Selenium, Glutathione-peroxidase and Dermatitis herpetiformis

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In patients with dermatitis herpetiformis, decreased blood levels of glutathione-peroxidase (GSH-Px) were common (1). GSH-Px is an index of selenium content and used to detect functional selenium deficiency. We report here the results of a double-blind study where selenium+vitamin E or placebo was added to the dapsone treatment in patients with dermatitis herpetiformis.

PATIENTS

Twenty-four patients (18 men and 6 women, age 32-75 years) were selected for the study. They had all a clinically classical dermatitis herpetiformis and granular deposits of IgA in the papillary tips. During the last year all had needed a constant dose of dapsone (25-200 mg daily) to be free from skin symptoms. For 12-72 months four of them had also used a gluten-free diet without being able to reduce their dapsone intake. Six patients on a normal diet had just started on a gluten-free diet. They were therefore excluded from the study, since it would be difficult to evaluate the clinical effect of selenium supplementation. The patients should be treated double-blind for 5 months twice daily either with placebo or tablets containing 0.2 mg of selenium (as Na2SeO3)+ 10 mg tocopheryl succinate.

RESULTS

When the study began there was unfortunately a report in press and on television about carcinogenic effects of selenium. Three patients who had read or heard this report stopped
the treatment after 2–6 weeks, because they felt worse and had been forced to increase their dapsone intake. They had all received placebo. Out of 15 patients, who completed the study, 10 had received selenium and 5 placebo. No changes in the need for dapsone were noted in either group. No side effects after selenium treatment was noted. The GSH-Px level increased in all patients treated with selenium. The mean ± SE was 268±65 µkat/l before and 406±79 µkat/l after 5 months treatment, which is a significant increase (p<0.001). In the placebo group no significant change was found. The GSH-Px value was correlated to the selenium content in the erythrocytes (Fig. 1). The plasma levels of selenium were 20–30% lower than in the red cells.

DISCUSSION

Since treatment with selenium and vitamin E increased the blood level of GSH-Px without really improving the patients’ clinical condition as evaluated from their need for dapsone, the GSH-Px level does not seem to be related to the skin symptoms of the disease itself. The possibility of dapsone influencing the GSH-Px level seems less likely, since there was no correlation with the dose used. It therefore seems most probable that the low GSH-Px and selenium levels in blood from patients with dermatitis herpetiformis are due to malabsorption of selenium. Supplementary selenium and vitamin E treatment might therefore still be indicated in such patients for reasons which have not become evident from our study.

REFERENCES