CASE REPORT

Healing of Graves' Disease Thorough Lifestyle Changes: A Case Report

Kelly Brogan, MD; Gisela Marcelino, Christina Pedro, Alyssa Siefert, PhD

ABSTRACT

Background • Graves' disease is known as a chronic and incurable disease. The typical treatment is symptom-based and consists of medications, radioiodine, or surgery. These last two treatments are routinely offered to the 50% of patients that do not respond to drug therapy. Here we report the case of a patient who was able to normalize her thyroid hormones as well as her autoimmune markers in 6 months with the exclusive implementation of lifestyle interventions.

Summary • A 34-year-old Dutch, Caucasian female diagnosed with Graves' disease since 2014 implemented lifestyle modifications, which included dietary change to an ancestral type of diet, oral health interventions, practice of kundalini yoga, avoidance of environmental toxicants (by only eating organic food, drinking filtered water, and using natural products to clean her house or for her personal hygiene) and supportive supplements when necessary. The patient did not take any antithyroid drugs or beta-blockers during this period nor any other type of medication that could have had immunosuppressant effects. After 6 months of engaging in these lifestyle interventions, her thyroid analysis normalized and no anti-thyrotropin receptor antibodies were negative.

Conclusion • This case report demonstrates that Graves' disease can effectively be put into lasting remission without conventional medical interventions. It also emphasizes the importance of a healthy lifestyle as a first line intervention for all patients but especially in the particular case of patients suffering from Graves' disease. (*Adv Mind Body Med.* 2018;33(2):4-11.)

Kelly Brogan, MD, is a practicing psychiatrist; Gisela Marcelino is a resident of internal medicine, Department of Internal Medicine, Cantonal Hospital of Fribourg, Switzerland and student in the Master of Human Nutrition at the University of Bridgeport, USA; Christina Pedro is a student at the Indiana University School of Medicine, USA; Alyssa Siefert, PhD, is in the Department of Biomedical Engineering at Yale University, USA.

Corresponding author: Kelly Brogan, MD
E-mail address: drbrogan@kellybroganmd.com

INTRODUCTION

Graves' disease is an autoimmune condition in which activated lymphocytes infiltrate the thyroid gland, leading to local inflammation or chronic thyroiditis.[1] These lymphocytes also produce antibodies that can affect thyroid gland functioning by stimulating the production of thyroid hormones or by blocking their production.[1] It is also possible for antibodies characteristic of Graves' disease and of Hashimoto's disease to coexist, but symptoms will depend on the levels of each of these antibody isotypes.[1] Still, the most frequent antibodies found in Graves' disease are antibodies that stimulate the Thyroid-Stimulating Hormone (TSH) receptor, which consequently leads to cellular proliferation, an increase in the production of thyroid hormones, and possible goiter formation.[1]

Furthermore, antibodies against the TSH receptor (TRAb) can have stimulating effects on other sites of the body, as TSH receptors are also localized in adipocytes and fibroblasts.[2] This systemic activation may underlie the orbitopathy and pretibial myxedema observed in patients with severe disease.[2] Increased circulating thyroid hormones in the body affect many bodily systems, including the cardiovascular system, which may lead to tachycardia, atrial fibrillation, high systolic blood pressure, dyspnea, high HDL cholesterol, and low total cholesterol.[2] Elevated thyroid hormones can impair glucose metabolism, producing hypoglycemia or hyperglycemia. Additionally, the gastrointestinal system is impacted, leading to diarrhea, malabsorption, rarely steatorrhea, abdominal pain, vomiting, and dysphagia (if the goiter is large enough to compress the trachea). The genitourinary symptoms induced by elevated

thyroid hormones include low libido, oligo/amenorrhea in females, infertility, gynecomastia in males, pollakiuria and nocturia, and the hematologic system is also affected by anemia exhibited by low levels of ferritin. Importantly, neuropsychiatric symptoms can manifest as anxiety, restlessness, emotional lability, depression, psychosis, insomnia, impaired concentration, orientation, and memory. Cutaneous signs may include alopecia, onycholysis, sweating, hives, pruritus, hyperpigmentation, and ophthalmological problems are especially characterized by an orbitopathy (ocular proptosis and pain, diplopia). Adrenal function can also be impaired due to a decrease in the free cortisol levels. Finally, Grave's disease can affect the osteoarticular system by leading to osteoporosis and thyroid acropachy.[2]

The etiology of Graves' disease is multifactorial and somewhat unknown.[1] Medical literature recognizes a genetic predisposition, but environmental and personal factors may also be responsible for precipitating Graves' disease.[1] Environmental factors include viral triggers, radiation, and medication exposure.[1] Personal factors include female gender, stress, and smoking cigarettes.[1]

