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Review Article

Hashimoto's Thyroiditis: Clinical Blind Spots and Solutions for a Hidden Epidemic

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ABSTRACT

Autoimmune thyroiditis is epidemic especially in women, but much too often it is misdiagnosed as hypothyroidism, resulting in medical treatments that are frustrating to patients as they fail to provide a lasting solution or medication-free thyroid recovery. The goal of this paper is to suggest simple clinical solutions for a fast and accurate diagnosis of Hashimoto's Thyroiditis and to offer most common causes and practical solutions.

KEYWORDS

Hypothyroidism, Hashimoto's Thyroiditis, Hashimoto's, Thyroid Dysfunction, EBV, *H. Pylori*, Mycotoxins, Mold

ABBREVIATIONS

HT: Hashimoto's Thyroiditis; TPOAb: Antiperoxidase Antibody; TgAb: Antithyroglobulin Antibodies; TSH: Thyroid Stimulating Hormones; RT3: Reverse T3; DR3-DQ2 and DR4-DQ8: Celiac Genes; CAMP: Cathelicidin Antimicrobial Peptide; Tregs: Regulatory T Cells; SIBO: Small Intestinal Bacterial Overgrowth; SCFAs: Short Chain Fatty Acids

INTRODUCTION

Hashimoto's Thyroiditis is the most prevalent autoimmune thyroid disorder and the most common cause of hypothyroidism in the US [1]. In fact, it accounts for as much as 90-97% of hypothyroid cases. Between 20 to 27mln Americans are afflicted with thyroid-related illness and up to 60% of those with thyroid disease are undiagnosed [2]. More than 12% of the US population will develop thyroid disease in their lifetime.

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While thyroid autoimmunity is epidemic [3], it continues to be misdiagnosed as hypothyroidism due to inadequate testing, a common blind spot that can easily be fixed, potentially helping millions of thyroid patients finally get the right diagnosis and therefore a more appropriate treatment and better results for thyroid recovery.

Diagnosis of HT is supported by elevated antiperoxidase antibody (TPOAb) and/or antithyroglobulin antibodies (TgAb), in most cases without biopsy. As a result of a widespread availability of thyroid function tests, subclinical HT with raised antibodies can easily be diagnosed even with normal thyroxine (T4) and normal or mildly raised thyroid stimulating hormone (TSH) [1]. It is not uncommon for a patient to have normal TSH, T4 and triiodothyronine (T3) levels but already show thyroid autoimmunity. In a group of 67 people with chronic HT, 23.8% were diagnosed with euthyroidism, 29.8% with subclinical hypothyroidism, 41.7% with primary hypothyroidism, and 4.47% with hashitoxicosis [4], so almost a quarter of HT patients could be misdiagnosed as having a normal function unless antibodies were tested. In addition, often only TSH is retested to monitor patient's thyroid status, and TSH is used exclusively for thyroid diagnosis even though studies confirm that it is not sufficient [5].

Given the wide range of normal values for TSH in HT, early Hashimoto's autoimmune process may be clinically missed. Subclinical and clinical hypothyroidism is associated with increased cardiovascular and neuropsychiatric morbidities as well as poor obstetrical and fetal outcomes to name a few, even with a relatively mild or subclinical T4 deficiency. New mothers and pre-menopausal women constitute a high percentage of HT patients. As the incidence of HT is growing, so is the likelihood of missed diagnosis**.** At certain point, thyroid damage caused by the immune system cannot be reversed, forcing the patient to rely on long term thyroid medication to function. Since HT is 5 to 10 times more common in women than men [1], all women suspected of hypothyroidism should be routinely tested for HT antibodies. Therefore, it is clinically relevant to improve diagnostic strategies and treatment of thyroid dysfunction, especially among female patients.

Blind Spots in Current Standard of Care

If HT is confirmed, standard of care is to prescribe Synthroid or a similar source of synthetic T4. Synthroid is the most common prescription for hypothyroidism and is currently (as of 2025) the fourth most prescribed medication in the USA, with approximately 23 million Americans on it, according to thyroid.org. Prescription of T4 follows elevation of TSH and/or insufficiency of T4. Following this model, one would assume that HT is a T4 or T3 deficiency, which it is not. It is an autoimmune disorder, which means that inflammatory pathways and immune triggers are causing the body's immune system to attack its own thyroid tissue.

