## **Brief Communication**

# Selenium and the thyroid: A close-knit connection

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## ABSTRACT

**Introduction:** In areas with severe selenium deficiency higher incidence of thyroiditis has been reported due to a decreased activity of selenium-dependent glutathione peroxidase enzyme within thyroid cells. **Aims and Objective:** To study the effect of selenium supplementation in patients with autoimmune thyroid disease. **Materials and Methods:** This is a blinded placebo-controlled prospective study done in 60 patients with autoimmune thyroid disease (as defined by an anti-thyroid peroxidase antibody (TPOAb) level more than 150 IU/ml) irrespective of the baseline thyroid status. Patients with overt hyperthyroidism who are on antithyroid drugs, patients on any other medication, which may alter the immunity status of the patients, and pregnant patients were excluded from the study. Patients were randomized into two age and TPOAb-matched groups; 30 patients received 200 µg of sodium selenite/day, orally, for 3 months, and 30 patients received placebo. All hypothyroid patients were given I-thyroxine replacement. **Results:** Of 30 patients in the selenium treated group, 6 patients were overtly hypothyroid, 15 were subclinical hypothyroid, 6 were euthyroid, and 3 were subclinical hyperthyroid. The mean TPOAb concentration decreased significantly by 49.5% (*P* < 0.013) in the selenium treated group versus 10.1% (*P* < 0.95) in the placebo-treated group. **Conclusion:** Selenium substitution has a significant impact on inflammatory activity in thyroid-specific autoimmune disease. It would be of interest to determine whether early treatment with selenium in patients with newly developed autoimmune thyroiditis may delay or even prevent the natural course of these diseases.

Key words: Selenium, thyroid

### INTRODUCTION

Selenium, from the Greek word *Selene* (meaning moon), is a chemical element (atomic number 34) that was discovered as a by-product of sulfuric acid in 1817. In 1967, it was found that the thyroid gland had the maximum amount of selenium per gram of tissue.<sup>[1]</sup>

Autoimmune thyroiditis (AIT), the prototype of autoimmune diseases, is characterized by T-cell-mediated autoimmune destruction of thyroid cells. Environmental factors, such as iodide intake, immunotherapeutic agents, or viral infections that may initiate the disease.<sup>[2]</sup> In areas, where selenium deficiency is prevalent, higher incidence of

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thyroiditis has been reported due to a decreased activity of selenium-dependent glutathione peroxidase enzyme within thyroid cells. Severe nutritional selenium deficiency leads to an increased rate of thyroid cell necrosis and invasion of macrophages. Whether this also may induce a higher incidence of autoimmune thyroiditis is unknown. It may be assumed, however, that thyroid cell damage may initiate or maintain autoimmune thyroiditis, especially in patients susceptible to the development of autoimmune diseases.<sup>[3]</sup>

#### Aims and objective

To study the effect of selenium supplementation in patients with autoimmune thyroid disease.

## **MATERIALS AND METHODS**

This is a blinded placebo-controlled prospective study done in 60 patients.

#### **Inclusion criteria**

Patients of all age groups and both sexes with autoimmune thyroid disease (as defined by an anti- thyroid peroxidase

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antibody [TPOAb] level more than 150 IU/ml) irrespective of the baseline thyroid status.

#### **Exclusion criteria**

Patients with overt hyperthyroidism who are on antithyroid drugs, patients on any other medication, which may alter the immunity status of the patients, and pregnant patients were excluded from the study.

Patients were randomized into two age and TPOAb-matched groups; 30 patients received 200 µg sodium selenite/day, orally, for 3 months, and 30 patients received placebo. All hypothyroid patients were given l-thyroxine replacement.

TPOAb level was measured using chemiluminiscence. The differences in antibody concentrations at the beginning and end of the study were determined by *t*-test for paired samples. The *P* values were corrected for the numbers of tests performed.

### RESULTS

In the selenium treated group 27 patients were female, and three patients were male (M:F = 1:9), which was comparable with the ratio in the placebo-treated group (1:7.3) The mean ages at presentation in both the groups were  $34 \pm 2.5$  and  $31 \pm 3.4$  years, respectively. At study entry, the mean TPOAb concentrations were identical for both groups (selenium treated group,  $669 \pm 205$  IU/ml; placebo,  $729 \pm 277$  IU/ml). Out of the total 30 patients in the selenium treated group, 6 patients were overtly hypothyroid, 15 were subclinical hypothyroid, 6 were euthyroid, and 3 were subclinical hyperthyroid. There were comparable numbers of patients in each subgroup in the placebo-treated group also. The mean TPOAb concentration decreased significantly by 49.5% (P < 0.013) in the selenium treated group versus 10.1% (P < 0.95) in the placebo-treated group. In subgroup analysis, the decrease in the mean TPOAb titre was highest in the subclinical hyperthyroid group (up to 64.42%), and comparable in the other three groups (41.13%, 47.18%, and 42.64% in the euthyroid, hypothyroid, and subclinical hypothyroid groups respectively). One patient with hypothyroidism in the selenium treated group with a TPOAb concentration of >1000 IU/ml, had completely normalized antibody concentrations after 3 months. It was also found that those patients with TPOAb greater than 1000 IU/ml revealed a mean 31.38% reduction in the selenium-treated patients, compared with no significant change in TPOAb in the placebo group.

## DISCUSSION

Selenium substitution may improve the immunity status in patients with autoimmune thyroid disease. Seleniumdependent enzymes are both anti-oxidative and antiinflammatory. Glutathione peroxidase can reduce hydrogen peroxides and phospholipid hydroperoxides, and hence can reduce the production of free radicals and reactive oxygen species. Lower hydroperoxide tissue concentrations diminish the production of inflammatory prostaglandins, and leukotrienes. These mechanisms may contribute to reduced inflammatory activity in the organ-specific autoimmune response, and may explain the improvement of autoimmune thyroiditis in our study.

Based on the link described above between selenium and the thyroid, several studies applying organic and inorganic selenium compounds were undertaken in patients, with AIT in areas with low to borderline-low-selenium content. A prospective placebo-controlled clinical study with selenium in AIT conducted in the selenium deficient area of Bavaria in southern Germany, by Gärtner *et al.* in 2002<sup>[4]</sup> showed a 36% reduction in anti-TPO titers in the selenium-treated group, whereas a further reduction of up to 60% was seen in a subgroup of patients with basal anti-TPO levels above 1200 IU/ml.

## CONCLUSION

Supplementation of selenium has a significant impact on inflammatory activity in thyroid-specific autoimmune disease. It would be of interest to determine whether early treatment with selenium in patients with newly developed autoimmune thyroiditis may delay, or even prevent the natural course of these diseases.

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