The three gold standard treatments for thyrotoxicosis are antithyroid drugs, surgery, and radioiodine; treatment choice varies by geography.[3] Symptoms frequently subside with thioamide medications (which reduce the production of thyroid hormones as well as the peripheral conversion of T4 to T3) and with beta-blockers (which block sympathetic activation).[3] However, thioamide medications are not curative and are associated with a 37% of relapse rate after drug withdrawal. Alternative treatments include radioiodine (which leads to the destruction of the gland) or surgery (removing the thyroid gland), offered to approximately 50% of patients that do not respond to drug therapy.[3] Complications of radioiodine therapy include worsening of pre-existing eye disease and radiation thyroiditis.[4] Risks of surgery include postoperative hemorrhage, damage to the recurrent laryngeal nerve, and induced hypoparathyroidism.[5] Radioiodine is preferred in the United States and areas where thioamide medications are not available.[3] In contrast, European endocrinologists prefer a conservative approach in the majority of these patients, with radioiodine and surgery less frequently prescribed.[3]

To the authors' knowledge, lifestyle change as a therapeutic option to patients suffering from Graves' disease has never been reported in the literature. Here we present the dramatic healing of a patient with Graves' disease with only dietary modification, oral health interventions, physical exercise, meditation, natural supplements, and deliberate avoidance of environmental toxins.

PATIENT INFORMATION

Social and Family History

The patient is 34 years old, born and raised in Holland, and has one sister who is 6 years older. Her parents divorced when she was 12. She married in 2010 and moved to New York City, USA, in 2011 due to her husband's work. Her mother may have had undiagnosed depression.

Table 1. Physical examination, natural supplements, and typical diet before treatment

Height & weight	5'11" & 135 lbs
Supplements	bugleweed, cache, lemon balm, red clover, detox, B_{12}-2000
Typical day's diet	
Breakfast	oatmeal with blueberries, blackberries and strawberries or banana shake with coconut milk and raw cacao
Lunch	salad
Dinner	vegetables with meat and quinoa

Psychiatric History

In her early 20s, patient exhibited low mood, cloudiness, cutting behavior, and chronic irritability. The chronic irritability still persists until today. She has seen a therapist for 2 years. No history of eating disorders, obsessive compulsive disorder, suicidality, mania, or psychosis. She has a history of panic disorder without agoraphobia and generalized anxiety disorder but she has never been hospitalized in a psychiatric hospital.

Medical History

Graves' disease was diagnosed on January 7, 2014 by laboratory and radioactive uptake testing.

One pregnancy and one birth, with a female baby born on 5/27/13. The patient had epidural anesthesia and episiotomy. Menarche at 13. No surgeries.

Personal history of substance abuse

The patient consumed 1-2 glasses/week of alcohol. The consumption started at 14 years old with a more intensive use between the ages of 18 and 20 years old. She did not report any other substance use.

Physical examination, supplements that the patient was taking, and typical diet

The first physical exam, the natural supplements, and the typical diet of the patient before treatment are summarized in Table 1.

Of note, the patient stopped taking metoprolol before seeing Dr. Brogan because her heart rate had normalized. She also took many natural supplements (see Table 1) and her typical diet was already what is considered a "healthy diet" rich in fruits and vegetables, without processed foods or refined sugars.

Mental Status Examination

The patient was casually dressed with shoulder length brown hair and makeup. No gait disturbance. She was friendly, appropriate and exhibited good eye contact. She was oriented in space, time, and to person. She was euthymic.

She spoke normally but with accent. Her thought process was logical and goal-directed. No suicidal, homicidal, or persecutory ideation. No ruminations were present. She did not present auditory or visual hallucinations. Insight and judgment fair.

DIAGNOSTIC ASSESSMENT

DSM-IV Impression

Axis I. The patient was experiencing anxiety and mood disturbances secondary to her Graves' disease

Axis II. Evaluation was deferred for continued observation.

Axis III. Graves' disease is the medical conditions that is affecting the patient's current psychiatric health.

Axis IV. Stressors include motherhood, tension with her own mother, her stressful job, and being an immigrant.

Axis V. The patient's global assessment of functioning at time of presentation was determined to be a score of 70 which corresponds to mild symptoms or some difficulty in social or occupational functioning, but generally able to function adequately with meaningful interpersonal relationships.

Formulation. Patient is a 34 year old married, Caucasian female with a history of postpartum-onset Graves' disease presenting for consideration of lifestyle interventions as an alternative to surgical and radiotherapy.

