

Adrenal and Thyroid Supplementation Outperforms Nutritional Supplementation and Medications for Autoimmune Thyroiditis

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Abstract

One of the many challenges for any physician is determining the correct course of treatment for patients with more than 1 area of complaint. Should the physician treat the symptoms or the underlying cause of a condition? If treating the cause, what and who determines the cause? Further complicating the issue, doctors must succeed in getting patients to follow the prescribed treatment, which has always been and will continue to be an issue in reaching therapeutic goals.

In late 2009, a 49-year-old Caucasian woman visited the Natural Health Center of Medical Lake (NHCML) in Medical Lake, WA, complaining of multiple symptoms. One symptom was a goiter that had not been relieved with a prescription for 0.375 mg of Synthroid daily. Her comorbidities included mixed hyperlipidemia; multiple joint pains; alopecia; fatigue; bilateral, lower-extremity edema; and severe gastric disruption with bloating and acid reflux.

After initial success from treatment, with a complete reduction of her presenting goiter and most of her other symptoms, the patient withdrew herself from her prescription medication and her nutritional supplementation. After 4 wk, the patient visited

NHCML with indications of severe hypothyroidism, including a severely enlarged goiter of the right wing. After 6 wk of treatment with iodine and a glandular nutritional supplement (GTA Forte), her symptoms of severe hypothyroidism abated. Subsequent treatment for adrenal insufficiency, which was diagnosed at NHCML using salivary adrenal stress-index testing for cortisol rhythm and load, allowed complete resolution of her presenting complaints. This result persisted even at the 3-y follow-up to a greater degree than did the results from the use of thyroid nutritional supplementation and Synthroid, both alone and combined.

The hypothalamus-pituitary-adrenal (HPA) axis may contribute to the existence of thyroid-type symptoms, particularly for those individuals with subclinical thyroid conditions. The treatment of the feedback mechanisms for the HPA axis may provide a valuable framework for treatment of mixed hyperlipidemia because normalizing or improving thyroid stimulating hormone (TSH) levels can reduce serum cholesterol levels.

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Case History

Health care practitioners should always think of the simplest and most effective ways to help a patient. In September of 2009, a 49-year-old Caucasian female visited the Natural Health Center of Medical Lake (NHCML) in Medical Lake, WA, because her “body felt all out of balance.” At that time, she exercised 5 days per week, using a combination of core strengthening and aerobic activities but had gained 18 kg in 2 years. She had also recently been diagnosed with hypercholesterolemia, which continued to worsen, even with dietary modification to provide a low-fat, low-salt intake.

She indicated that her primary care physician had diagnosed her and that she was suffering from a history of (1) vertigo, (2) hypercholesterolemia, (3) mitral valve prolapse, (4) Hashimoto's thyroiditis, (5) Ehlers-Danlos syndrome (unknown category), (6) heavy menstruation with severe cramping, (7) an asymptomatic bulging L5 disc, (8) gastroesophageal reflux disease, (9) bilateral ankle and hand swelling, (10) multijoint pain—upper and lower extremity and spinal, (11) a ripping sensation with bloating in her midabdominal region, (12) chronic allergies, and (13) a history of goiter for the past year.

Her diagnostic workup by her primary care physician, cardiologist, and an endocrinologist over the previous 2 years had included an (1) ultrasound of her goiter, (2) electrocardiogram (EKG) for her mitral valve prolapse, and (3) multiple blood draws to determine the appropriate dosage for her thyroid and hormone medications.

Upon examination at NHCML, she was afebrile, weighed 78.47 kg, had a blood pressure of 118/77, and had a pulse rate of 67 beats per minute (BPM). The physical examination revealed (1) nontender, bulbous, bilateral thyroid wings; (2) nontender, enlarged submandibular glands; (3) sunken eyes; (4) mild alopecia; (5) mild, bilateral, lower-extremity edema, without pitting; and (6) tender epigastric and hypogastric regions, without rebound tenderness or referral.

Her childhood included a health history of antibiotic use for rheumatic fever, pericarditis, and multiple bladder infections. Her social history included mild, infrequent intake of alcohol and use of tobacco up until 4 years prior to the current visit, with a 14-year history of smoking.

The woman's bowel movements occurred once per day but required the addition of fiber for regularity. Her stools were formed, did not sink to the bottom of the bowl, did not stick to the side of the bowl, and had no apparent melena or mucous. Her current care included chiropractic adjustments with a combination of self-treatment that included sublingual B₁₂ (methylcobalamine), *Curcuma longa* (turmeric), vitamin D₃, *Uncaria tomentosa* (cat's claw), calcium, CoQ₁₀, and a multivitamin. Her prescription medications included 0.375 µg of Synthroid as thyroid supplementation and 10 mg of medroxyprogesterone for her heavy menstruation.

