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Review Article

Two unclear issues in Hashimoto's disease: gluten-free diet and selenium support

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Abstract

Hashimoto's disease is one of the most common endocrine disorders in society. Although the accepted standard treatment is hormone replacement, social media has offered nutritional support based on a gluten-free diet and selenium supplementation. This study aimed to clarify this issue by reviewing the literature for awareness of clinicians.

Keywords: Celiac disease, Hashimoto's disease, gluten, selenium.

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INTRODUCTION

Gluten

First of all, it should be well known that gluten-sensitive enteropathy and gluten sensitivity are not the same disorder. Gluten-sensitive enteropathy, or Celiac disease, is an autoimmune disease caused by gliadin intolerance. Gluten is a protein complex consisting of gliadin and glutenin, found in some foods such as wheat, rye, and barley. Gliadin reaches the intestine undigested, where it is broken down and digested by tissue transaminase. Antibodies are produced in Celiac disease against these tissue transaminases. The immune-mediated inflammatory response is damaged in the intestinal mucosa. As a result, symptoms including abdominal pain, bloating, constipation, diarrhea, weight loss, nausea, vomiting, and vitamin deficiency-related problems occur. A gluten-free diet is essential for these patients throughout their lives, and it is neither cheap nor sustainable. Although patients with gluten sensitivity have similar symptoms such as gas, bloating, headaches, and fatigue that reduce their quality of life, there is no inflammatory condition in the intestines. When they eat gluten, they can continue their lives without showing as severe symptoms as Celiac patients. Celiac and Hashimoto diseases, which are autoimmune diseases, can occur simultaneously. Even so, the same gene is responsible for both diseases. Abbott et al. found that implementing an autoimmunity diet, which excludes sugar, grains, dairy products, and additives, along with lifestyle modifications, did not result in any changes in thyroid stimulating hormone (TSH), free triiodothyronine (fT3), free thyroxine (fT4), anti-thyroid peroxidase antibody (Anti-TPO), and anti-thyroglobulin antibody (Anti-Tg) levels. In contrast, the quality of life was increased (1). Kaličanin et al. found no association between food sensitivity and Hashimoto autoimmunity, assessed by measuring specific IgG linked to 125 nutrients, including gluten, and markers such as anti-thyroid peroxidase antibody (Anti-TPO) and antithyroglobulin antibody (Anti-Tg) (2). Ostrowska et al. administered an elimination/reduction diet to Hashimoto patients for six months and compared them with other patients who took the same calories but did not use this diet (3). They found a decrease in TSH, Anti-TPO, and Anti-Tg levels and an increase in fT3 and fT4 levels in patients in the diet group. Krysiak et al. found a decrease in thyroid autoantibodies with a gluten-free diet for six months in female patients with Hashimoto's disease, which had high tissue transglutaminase antibodies (4). Kus et al. conducted a survey study to investigate the effects of a gluten-free diet on Hashimoto's patients (5). They reported that hypothyroidism symptoms, especially digestive problems, decreased in 75% of the patients. However, this study has serious scientific deficiencies. Firstly, it was conducted as an observational study. Secondly, the presence of celiac disease was not an exclusion criterionin their study. Thirdly, the patients did not receive any information on a gluten-free diet. Lastly, laboratory tests were not confirmed by the authors; they were only based on the statements of the patients. As a result, recommending a gluten-free diet for all Hashimoto's patients is not feasible due to the inconsistent findings in studies regarding its benefits on the clinical and laboratory values of hypothyroidism. Hashimoto's disease is common with gluten sensitivity, even if Celiac disease is not diagnosed. There are no specific tests to determine gluten sensitivity. The clinical response of patients may be observed with an elimination diet. In patients whose complaints do not improve despite hypothyroidism treatment, the decision to administer a gluten-free diet can be made individually, depending on whether the patients receive clinical benefit from this diet.

Selenium

Selenium is a name of Greek origin (selene), meaning moon. It has antioxidant and anti-inflammatory activity and is necessary for the healthy functioning of the immune system, reproduction, and thyroid functions. It is found mostly in the thyroid gland in our body and is included in the structure of selenoproteins that play a crucial role in thyroid functions. Selenoproteins are included in three enzyme families: glutathione peroxidase (GPX), thioredoxin reductase (TR), and deiodinases. GPX protects cells from the toxic effects of free oxygen radicals. TR also plays a role in antioxidant events. Deiodinases provide thyroid hormone activation and inactivation.

In brief, these enzymes protect thyroid cells from harmful effects from radicals and play an important

role in thyroid hormone production. Selenium intake depends on the geographical region of residence. If selenium is low in soil and water, there will be a deficiency in plants and animals living there. The main sources of selenium are red meat, chicken, fish, rice, oats, beans, eggs (especially yolks), onions, broccoli, Brazil nuts, and garlic. The average selenium requirement is 55–75 mcg/day. It is increased to 70–80 mcg/day during pregnancy and lactation. The daily intake should not exceed 400 mcg. Normal serum selenium level is between 70 and 85 µg/l. However, serum selenium measurements may not be sufficient to indicate body levels. Measurement of intra-erythrocyte selenium and selenioprotein P gives more accurate results. Selenium deficiency is commonly seen in inflammatory bowel diseases, gastric by-pass surgery, and serious nutritional disorders, and it may cause hair loss, nail disorders, weight gain, fatigue, frequent infections, and infertility. In selenium intoxication, garlic-like bad breath, nausea, diarrhea, alopecia, broken nails, and neuropathy symptoms may occur. Long-term use of selenium may lead to an increased risk of insulin resistance, diabetes, prostate cancer, and skin cancer. Low selenium levels in patients are associated with thyroid cancer, Hashimoto's disease, and Graves' disease. Although most studies have shown that selenium reduced the Anti-TPO levels in patients with Hashimoto's disease, some publications have demonstrated that it did not affect the Anti-TPO levels (6–9). The decrease in autoantibody levels will be greater in patients with higher autoantibody levels when using selenium, but there is no definitive evidence that it has a positive effect on the clinical survey of the disease. Selenium supplementation did not yield any improvement in thyroid tissue, and its purported positive effects on TSH, fT3, and fT4 levels remain unproven. Additionally, the use of selenium did not reduce the levothyroxine dose. The risk of postpartum thyroiditis is higher in pregnant women who are anti-TPO-positive. Using selenium may also reduce this risk (10). Positive effects of selenium replacement have been shown in patients with mild Graves' ophthalmopathy (11).

CONCLUSION

The main element responsible for the growth of the thyroid gland is iodine. In cases where iodine is sufficient, selenium deficiency may cause growth in goiter and thyroid nodules independent of iodine. However, the results of the studies revealed on this subject contradict each other. In thyroid cancer, selenium levels may be low. As a result, patients with hypothyroidism are not recommended to use selenium routinely, but adequate selenium intake should be ensured through diet. The use of selenium should only be considered in Hashimoto's patients with selenium deficiency and patients with Graves' ophthalmopathy.

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