Laboratory Testing

Thyroid analysis results are summarized in Table 3. Further blood analyses were also performed (but only out-of-range results were included in Table 3) which included hemogram, hepatic and renal function tests, electrolytes, lipid panel, glucose, hemoglobin A_{1c}, C-reactive protein, sedimentation rate, uric acid, vitamins B_6, B_{12} and D, methylmalonic acid, ferritin, zinc, copper, selenium, homocysteine, cortisol, and DHEA (dehydroepiandrosterone) sulfate plasma levels as well as an MTHFR (methylenetetrahydrofolate reductase) status and a carnitine panel. Other autoimmune antibodies were also assayed, including antinuclear antibody, rheumatoid factor, anti-transglutaminase and anti-endomysial antibodies and immunoglobulin A serum levels. Metals like lead, arsenic, cadmium, iodine and mercury were also tested. All the analyses were performed at Manhattan Labs (New York, USA).

Table 2. Laboratory results of note from March 2014 and before lifestyle interventions

Laboratory Test	Result	Reference Range
TSH	0.0019 uIU/ml	0.35 – 4.94 uIU/ml
Free T3	7.04 pg/ml	1.7 – 3.7 pg/ml
Reverse T3	41.8 ng/dL	9.0 – 27.0 ng/dL
Free T4	2.14 ng/dL	0.7 – 1.48 ng/dL
TRAb	4.0 IU/L	≤1.75 IU/L
TgAb	4.23 IU/ml	< 4.11 IU/ml
TPOAb	4.51 IU/ml	< 5.61 IU/ml
Vitamin D, 25-OH	24.4 ng/ml	30 – 100 ng/ml
DHEA sulfate	172.7 µg/dL	12 – 535 µg/dL
Zinc	328.9 µg/dL	440 – 860 µg/dL
MTHFR status		
MTHFR mutation: C677T	Heterozygous	
MTHFR mutation: A1298C	Heterozygous	
Carnitine panel		
C2, acetyl	17.94 µmol/L	3.74 – 16.56 µmol/L
C5-DC, glutaryl	0.13 µmol/L	0.00 – 0.09 µmol/L
C6, hexanoyl	0.36 µmol/L	0.00 – 0.12 µmol/L
C8, octanoyl	0.97 µmol/L	0.00 – 0.23 µmol/L
C10, decanoyl	1.63 µmol/L	0.00 – 0.31 µmol/L
C10:1, decenoyl	0.76 µmol/L	0.00 – 0.31 µmol/L
C12, dodecanoyl	0.44 µmol/L	0.00 – 0.12 µmol/L
C12:1, dodecenoyl	0.46 µmol/L	0.00 – 0.17 µmol/L
C12-OH, 3-OH-dodececanoyl	0.03 µmol/L	0.00 – 0.02 µmol/L
C14, tetradecanoyl	0.09 µmol/L	0.00 – 0.05 µmol/L
C14:1, tetradecenoyl	0.41 µmol/L	0.00 – 0.16 µmol/L
C14:2, tetradecadienoyl	0.16 µmol/L	0.00 – 0.12 µmol/L
C14:1-OH, 3-OH-tetradecenoyl	0.05 µmol/L	0.00 – 0.02 µmol/L
C16:1, palmitoleyl	0.07 µmol/L	0.00 – 0.04 µmol/L
C18:1, oleyl	0.20 µmol/L	0.00 – 0.17 µmol/L

Abbreviations: DHEA, dehydroepiandrosterone; FT3, free T3; FT4, free T4; MTHFR, methylenetetrahydrofolate reductase; RT3, reverse T3; TgAb, thyroglobulin antibody; TPOAb, thyroid peroxidase antibody; TRAb, thyrotropin receptor antibody; TSH, thyroid-stimulating hormone.

INTERVENTIONS

The patient benefited from lifestyle interventions based on the book of the main author entitled *A Mind of Your Own*.[6] The protocol included dietary change to an ancestral diet and included daily physical exercise, meditation sessions, and some natural supplements when needed (listed in Table 4). The patient was also encouraged to drink only filtered water and to maintain a healthy oral hygiene by including oil pulling and Hydro Floss in her daily routines. Resources for self-education on how to replace toxic home products and personal cosmetic/

Table 3. Symptoms and thyroid analysis during follow-up by Dr. Brogan.

