

STUDY DECREASES OF THYROID PEROXIDASE ANTIBODIES CONCENTRATIONS IN PATIENTS WITH AUTOIMMUNE THYROIDITIS

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In areas with severe selenium deficiency there is a higher incidence of thyroiditis due to a decreased activity of selenium-dependent glutathione peroxidase activity within thyroid cells. Selenium-dependent enzymes also have several modifying effects on the immune system. Therefore, even mild selenium deficiency may contribute to the development and maintenance of autoimmune thyroid diseases.

Purpose. Study decreases thyroid peroxidase antibodies concentrations.

Materials and methods. We performed a blinded, placebo-controlled, prospective study in female patients ($n = 70$; mean age, 47.5 ± 0.7 yr) with autoimmune thyroiditis and thyroid peroxidase antibodies (TPOAb) and/or Tg antibodies (TgAb) above 350 IU/ml. The primary end point of the study was the change in TPOAb concentrations. Secondary end points were changes in TgAb, TSH, and free thyroid hormone levels as well as ultrasound pattern of the thyroid and quality of life estimation. Patients were randomized into 2 age- and antibody (TPOAb)-matched groups; 36 patients received 200 μg (2.53 μmol) sodium selenite/d, orally, for 3 months, and 34 patients received placebo. All patients were substituted with L-T₄ to maintain TSH within the normal range. TPOAb, TgAb, TSH, and free thyroid hormones were determined by commercial assays. The echogenicity of the thyroid was monitored with high resolution ultrasound. The mean TPOAb concentration decreased significantly to 63.6 % ($P = 0.013$) in the selenium group vs. 88 % ($P = 0.95$) in the placebo group. A subgroup analysis of those patients with TPOAb greater than 1200 IU/ml revealed a mean 40% reduction in the selenium-treated patients compared with a 10% increase in TPOAb in the placebo group.

The results obtained. TgAb concentrations were lower in the placebo group at the beginning of the study and significantly further decreased ($P = 0.018$), but were unchanged in the selenium group. Nine patients in the selenium-treated group had completely normalized antibody concentrations, in contrast to two patients in the placebo group (by χ^2 test, $P = 0.01$). Ultrasound of the thyroid showed normalized echogenicity in these patients. The mean TSH, free T₄, and free T₃ levels were unchanged in both groups.

Conclusions of the findings. We conclude that selenium substitution may improve the inflammatory activity in patients with autoimmune thyroiditis, especially in those with high activity. Whether this effect is specific for autoimmune thyroiditis or may also be effective in other endocrine autoimmune diseases has yet to be investigated.