnover in the hypothalamus, as claimed by McIntosh (1976) and by Delitala et al. (1976). These findings could not be reproduced by Tolis et al. (1977) and Lehtovirta et al. (1978). The presented data indicate that treatment with 600 mg pyridoxine/day results in an insignificant decrease of basal as well as of stimulated PRL levels. Stimulated TSH levels are
slightly reduced. Because of considerable individual variability of the hormone levels the reduction is insignificant. Delitala et al. (1976) noted a PRL-decrease and an HGH-increase to a single pyridoxine injection of 300 mg in normals, however Tolis et al. (1977) reported variable results after the administration of pyridoxine. Similar findings were obtained in the galactorrhoea-amenorrhoea syndrome. McIntosh (1976) treated three women successfully with 600 mg pyridoxine per day, and elevated PRL values fell to normal. In comparison, Lehtovirta et al. (1978) used pyridoxine in 22 patients with galactorrhoea-amenorrhoea syndrome only with partial success. Normalization of elevated PRL levels was observed only in 6 patients.

The inconsistent results under chronic administration of pyridoxine are possibly explained by the short duration of the dopamine action. The acute bolus of L-dopa results in a suppression of PRL levels lasting for only a few hours followed by an increase (Frantz et al. 1973). Chronic L-dopa administration does not result in consistently lowered PRL levels (Frantz et al. 1973) nor in consistently elevated HGH levels as demonstrated by von Werder et al. (1970).

Based on the results on the present paper it is concluded that the stimulation of the dopaminergic pathway by pyridoxine should be regarded as an inadequate tool for the management of hyperprolactinaemia.

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REFERENCE


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