2014	2015				2016	2017		2018
	February----------PREGNANCY----------October							
May	October	March	July	September	April	June	December	April
Symptoms								
Night sweats, Hair loss, Tearfulness, Forgetfulness, Palpitations, Weight loss, Agitation, Low libido, Vaginal dryness, Intense morning appetite, Perpetual snacking, Energized by meals, Acne and Dry skin	Hair loss, Fatigue, Low libido	Fatigued, Nausea, Dizziness, Acne, Palpitations (<100bpm)	Fatigue, Hair loss, Dry skin, Acne, Diarrhea after recent exposure to gluten	Hair loss, some Dizziness, Nausea	Nausea after breakfast, Loose stools	Hair loss, Diarrhea	Hair loss	Hair loss
Outcomes								
TSH 0.0006 uIU/mL **TRAb 2.05 IU/L** TgAb 3.13 IU/ml TPOAb 2.11 IU/ml FT3 3.45pg/ml RT3 22.9ng/dl FT4 1.39ng/dl	TSH 0.4611 uIU/ml TRAb <0.90 IU/L TgAb 2.46 IU/ml TPOAb 1.36 IU/ml FT3 2.88 pg/ml RT3 15.1 ng/dl FT4 1.00 ng/dl	**TSH 0.18 uUI/mL** TRAb <0.90IU/L TgAb 1.30 IU/ml TPOAb 1.30 IU/ml FT3 2.8 pg/ml **RT3 27.7 ng/dl** **FT4 0.18 ng/dl**	TSH 0.62 uUI/mL TRAb 119% (N ≤122%) TPOAb 0.80 IU/ml FT3 2.8 pg/ml **FT4 0.59 ng/dl**	**TSH 0.09 uUI/mL** TRAb 110% (N ≤122%) TPOAb 0.50 IU/ml FT3 2.7 pg/ml FT4 0.94 ng/dl	TRAb 109% (N ≤140%) **TgAb 2 IU/ml** TPOAb 2 IU/ml FT3 3.3 pg/ml FT4 1.6 ng/dl	TSH 0.918 uUI/mL TRAb <0.10 (N <0.55) TPOAb 3 IU/ml FT3 3.2 pg/ml FT4 1.34 ng/dl	TSH 1.110 uUI/mL TRAb 0.10 (N <0.55) TPOAb 2 IU/ml FT3 3.2 pg/ml FT4 1.43 ng/dl	TSH 1.10 uUI/mL TRAb <0.10 (N <0.55) TPOAb 1.0 IU/ml Tg 6.5 ng/ml TPO 2 IU/ml FT3 3.0 pg/ml FT4 1.33 ng/dl

Note: Abnormal laboratory results are presented in bold. All analyses until 2015 were performed at Manhattan Labs in New York, NY USA. The analyses from 2016-2017 were performed at Quest Diagnostic Incorporated, in Irving, USA. After 2017, analyses were performed at the Clinical Pathology Laboratories, in Austin, TX, USA.

Abbreviations: N, normal range; FT3, free T3; FT4, free T4; RT3, reverse T3; TgAb, thyroglobulin antibody; TPOAb, thyroid peroxidase antibody; TRAb, thyrotropin receptor antibody; TSH, thyroid-stimulating hormone.

hygiene products with more natural alternatives were also provided.

Of note, the protocol outlined in *A Mind of Your Own* is a modified type of ancestral diet, characterized by moderate carbohydrate consumption, according to Dr. Weston A. Price's discoveries that no traditional human group followed a vegetarian diet or a low-fat diet; all traditional diets were local, natural, and whole and included some raw food.[7] It also distinguishes from the paleo diet because gluten and beans are allowed to be reintroduced if necessary. Therefore, in this type of ancestral diet, patients are advised to eat meat, wild fish, eggs, nuts and seeds, and fruits and vegetables, while removing all processed foods, refined carbohydrates, dairy, gluten, all grains (not including quinoa and buckwheat), legumes, corn, and soy for one month. After one month, the removed foods can be reintroduced back to the diet if desired by the patient and according to their tolerance to these foods. All food should be organic.

The patient also started Kundalini yoga, a practice that combines physical exercise with meditation and provides full relaxation to the mind and body.

All interventions began on the date of the patient's first consultation in Dr. Brogan's office in 2014.

OUTCOMES AND FOLLOW-UP:

Since the patient began working with Dr. Brogan, she reported a progressive improvement of her symptoms.