The pituitary gland in the brain stimulates TSH to increase metabolism and produce more T4 when T4 and/or T3 are insufficient. The inactive form T4 is made first and must be converted into the active T3, a vital step that requires specific nutrients and can be affected by various factors. T3 then travels into the body providing the needed functions. In HT, immune system slowly attacks thyroid, and over time, the loss of thyroid tissue leads to decreased T4 and T3 production with eventual elevated TSH, which mimics hypothyroid. With ongoing damage to thyroid, eventually

there is so little of it left that medications become necessary, even though they do not stop the causes of the autoimmunity from continued damage. If immune system is not addressed, patient will continue requiring a higher dose of medication, which is a vicious cycle. This is what proper early testing can prevent.

To complicate things, under certain circumstances such as trauma, surgery [6], critical illness [7], severe chronic stress, or during infections, T4 converts to an inactive form called Reverse T3 (RT3), which binds to the T3 cell receptors instead of T3 but is inactive, starving cells of needed thyroid hormone activity. In that case, more T4 medication will not necessarily correct the T3 deficiency either, and infections or stress have to be addressed. Unfortunately, RT3 is another marker that is rarely tested.

TSH is another clinical blind spot because it may fluctuate between hyper-, hypo-, or normal levels, with corresponding hyper- or hypo- thyroid symptoms, increasing the risk of the wrong diagnosis and the wrong medication intervention [2]. Unless the lab work for both TSH and antibodies is repeated every few months, patients risk misdiagnosis with hyperthyroidism, hypothyroidism or normal thyroid. While other thyroid markers will fluctuate, the antibodies will remain elevated in HT.

If tested routinely, HT can be prevented from fully developing. If caught early, it can be put into remission. Since doctors already test TSH, T3 and T4, all they need to do is add RT3, TPOAb and TbAb to the lab testing requisition form. It is ethical and the right thing to do to run all these markers in every woman's annual physical lab work and/or thyroid panel. This standard of care will prevent unnecessary suffering and irreversible thyroid damage.

Triggers and Molecular Mimicry in Autoimmune Thyroiditis

One in 12 people in the US has an autoimmune disease, with HT being the most common one. Immune T-cells target thyroid for destruction, often as a result of inflammatory cascade. Autoimmune disorders are triggered by a variety of factors. In an award-winning investigative book, Donna Jackson Nakazawa reports in detail how increased incidence of autoimmune disorders is caused by increase in environmental toxicity [8]. Since increase in toxic exposure [9] can contribute to hypothyroidism and possibly HT, these patients should decrease their toxic load from: pesticides, herbicides, fluoride, chlorine, bromine, PCB, BPA, food colorings, lead, cadmium, arsenic, mercury, to name a few. Among other factors involved in HT are: oral contraceptives, pregnancy/postpartum, low-calorie diets, inflammation, certain medications, liver or kidney dysfunction, trauma, adrenal dysfunction, tobacco, and pathogenic infections. The flow chart below reiterates the complexity of HT contributing factors, with permission from Dr. Hedberg.

