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THE IMPLICATIONS OF ALIMENTATION IN THYROID DISORDERS

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Abstract

Thyroid diseases are frequent all over the world. The environment and the alimentary habits have in impact on the appearance and evolution of these pathologies. This is the reason why a proper diet will help the patients to improve their health. The appliance of a diet depends on the concern of the patient, age, comorbidities and costs.

Key words: diet, thyroid

INTRODUCTION

The thyroid pathology is widely spread. Thyroid dysfunction is one of the leading endocrine disorders. It represents around 30% to 40% of the patients seen in an endocrine practice (Galofré J.C. et al, 1996). The most frequent thyroid diseases are: hypothyroidism, hyperthyroidism, nodular goiter and autoimmune thyroiditis. The mean prevalence of total thyroid dysfunction in Europe was determinate to be 3.82% (Madariaga A.G. et al, 2014)

The mean annual incidence of spontaneous hypothyroidism during a 20-year follow-up period was 3.5 per 1000 and 0.6 per 1000 in surviving women and men, respectively. The incidence data available for overt hyperthyroidism in men and women from large population studies are comparable, at 0.4 per 1000 women and 0.1 per 1000 men, but the age-specific incidence varies considerably. The prevalence of hyperthyroidism in women is between 0.5 and 2%, and is 10 times more common in women than in men in iodine-replete communities (Vanderpump M.P.J. et al, 1995).

Thyroid nodules have been reported to be founded in 4% to 7% of population on neck palpation and 30% to 50% population on ultrasonography (Bhatti Z.A. et al., 2007).

The prevalence of autoimmune thyroid disorders is estimated to be 5%; however, the prevalence of antithyroid antibodies may be even higher (Antonelli A. et al, 2015).

MATERIAL AND METHODS

The study was conducted in the first 6 months of 2018 and included patients consulted in the Endocrinology service of the Clinical County Hospital of Oradea. At the same time, the results were correlated and interpreted in the context of international references related to the subject.

RESULTS AND DISCUSSIONS

Thyroid disorders have their own protocol of treatment. Correlating these protocols with a proper diet can, sometimes, improve the outcomes of the treatment and the patient's state of health. Even so, out of 1152 patients with thyroid disorders that have been consulted and treated during January 2018 and June 2018 in the Clinical County Hospital of Oradea, only 17.18% (198 patients) of them were interested in knowing/ following a diet that is correlated to their pathology. The majority of these patients that were asking about a specific diet were young women (79.3%). The main two causes for this situation are: the concept that a more "natural" and "hormone free" approach to their problem is better for their health and weight, and the lack of other comorbidities. Usually older patients have already many diet and lifestyle indications from their cardiologist, diabetologist etc., and they try to avoid getting other restrictive indications.

Endemic iodine deficiency is one of the leading cause of hypothyroidism globally. Iodine is an indispensable component of the thyroid hormones, comprising 65% of T4's weight, and 58% of T3's. The thyroid hormones are the only iodine-containing compounds with established physiologic significance in vertebrates (Rousset B. et al, 2015).

The US Recommended Dietary Allowance for iodine is 150 μ g/day in adults, and 220 μ g/day and 290 μ g/day in pregnant and lactating women, respectively (www.nap.edu). Common dietary sources of iodine include iodized salt, seaweed, cod, shrimp, tuna, yogurt, eggs, prunes, and some grains (https://www.healthline.com/nutrition/iodine-rich-foods#section9).

In Romania, according to the law (HG nr. 568/2002), in human alimentation it is mandatory to use only ionized salt. In animal feed and in the food industry the use of iodized salt is optional, with the exception of bread and bakery products. The NaCl content of the ionized salt should not be less than 97%. Ionized salt should contain 30 mg iodine/ kg of salt, respectively 50.6 mg of potassium iodate/ kg of salt or 39.2 potassium iodide/ kg of salt. The minimum limit is 25 mg of iodine/ kg of salt, ie 42 mg of potassium iodate/ kg of salt or 32.5 mg of potassium iodide / kg of salt, and the maximum content of 40 mg iodine/ kg of salt, respectively 67.2 mg of potassium iodate/ kg of salt or 52 mg of potassium iodide/ kg of salt.

While a deficiency of iodine creates hypothyroidism due to a lack of building materials for thyroid hormone, an excess of it also creates thyroid disorders. Today, iodine excess is recognized as a risk factor for autoimmune thyroid diseases (Wentz I., 2017)

Selenium has an important impact on immune function. Selenium deficiency is accompanied by loss of immune competence. Severe nutritional selenium deficiency leads to an increased rate of thyroid cell necrosis and invasion of macrophages. It may be assumed that thyroid cell damage may initiate or maintain autoimmune thyroiditis, especially in patients susceptible to the development of autoimmune diseases (Contempre B. et al, 1992).

The recommended daily allowance of selenium is 55 micrograms/day (for an adult). The safe upper limit for selenium is 400 micrograms a day in adults. Anything above that is considered an overdose (www.webmd.com). Natural food sources of selenium include: Brazil nuts, fish, meat, cottage cheese, eggs, brown rise, sunflower seeds, mushrooms, spinach, milk, lentils, bananas etc. (www.healthline.com).

Based on our experience, dietary supplementation with 100-200 micrograms of Selenium/ day reduced the value of antibodies (Thyroid Peroxidase Antibodies) in less than 50% of the patients (and never in the normal range).