The patient presented with signs and symptoms of Graves' disease, including hair loss, tearfulness, forgetfulness, palpitations, weight loss, agitation, and low libido, as confirmed by laboratory testing: low TSH (0.0019 uIU/ml), high free T4 and free T3 (2.14 ng/dL and 7.04 pg/ml, respectively) and positive TRAb (increased to 4.0 IU/L). In the first analysis performed by Dr. Brogan, the patient also presented with vitamin D deficiency (24.4 ng/ml), adrenal fatigue corroborated by a low DHEA sulfate (172.7 µg/dL), and zinc deficiency (328.9 µg/dL) that were corrected through supplementation. Carnitine deficiency was also identified due to an increase in the serum levels of many free fatty acids, and therefore, the patient received carnitine supplementation. An MTHFR status was also performed, but the mutations identified (C677T and A1298C) are actually known as protective polymorphisms for Graves' disease.[8]

As the patient followed the program (see Table 3), the signs and symptoms of Graves' disease gradually disappeared. Notably, 6 months after the start of the program (in October 2014), and despite residual fatigue and low libido, the patient's thyroid analyses normalized, including a negative TRAb (which continues to be negative at present). The patient's FT3 and FT4 normalized after only one month after the start of the program. In the beginning, the patient also presented symptoms of glucose dysregulation (frequent snacking, intense appetite in the morning, and only feeling energized by meals) but these symptoms resolved as well. As the patient's symptoms of vaginal dryness could be due to a dysregulation of the vaginal flora induced by the hyperthyroidism, the patient received a vaginal probiotic (Fem-Dophilus® by Jarrow that

Table 4. Supplements prescribed before, during, and after pregnancy and intermittently according to the needs of the patient.

Before pregnancy	During pregnancy	After pregnancy
General support and correction of deficiencies		
Carnitine		
Fatty acid support		
Selenium		
Prenatal 3 twice daily	Prenatal 3 twice daily	Multivitamin
	Netti Pot	
		Maca
		Flavonoids
		Vitamin C
	5MHTF 1mg	
	Vitamin D/K 5000IU	Vitamin D
	Cod Liver 2-3/week	Liver – 3 twice daily
	P50	
	Mag oil	Mag
Supplements for energy/mood support		
		Hypothalamus
	Adrenal – increased progressively	Adrenal – increase to 3 in the morning and 2 at night
	Thyroid –1pill 2x/day	Two twice daily
	Methylcobalamine – once daily	Methylcobalamine – once daily
		PC ¼ tsp per day
Supplements for digestive support		
Antimicrobial herbs	Antimicrobial herbs	
Probiotics	Probiotics	
		Liver flush adapted from Dr. Nicholas Gonzalez
		HCL
	Digestive	Digestive
	Pancreas 3 with breakfast	Pancreas 3 with breakfast
	Ohirra	Ohirra
		Enzy mes 2 w meals
		Sonne's Bentonite clay – 1 tbsp twice a
		Vital 10 probiotic
		Progurt
		Duozyme
		GSE
		Biocidin
		EPO
		CLO
Supplements for vaginal dryness		
	Femdophilus by Jarrow: insert one vaginally every night	
Supplements for cognitive support		
	Barleans 3-6-9 one tbsp daily Gargle for 1 week with a whole glass of water, each sip gargling forcefully until you tear	
	Chlorophyll	
Detox		
		Gi Detox
		Coffee enema
Hair loss		
		Lysine

contains the strains Lactobacillus rhamnosus, GR-1® and Lactobacillus reuteri, RC-14®), and the symptoms resolved. Low grade hair loss persisted during all follow-up, as can be observed in Table 3, but unexpectedly, it does not appear to be a result of Graves' disease, vitamin A, vitamin B, vitamin C, vitamin D, iron, selenium, and zinc deficiency[9] or due to diabetes[10] (results not shown) and persists.

In February 2015, the patient was able to conceive, and with the exception of the first trimester in which Graves' disease relapsed (the patient was tired, had palpitations, and her TSH lowered and FT4 increased again), pregnancy progressed without any other significant problems. Ultrasounds during pregnancy were normal. Labor occurred at 40 weeks and 5 days, in October 2015, also without any complications. The newborn was a girl, born at home, weighed 8lbs and 10 oz, lengthened 21 inches, and had no signs of neonatal hyperthyroidism. The patient also informed the authors that her daughter, at the time of the writing of this text, is healthy and has had normal development.

During follow-up, the patient frequently presented gastro-intestinal problems, namely nausea, bloating, and tendency to diarrhea that could have been aggravated by pregnancy, but were specially attributed to gut dysbiosis and intestinal permeability. In fact, a stool analysis performed in May 2016 confirmed the presence of an imbalanced flora or dysbiosis due to the presence of 4+ *Escherichia coli* and many *Blastocystis hominis* species. Despite maintaining the same diet (having had only one intake of gluten in July 2015 which lead at the time to diarrhea), gut dysbiosis was ultimately corrected in the end of 2017 with the help of supplements (namely, anti-microbial herbs, probiotics, and digestive enzymes) directed to improve the gut, including the gut flora, the gut lining and the digestion. The acne and the dry skin presented by the patient, even before pregnancy, could also be a sign of intestinal permeability because those symptoms did not reappear once the gut function was restored.