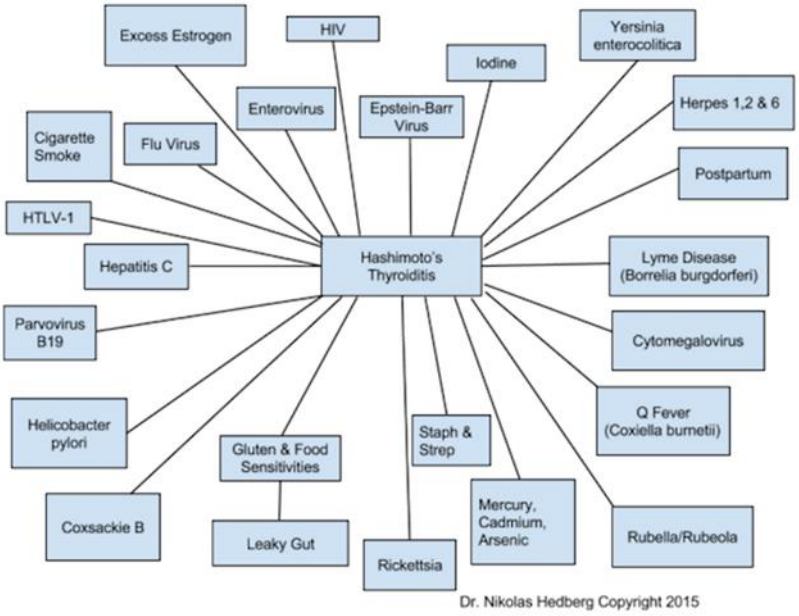

Figure 1: Dr. Nikolas Hedbergn2015.

Molecular mimicry between thyroid, the immune system, and "invaders" may be a factor in HT. In this scenario, the immune system attacks the thyroid instead of the targeted invader due to molecular semblance between both. For example, the surface peptides of bacterium *Yersinia enterocolitica* have been shown to provoke autoimmune attacks because they look like receptors on the thyroid - similar enough for the immune system to confuse thyroid for *Yersinia* [10]. Viruses such as HTLV-1, enterovirus, rubella, mumps virus, HSV, EBV, and parvovirus [11] and bacterium *H. pylori* [12] may cause HT. Functional testing is recommended to rule out these pathogenic infections.

Gluten is the best-studied example of molecular mimicry in HT [13]. Celiacs have a clinically significant incidence of elevated thyroid antibodies [14]. Studies also suggest specific celiac genes DR3-DQ2 and DR4-DQ8 predisposing to endocrine autoimmune diseases like HT. "In distinct cases, gluten withdrawal may single-handedly reverse the abnormality" [15]. HT support should include a gluten-free diet and a recommendation of DR3-DQ2 and DR4-DQ8 testing. Of note is the fact that thyroid medications are not guaranteed to be gluten free and may also be cross-contaminated [16], so care must be taken to ensure that any thyroid medication or supplement taken is gluten free.

Functional Nutrition: Solutions for Autoimmune Thyroiditis

Current standard of care for HT does not include much nutritional support even though nutrients are paramount for a healthy thyroid. Nutritional deficiencies should be assessed for selenium, vitamin E, D, A, zinc, iron and antioxidants [17], not only to provide thyroid with needed nutrients for functions but also to support "immune tolerance" [18] and decrease inflammation, both involved in HT [19]. A whole-food diet highly anti-inflammatory and rich in antioxidants, liver support, and selenium are all recommended.

The single most important nutrient in HT management is selenium [20], which by itself can lower thyroid antibodies [21]. Its deficiency may increase cellular damage of the thyroid and even set off autoimmunity. Unfortunately, selenium is commonly

deficient in diets worldwide due to a gradual deterioration of nutrient density in produce and soil [22]. In HT, daily supplementation with selenium is the best option to ensure a predictable level of daily selenium intake.

Vitamin D is a hormone well studied for its importance against autoimmune diseases, including HT [23], and it is needed for tight junctions in the intestinal lining, preventing intestinal permeability. A single layer of enterocytes (the intestinal gut cells) lines the small intestine - just one single layer decides what is safe or unsafe to enter the blood stream. Enterocytes are only as tight as the "tight junctions" between them, the gates that open or close depending on what is trying to pass. One of the regulatory proteins called zonulin triggers temporary opening of the tight junctions and can over-react when exposed to gluten, parasites, candida, SIBO and other infections, toxins, or stress [24], and Vitamin D is needed for regulation of these junctions. One more benefit of Vitamin D is that adequate levels increase the cathelicidin antimicrobial peptide (or CAMP), an antimicrobial and antibiotic that might be particularly relevant since HT has been associated with various pathogens [25], including viral infections like Epstein-Barr Virus [26].

Iodine is regarded as number one nutrient for thyroid support in standard of care, but the contrary is suggested by both animal and human studies for individuals with genetic predisposition to HT [27]. Excessive iodine causes more autoimmune reactivity [28], possibly by increase in hydrogen peroxide. A 3-month restriction of iodine to less than 100 mcg a day in a group of 23 patients with HT resulted in 18 patients (78.3%) recovering to euthyroid state [29]. As a comparison, a typical multivitamin contains 150mcg iodine. Iodine is needed for T4-T3 conversion and every cell in human body has iodine receptors, but care should be taken to limit iodine intake in HT. Also, switching from iodized to a more natural form of salt may be helpful.

