Effect of pyridoxine or riboflavin supplementation on plasma homocysteine levels in women with oral lesions

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ABSTRACT

Background. A moderate increase in plasma homocysteine level has been reported to be involved in neural tube defects, which can be prevented with folic acid supplementation. Folic acid, vitamins B6 and B12-dependent enzymes are required to metabolize homocysteine. A study in rats showed higher tissue homocysteine levels in riboflavin as well as pyridoxine deficiency. We studied the effect of treatment with pyridoxine or riboflavin on plasma total homocysteine concentration in women with clinical and biochemical deficiencies of riboflavin and pyridoxine.

Methods. Plasma total homocysteine concentrations were measured in 20 women with glossitis and angular stomatitis before and after supplementation with pyridoxine or riboflavin.

Results. Pyridoxine treatment significantly reduced plasma homocysteine concentration while riboflavin treatment did not have a significant effect.

Conclusions. Plasma total homocysteine levels tended to be higher in women with clinical and biochemical deficiency of vitamin B6 and therapy with pyridoxine reduced its level significantly. Riboflavin supplementation did not have a significant impact on plasma homocysteine concentration in women with glossitis and angular stomatitis.


INTRODUCTION

Raised plasma homocysteine concentration has been implicated in the aetiology of neural tube defects. Folic acid, pyridoxal phosphate and vitamin B12-dependent enzymes are involved in the metabolism of homocysteine. An earlier study in rats has documented higher tissue homocysteine levels in riboflavin as well as vitamin B6 deficiency. The incidence of biochemical and clinical deficiencies of vitamin B6 and riboflavin is high in India, particularly in women belonging to the low-income group. Hence we studied the effect of pyridoxine and riboflavin supplementation on plasma total homocysteine concentrations in women with clinical and biochemical deficiencies of vitamins B6 and B12.

SUBJECTS AND METHODS

Twenty women in the age range of 20–45 years with mucocutaneous lesions such as glossitis and angular stomatitis were included in the study. The mean (SE) body mass index of the subjects was 19.8 (0.82). Fasting blood samples were obtained before and after treatment with either 20 mg pyridoxine hydrochloride or 10 mg riboflavin for a period of 15 days. The blood samples were chilled on ice and transported to the laboratory, where plasma and cells were separated within 3 hours of obtaining the samples.

Plasma homocysteine was derivatized with ammonium 7-fluro-2-oxa-1,3 diazole-4-sulfonate (SBD-F) and the SBD-derivative of homocysteine was determined by high performance liquid chromatography. This method measures total (free and protein bound) plasma homocysteine concentration.

The riboflavin and vitamin B6 status of women was assessed by enzymatic tests: erythrocyte glutathione reductase activation coefficient (EGR-AC) and erythrocyte aspartate amino transferase activation coefficient (EAAT-AC) respectively. The data were analysed by paired ‘t’ test.

RESULTS

The biochemical deficiencies of vitamin B6 and riboflavin were established by higher EAAT-AC (> 1.8) and higher EGR-AC (> 0.4) values, respectively (Table I). The subjects responded to treatment with these two vitamins clinically and also biochemically, as judged by the above parameters.

The mean plasma total homocysteine concentration tended to be higher in the vitamin B6-deficient group and supplementation with pyridoxine significantly reduced its level. However, riboflavin supplementation did not have a significant effect on plasma homocysteine levels (Table I).

DISCUSSION

All co-factors required for homocysteine metabolism may be important determinants of circulating homocysteine concentrations.

In humans, deficiencies of folate and vitamin B12 are known to increase plasma homocysteine levels. Vitamin B6 deficiency also appears to be associated with a higher plasma homocysteine concentration. In the rat study, deficiencies of both vitamins B6 and B12 increased skin homocysteine levels. This difference in the effect of riboflavin deficiency could be due to a variation in the severity of deficiency, since rats received a diet completely devoid of riboflavin. Elevated levels of homocysteine were also reported in the plasma of pyridoxine-deficient pig and rat.

Apart from being considered as a risk factor for premature cardiovascular disease, elevated plasma homocysteine has been implicated in the aetiology of neural tube defects. Mothers with neural tube defect neonates had higher circulating concentrations of homocysteine compared with controls. Maternal folic acid supplementation during the periconceptional period was reported to reduce the risk of giving birth to a baby with neural tube defect. The protection conferred by folic acid supplementation...
may be explained by its homocysteine lowering effect. A reduction in plasma homocysteine levels was observed after therapy with pyridoxine in patients with clinical and biochemical deficiency of this vitamin.

In developing countries like India, deficiencies of folate and vitamin B_6 are common in women belonging to the low-income group. A combined supplementation of folic acid and pyridoxine may be more effective in lowering plasma homocysteine concentration in this population.

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REFERENCES