Of note, the interventions of this program also led to a decrease in inflammation markers: the patient's sedimentation rate decreased from 10mm/hr (in the start of the program) to 2mm/hr at the end of the follow-up.

DISCUSSION

The remarkable improvement of this patient with only lifestyle changes demonstrates that Graves' disease can be healed with a natural, integrative approach. Most importantly, this patient was spared from invasive treatments with lifelong impact, such as radioiodine or surgical removal of the thyroid gland.

Recent literature recognizes the importance of a healthy gut microbiome and of a healthy intestinal barrier as a means of avoiding autoimmune disease.[11] In fact, gut dysbiosis, which includes low diversity and low keystone bacterial strains in the gut, can lead to the proliferation of opportunistic bacteria with Paneth cells in the gut lining that then produce inflammatory cytokines that summon lymphocytes, further increasing the inflammatory cascade.[12] Following these events, there is a loss of the auto-control of the intestinal immune system and the gut barrier is disrupted, leading to intestinal permeability, and oral tolerance is lost.[13] That is, many proteins from food or bacterial toxins that are present in the intestines can enter the circulation, and by a mechanism of molecular mimicry, lead to the production of autoantibodies, which in the beginning may be subclinical but when in a more advanced stage can lead to clinically symptomatic autoimmune diseases like type 1 diabetes,[11] lupus,[14] ankylosing spondylitis,[13] celiac disease,[13] autoimmune hepatitis,[15] and primary sclerosing cholangitis.[16] Celiac disease is one of the most well-known diseases associated with thyroid autoimmune disease, and its physiopathology is due to the production of antibodies against the thyroid gland by a process of molecular mimicry to gluten proteins.[17] However, due to the loss of oral tolerance in intestinal permeability, gliadin proteins may be rendered antigenic. This clinical phenomenon is referred to as non-celiac gluten sensitivity and may predispose to autoimmunity.[18] As a Western-type diet has also been associated with gut dysbiosis and mucosal inflammation of the gut lining, both in animal and human studies,[19,20] an anti-inflammatory diet was also included in the patient's treatment protocol.

Natural supplements were also recommended to restore vitamin and mineral deficiencies. A wealth of medical literature supports that vitamin D[21] and zinc[22] deficiency can lead to autoimmunity and to thyroid autoimmune diseases in particular. Selenium supplementation has also been shown to improve thyroid function in patients with Graves' disease, according to a recent meta-analysis.[23] As carnitine levels can also be decreased in patients with hyperthyroidism, supplementation with carnitine can help control these symptoms, especially as carnitine is a natural inhibitor of the thyroid hormone.[24] Similarly, omega-3 fatty acid deficiency has been associated with many autoimmune diseases, and its supplementation has been shown to improve autoimmune titers for many diseases.[25] In fact, an anecdotal case of self-healing from Graves' disease with omega-3 fatty acid supplementation exists in the literature.[26]

Supplements to support adrenal function (including vitamins B and C) were taken by the patient, as it has been shown that they can help with adrenal fatigue.[27-29] As gut dysbiosis is associated with malabsorption, the patient was also prescribed digestive enzymes.[30]

Periodontitis has been associated with an increased risk of preterm labor and low birth weight.[31] In addition, patients with both Graves' disease and periodontal disease have higher inflammation than patients with each disease separately.[32] A recent meta-analysis suggested that oil pulling may be an efficacious and cost-effective method to improve oral health.[33] Likewise, Hydro Floss has also been shown to maintain oral health.[34] Therefore, the importance of oral hygiene cannot be emphasized enough, especially for pregnant women, and strategies were provided in accordance to the patient here reported.

Physical exercise is very important to our general health, and it has been shown to decrease the risk of relapse in patients with Graves' disease.[35] Likewise, meditation was proven to be a therapeutic ally in many autoimmune diseases.[36] Therefore, these two interventions were also included in the protocol of our patient.

Finally, the patient was also asked to eat only organic food, drink filtered water, and avoid environmental toxicants, because it is known that pesticides, food additives, and many other chemicals that can be found in our environment are associated with autoimmune diseases, including thyroid autoimmunity.[37-39]

CONCLUSION

We report here the first case of Graves' disease treated and healed with only lifestyle interventions. The scientific background that supports each lifestyle intervention is presented. We also provide, for the first time, an alternative and definitive strategy to heal patients suffering from Graves' disease. The remarkable amelioration of this patient's symptoms suggests that radioiodine treatment or surgical removal of thyroid gland, which are associated with many risks in the short and long-term, may not be the optimal therapeutic interventions for Graves' disease.