While individual nutrients are of importance, HT patients may not improve until their immune tolerance does. Dr. Kharrazian's studies on dietary and chemical reactions in HT led him to propose that peripheral tolerance can improve with digestive enzymes and hydrochloric acid for proper protein breakdown, splitting amino acid sequences from consumed foods, thus helping prevent cross reactivity. Otherwise, longer amino acid sequences of undigested protein become target sites for antibodies, which can lead to gut permeability, inflammatory cascade, and eventually immune reactions [17].

Interestingly, hypochlorhydria is a mostly missed complication of hypothyroidism and HT and so it should be part of patient assessment and management. A healthy thyroid provides proper stimulation of hydrochloric acid [30]. Hypochlorhydria is associated with increased risk of pathogenic infections such as SIBO or yeast overgrowth, and key vitamin and mineral deficiencies such as B6, B12, folate, iron, calcium, zinc and possibly more, and therefore should be assessed in patients with hypothyroidism. Hypo- and achlorhydria also impair absorption of Thyroxin and will require a 37% higher dose for the same therapeutic effect [31].

If T-cells target thyroid for destruction, T-regulatory (Treg) cells have the capacity to improve immune tolerance and block the inflammatory pathways. Patients should be encouraged to increase short chain fatty acids (SCFAs) acetate, butyrate, and propionate in diet to boost Treg activity. SCFAs are a byproduct of metabolism of microbiota, the good bacteria in the gut, another important factor ignored in HT care. In order to produce SCFAs, the beneficial colonic bacteria need to consume dietary fiber. Stress and lack of fiber will cause the microbiome to deteriorate. Diet low in fiber, high stress, and antibiotic-induced dysbiosis can also weaken Tregs, trigger imbalanced immunologic functions and thus contribute to HT and other autoimmune disorders. Additionally, the intestines convert about 20 percent of T4 to T3 but only in the presence of sufficient amount of beneficial gut bacteria [2].

The best way to increase the diversity of microbiota is to consume a variety of foods rich in fiber, manage stress, decrease the amount of meat and dairy, only consume antibiotic-free animal protein, and avoid antibiotics unless absolutely necessary. Probiotic supplementation as well as fermented or pre- and probiotic foods (except during SIBO) may help improve immune signaling and crowd out opportunistic pathogens, which is important in HT as in any autoimmune disorder.

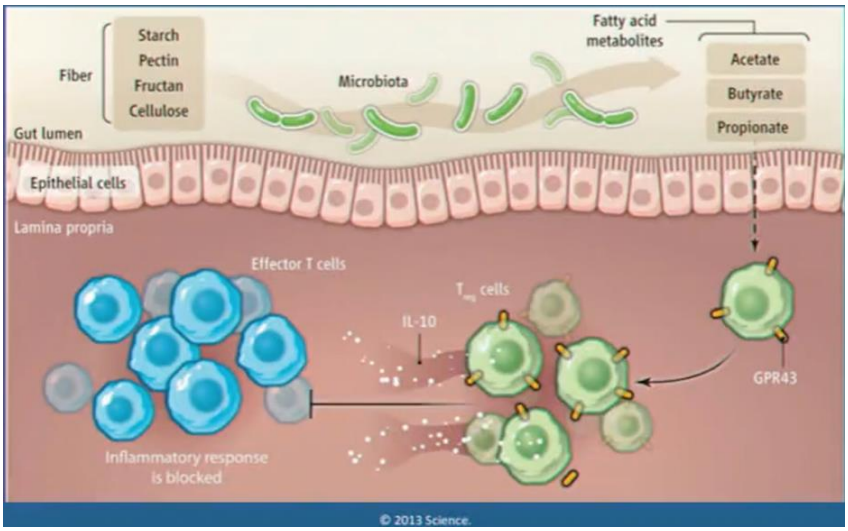

Figure 2: Bollrath J and Powrie F. *Science Magazine.*

While specific dietary protocols are available for patients with HT, these are beyond the scope of this paper and will depend on the cause of thyroid autoimmunity in the patient. For example, if HT is caused by a particular infection, that infection needs to be the target protocol. Otherwise a therapeutic diet for HT alone may fail.