Gluten-free diet is not usually recommended among our patients with autoimmune thyroiditis, unless they have other diseases that require this kind of diet. Gluten-free diet proved an improvement in the number of thyroid peroxidase antibodies (reducing their number to half, but not to the normal range). Even if there was an improvement, we have no statistical relevance due to the reduce number of patients that followed a gluten-free diet. This kind of died implies important changes in the alimentary habits (usually not only for the patients, but also for his/hers family) and high costs.

Goitrogens are substances that disrupt the production of thyroid hormones by interfering with iodine uptake in the thyroid gland. This triggers the pituitary to release thyroid-stimulating hormone, which then promotes the growth of thyroid tissue, eventually leading to goiter (Bender D.A., 2009).

The main goitrogen foods are the vegetables in the cruciferous category. Some of the more common and potent goitrogens include the following types of food: African cassava, Broccoli, Brussels sprouts, Cabbage, Cauliflower, Kale, Mustard, Peaches, Peanuts, Radishes, Spinach, Strawberries, Watercress etc. (www.verywellhealth.com). Totally avoiding these foods is not recommended, just not to be consumed in large amount by those with nodular goiter or hypothyroidism.

CONCLUSIONS

There are dietary indications for those with a thyroid problem that can help the patient when associated with the proper treatment.

Most of the times people are not interested or do not follow these dietary regimes.

Diets are not to be confused with the real treatment or replacing them.

Studies have shown benefits of thyroid diets; they bring improvement (sometimes with high costs), but not cures.

REFERENCES

- Antoneli A., S.M. Ferrari, A. Corrado, A. Di Domenicantonio, P. Fallahi, 2015, Autoimmune thyroid disorders, Autoimmunity Reviews, Volume 14, Issue 2, Pages 174-180, https://doi.org/10.1016/j.autrev.2014.10.016
- 2. Bender D.A., 2009, "Goitrogens". A dictionary of food and nutrition (3rd ed.), Oxford University Press. ISBN 9780199234875.
- 3. Bhatti Z.A, J.A. Phulpoto, N.A. Shaikh, 2013, Multinodular goiter; frequency of malignancy. Professional Med J 20(6): 1035-1041.
- Contempre B, O. Le-Moine, J.E. Dumont, J.F. Denef, M.C. Many, 1996, Selenium deficiency and thyroid fibrosis. A key role for macrophages and transforming growth factor β (TGF-β). Mol Cell Endocrinol 124:7–15
- Contempre B., J.F. Denef, J.E. Dumont, M.C. Many, 1993, Selenium deficiency aggravates the necrotizing effects of a high iodide dose in iodine deficient rats. Endocrinology 132:1866–1868
- Contempre B., N.L. Duale, J.E. Dumont, B. Ngo, A.T. Diplock, J. Vanderpas, 1992, Effect of selenium supplementation on thyroid hormone metabolism in an iodine and selenium deficient population. Clin Endocrinol (Oxf) 36:579–583
- 7. Dietary Reference Intakes: The Essential Guide to Nutrient Requirements, http://www.nap.edu/catalog/11537.html
- 8. Galofré J.C., R.V. García-Mayor, 1996, Epidemiología de Las Enfermedades Del Tiroides . Santiago de Compostela: Tórculo Edicions; 95–118.
- Gärtner R., B.C.H. Gasnier, J.W. Dietrich, B. Krebs, M.W.A. Angstwurm, 2002, Selenium Supplementation in Patients with Autoimmune Thyroiditis Decreases Thyroid Peroxidase Antibodies Concentrations, The Journal of Clinical Endocrinology & Metabolism, Vol. 87, Issue 4, 1687–1691, https://doi.org/10.1210/jcem.87.4.8421
- 10. HG nr. 568/2002, republicata 2009, privind iodarea universala a sarii destinate consumului uman, hranei animalelor si utilizarii in industria alimentara
- 11. https://www.healthline.com/health/selenium-foods#cashews
- 12. https://www.healthline.com/nutrition/iodine-rich-foods#section9
- 13. https://www.verywellhealth.com/all-about-goitrogens-3233164
- 14. https://www.webmd.com/a-to-z-guides/supplement-guide-selenium#1
- 15. Leung A. M., 2018, The Thyroid Diet: Is There Such a Thing? , Medscape
- Madariaga A.G., S. Santos Palacios, F. Guillén-Grima, J. C. Galofré, 2014, The Incidence and Prevalence of Thyroid Dysfunction in Europe: A Meta-Analysis, The Journal of Clinical Endocrinology & Metabolism, Volume 99, Issue 3, Pages 923– 931, https://doi.org/10.1210/jc.2013-2409
- 17. Mazzaferri E.L., E.T. de los Santos, S. Rofagha-Keyhani, 1988, Solitary thyroid nodule: diagnosis and management. Med Clin North Am 72:1177-211.

- 18. Pang H.N., C.M. Chen, 2007, The incidence of cancer in nodular goiters. Ann acad Med Singapore; 36:241-43.
- 19. Rousset B., C. Dupuy, F. Miot, J. Dumont, 2015, Thyroid Hormone Synthesis And Secretion, Endotext, https://www.ncbi.nlm.nih.gov/books/NBK285550/
- 20. Vanderpump M.P.J., W.M.G. Tunbridge, J.M. French, 1995, The incidence of thyroid disorders in the community: a twenty-year follow-up of the Whickham survey, Clin Endocrinol (Oxf), vol. 43 (pg. 55-69)
- 21. Wentz I., Iodine and Hashimoto's, 2017, https://thyroidpharmacist.com