PATIENT PERSPECTIVE

The last posted commentary in 2018 was the following:

> About my current update. Health-wise I still seem to be stable. Ever since the first time my antibodies were below the threshold back in the summer of 2014 (I believe), they remained there. My latest test results (from April this year) still showed optimal thyroid hormone levels. With everything else in the normal range. I started seeing Rachel Koenig [for endobiogeny] about hair loss. That's something that once reversed in all the time we worked together, before I got pregnant with my 2nd. And remains a mystery. We're working now mainly on improving blood sugar levels and adrenals. Besides this, I feel well and have few symptoms (there'll always be something more to wish for health wise ;-)). I'm feeling a lot better than many years even before my diagnosis with Graves' disease. And I'm grateful that I could save my thyroid and regain my health. That was the most empowering and amazing feeling in the world. That my body had my back all along. And just by giving it a little more of what it needed, it bounced back beautifully. Cheesy as it may sound, I finally found some peace with my body and me. And I feel grateful for having found you, Dr. Brogan. Your work and my healing journey inspired me to work with women like me, with Graves' disease and teach them how to take back their health and lives.

INFORMED CONSENT

Patient has reviewed this document and consented to all of the information herein.

REFERENCES

1. Davies TF. Pathogenesis of Graves' disease. https://www.uptodate.com/. Updated November 14, 2017. Accessed November 12, 2018.
2. Ross DS. Overview of the clinical manifestations of hyperthyroidism in adults. https://www.uptodate.com/. Updated March 2018. Accessed November 12, 2018.
3. Ross DS. Graves' hyperthyroidism in nonpregnant adults: overview of treatment. https://www.uptodate.com/. Updated July 2018. Accessed November 12, 2018.
4. Fanning E, Inder WJ, Mackenzie E. Radioiodine treatment for graves' disease: a 10-year Australian cohort study. *BMC Endocr Disord*. 2018; 18(1):94.
5. Stathopoulos P, Gangidi S, Kotrotsos G, Cunliffe D. Graves' disease: a review of surgical indications, management, and complications in a cohort of 59 patients. *Int J Oral Maxillofac Surg*. 2015; 44(6):713-7.
6. Brogan K. *A Mind of Your Own: The Truth About Depression and How Women Can Heal Their Bodies to Reclaim Their Lives*. First edition. HarperCollins Publishers; 2016.
7. Jaccard R. The work of Weston A. Price; diet and dental diseases. *Med Hyg (Geneve)*. 1950; 8(168):149.
8. Arakawa Y, Watanabe M, Inoue N, Sarumaru M, Hidaka Y, Iwatani Y. Association of polymorphisms in DNMT1, DNMT3A, DNMT3B, MTHFR and MTRR genes with global DNA methylation levels and prognosis of autoimmune thyroid disease. *Clin Exp Immunol*. 2012; 170(2):194-201.
9. Almohanna HM, Ahmed AA, Tsatalis JP, Tosti A. The Role of Vitamins and Minerals in Hair Loss: A Review. *Dermatol Ther (Heidelb)*. 2019; 9(1):51-70.
10. Lie C, Liew CF, Oon HH. Alopecia and the metabolic syndrome. *Clin Dermatol*. 2018; 36(1):54-61.
11. Brown K, DeCoffe D, Molcan E, Gibson DL. Diet-induced dysbiosis of the intestinal microbiota and the effects on immunity and disease. *Nutrients*. 2012; 4(8):1095-119.
12. Salzman NH, Bevins CL. Dysbiosis--a consequence of Paneth cell dysfunction. *Semin Immunol*. 2013; 25(5):334-41.
13. Arrieta MC, Bistritz L, Meddings JB. Alterations in intestinal permeability. *Gut*. 2006; 55(10):1512-20.
14. Katz-Agranov N, Zandman-Goddard G. The microbiome and systemic lupus erythematosus. *Immunol Res*. 2017 Apr;65(2):432-437.
15. Cai W, Ran Y, Li Y, Wang B, Zhou L. Intestinal microbiome and permeability in patients with autoimmune hepatitis. *Best Pract Res Clin Gastroenterol*. 2017; 31(6):669-673.