Example of Resolution of Thyroid Disfunction Using a Functional Nutrition Approach

My personal case is an example of an effective and comprehensive approach to thyroid health. I developed symptomatic but clinically barely "sub-optimal" T3 level due to sustained stress and overwork, with cortisol starting to affect the T4-T3 conversion, and resulting in increased RT3. My HT antibodies were negative, and I did not have common thyroid-affecting deficiencies: my iodine and selenium levels were healthy. Thyroid is very sensitive to environmental toxins and I was already consuming organically grown foods, drank filtered water (with key thyroid toxins chlorine and fluoride filtered out), and used air filtration, so I had limited toxic exposure. I also did not take any medications or oral contraceptives, which can affect thyroid. Doing thorough testing brought me a peace of mind and provided me with the right direction for personal thyroid recovery, and patients deserve that peace of mind as well. My thyroid self-regulated with proper supplementation for adrenal support, more sleep, and more rest. Testing antibodies to thyroid was important in order to exclude thyroid autoimmunity, and while diet was already healthy, adjusting the lifestyle and adding needed supplement support provided the answers needed, while preventing a need for a thyroid medication. Thyroid dysfunction is complex and multifaceted whether it is autoimmune or not, and responds well to a holistic approach, and finding the root cause of the dysfunction is the number one priority.

Most Common Causes of Autoimmune Thyroiditis as Seen in a Clinical Practice

I've seen a pattern of quite a number of my patients with chronic active EBV also showing positive antibodies to thyroid, sometimes undiagnosed. These patients are able to put HT into remission by clearing the EBV infection, while other therapeutic approaches to thyroid may have failed. This indicates that EBV is a major driver of HT and is consistent with studies suggesting a link between both conditions [32].

H. pylori has been implicated in autoimmunity, including HT [33], another infecting agent that I see in patients with both EBV and HT. Clearing both infections results in normalization of thyroid if caught early enough. It is therefore prudent to test patients with newly diagnosed HT for chronic active EBV and possibly *H. pylori*.

Finally, while more studies are needed, mycotoxins are the most common environmental toxin I have seen in chronic EBV patients, and certain mycotoxins may trigger autoimmunity, including possibly HT. It is not unusual for a chronically ill patient that has failed to respond to all prior medical interventions to suffer from the combination of chronic EBV, HT, *H. pylori*, and mold exposure. Some of these patients may not even be aware of the ongoing water damage in their environment. In these cases, identification of mold, removal from the mold exposure and mold remediation will be necessary in order for the EBV protocol to be effective and damage to thyroid to stop, possibly including HT[34-37].

CONCLUSION

Patients continue to suffer with undiagnosed or misdiagnosed HT, while proper diagnostic tools are available and simple to implement in medical practice. The current standard of care is antiquated and fails to recognize how common thyroid autoimmunity is. If the patient is lucky to be diagnosed properly, there is no nutritional or lifestyle support beyond prescription medication, be it immune-modulation, stress management, correcting deficiencies, treating infecting agents and improving diet. Years of suffering and irreversible thyroid damage can be prevented by proper thyroid testing, including thyroid antibodies and RT3 as part of an annual physical or routine blood testing, especially for female patients. We should educate these patients on how to improve their immune function, help them repair the gastrointestinal tract, and encourage them to consume organic, pesticide-free anti-inflammatory and anti-oxidant foods rich in fruits and vegetables, eliminate gluten, purify their drinking water, test for intestinal pathogens and EBV, and improve their Selenium, Vitamin D, Vitamin A and microbiota status. Hashimoto's Thyroiditis is a complex autoimmune condition but a lot more can be done to support this population and possibly help some of these patients put HT completely into remission.

CONFLICT OF INTEREST:

Kasia Kines is a CEO and Founder of EBV Global Institute and author of The Epstein-Bar Virus Solution book.

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