16. Ali AH, Carey EJ, Lindor KD. The Microbiome and Primary Sclerosing Cholangitis. *Semin Liver Dis*. 2016; 36(4):340-348.
17. Kahaly GJ, Frommer L, Schuppan D. Celiac Disease and Glandular Autoimmunity. *Nutrients*. 2018; 10(7). pii: E814.
18. Kim HS, et al. Autoimmune and Allergic Disorders are More Common in People With Celiac Disease or on a Gluten-free Diet in the United States. *J Clin Gastroenterol*. 2018.
19. Wu GD, Chen J, Hoffmann C, et al. Linking long-term dietary patterns with gut microbial enterotypes. *Science*. 2011; 334(6052): 105-8
20. Cani PD, Osto M, Geurts L, Everard A. Involvement of gut microbiota in the development of low-grade inflammation and type 2 diabetes associated with obesity. *Gut Microbes*. 2012; 3(4):279-88.
21. Ahn HY, Chung YJ, Cho BY. Serum 25-hydroxyvitamin D might be an independent prognostic factor for Graves' disease recurrence. *Medicine (Baltimore)*. 2017; 96(31): e7700.
22. Sanna A, Firinu D, Zavattari P, Valera P. Zinc status and autoimmunity: A systematic review and meta-analysis. *Nutrients*. 2018, 10:68.
23. Zheng H, Wei J, Wang L, et al. Effects of Selenium Supplementation on Graves' Disease: A Systematic Review and Meta-Analysis. *Evid Based Complement Alternat Med*. 2018; 2018:3763565.
24. Benvenga S, Lapa D, Cannavò S, Trimarchi F. Successive thyroid storms treated with L-carnitine and low doses of methimazole. *Am J Med*. 2003; 115(5): 417-8.
25. Yates CM, Calder PC, Ed Rainger G. Pharmacology and therapeutics of omega-3 polyunsaturated fatty acids in chronic inflammatory disease. *Pharmacol Ther*. 2014; 141(3): 272-82.
26. Breese McCoy SJ. Coincidence of remission of postpartum Graves' disease and use of omega-3 fatty acid supplements. *Thyroid Res*. 2011; 4(1):16.
27. Camfield DA, Wetherell MA, Scholey AB. The effects of multivitamin supplementation on diurnal cortisol secretion and perceived stress. *Nutrients*. 2013; 5(11):4429-50.
28. Patak P, Willenberg HS, Bornstein SR. Vitamin C is an important cofactor for both adrenal cortex and adrenal medulla. *Endocr Res*. 2004; 30(4):871-5.
29. Padayatty SJ, Doppman JL, Chang R et al. Human adrenal glands secrete vitamin C in response to adrenocorticotrophic hormone. *Am J Clin Nutr*. 2007; 86(1):145-9.
30. Dukowicz AC, Lacy BE, Levine GM. Small intestinal bacterial overgrowth: a comprehensive review. *Gastroenterol Hepatol (N Y)*. 2007; 3(2):112-22.
31. Kawar N, Alrayyes S. Periodontitis in pregnancy: the risk of preterm labor and low birth weight. *Dis Mon*. 2011; 57(4):192-202.
32. Wang YY, Lin XP. Detection and significance of IL-6 and TNF-α in patients with Graves disease and periodontitis. *Shanghai Kou Qiang Yi Xue*. 2018; 27(1):43-47.
33. Gbinigie O, Onakpoya I, Spencer E, McCall MacBain M, Heneghan C. Effect of oil pulling in promoting oro dental hygiene: A systematic review of randomized clinical trials. *Complement Ther Med*. 2016 Jun;26:47-54.
34. Johnson KE, Sanders JJ, Gellin RG, Palesch YY. The effectiveness of a magnetized water oral irrigator (Hydro Floss) on plaque, calculus and gingival health. *J Clin Periodontol*. 1998; 25(4):316-21.
35. Cutovic M, Konstantinovic L, Stankovic Z, Vesovic-Potic V. Structured exercise program improves functional capacity and delays relapse in euthyroid patients with Graves' disease. *Disabil Rehabil*. 2012; 34(18):1511-8.
36. Jindal V, Gupta S, Das R. Molecular mechanisms of meditation. *Mol Neurobiol*. 2013; 48(3):808-11.

37. Chighizola C, Meroni PL. The role of environmental estrogens and autoimmunity. *Autoimmun Rev*. 2012; 11(6-7):A493-501.
38. Duntas LH. Environmental factors and thyroid autoimmunity. *Ann Endocrinol (Paris)*. 2011; 72(2):108-13.
39. Lerner A, Matthias T. Changes in intestinal tight junction permeability associated with industrial food additives explain the rising incidence of autoimmune disease. *Autoimmun Rev*. 2015; 14(6):479-89.