The authors ordered a screening blood analysis that included the following tests: (1) comprehensive metabolic panel (CMP), (2) glomerular filtration rate (GFR), (3) complete blood count (CBC) with differential, (4) amylase, (5) lipid panel, (6) thyroid stimulating hormone (TSH), (7) free triiodothyronine (fT₃), and (8) free thyroxine (fT₄). An ultrasound was recently negative and, therefore, was not ordered.

Pretreatment Analysis and Assessment

Table 1 contains the initial results from the blood lab. The authors' initial concern was that the patient continued to suffer from signs of a thyroid condition while on

Synthroid medication. At that time, the authors hypothesized that she was suffering from an alteration in the hypothalamus-pituitary-adrenal (HPA) axis. Such an alteration could produce the signs and symptoms of a thyroid-like condition due to a chronic inflammatory condition, among many possible factors. This condition could cause cortisol hypersecretion, which would inhibit TSH and T₄ production.^{1,2} In effect, the Synthroid medication could be normalizing the TSH marker, masking the underlying HPA-axis alteration and, thereby, causing her to suffer from the condition of the hypothyroid state.

Gastrointestinal and Thyroid Treatment Plan

Because the immune system relies heavily on gastric motility and detoxification,^{3,4} and because gastric motility is a pathway for chronic inflammatory conditions, treating the gastrointestinal (GI) system was the first step of the treatment regimen. The treatment itself was designed to reduce chronic inflammation in an attempt to reduce the hypersecretion of cortisol from which the woman was suffering. The treatment included (1) 4 capsules per day of Dual-Tox from NuMedica (Tulsa, OK, USA) to optimize the detoxification pathways of the liver; (2) 4 tablets per day, without food, of Intenzyme Forte from Biotics Research Corporation (Rosenberg, TX, USA) to reduce inflammation in the colon; (3) 2 tablets per day, with food, of Intenzyme Forte, to help with digestion via enzymes; (4) 1600 mg per day of acetyl-L-carnitine to stimulate mitochondrial biogenesis of adenosine triphosphate (ATP) that is necessary for normalizing thyroid hormone⁵; (5) 4 tablets per day of ADP (oregano oil) from Biotics to perform a mild dysbiotic treatment and improve gastric function; (6) 6 tablets per day of Thyrophin PMG from Standard Process Inc (Palmyra, WI, USA) for thyroid glandular support; and (7) iodine supplementation at 13 mg per day.

In addition to the supplements, the patient was told to drink 14.8 mL of water daily per 0.45 kg of her body weight (2.54 L) and to (1) follow a diet of 500 calories per day to reduce inflammatory proteins; (2) avoid gluten, dairy, and sugars; and (3) avoid fluorinated/chlorinated/brominated materials due to the halogen interactivity. This directive included ceasing her daily hot-tub use, and using bottled water for her daily teeth cleaning, to reduce the fluoride within her system, which can interfere with normal thyroid function.⁶

Initial Results

At the initial 4-week follow-up, the woman had lost 5.9 kg, going from 78.47 kg to 72.57 kg. Her energy and affect had improved, and she said that she "felt fantastic." A physical examination showed (1) she had little edema in her lower extremities; (2) her submandibular glands were nonpalpable; (3) her right thyroid wing was nonpalpable, with complete goiter resolution; and (4) her left goiter was reduced by approximately 50%.

Table 1. 3-Year Comparative Blood Analysis^a

	Pretreatment ^c	1st Lab ^d	Goiter (+) ^e	02/23/11	06/14/11	10/02/12	Normal Thyroid
Test ^b	09/24/10	11/09/10	01/06/11	02/23/11	06/14/11	10/02/12	01/09/13
Total cholesterol (<200 mg/dL)	217 (h)	171		173	170	186	
LDL (<100 mg/dL)	131 (h)	105 (h)		93	90	94	
HDL (>39 mg/dL)	62	52		59	64	73	
TSH (0.4-5.0 mIU/mL)	0.74	0.14 (l)	>100 (h)	0.06 (l)	0.01 (l)	<.01 (l)	0.48
Free T ₄ (0.7-1.5 ng/dL)	1.2	1.3	0.2 (l)	1.2	0.9	1.1	0.8
Total T ₄ (4.5-12.0 ug/dL)			0.9 (l)	5.2	4.4 (l)	5.8	3.4 (l)
Free T ₃ (2.0-4.4 pg/mL)	3.2		1.7 (l)	4.1	3.5	4.6 (h)	3.2
TAB (<40.0 IU/mL)			46.3 (h)	24.6	28.1	35	
TPO (<35 IU/mL)			>1000 (h)	>1000 (h)	>1000 (h)	1000 (h)	
Glucose (65-99 mg/dL)	94	85	102		97	96	
AST (5-40 U/L)	14	17	29		17	17	
ALT (5-50 U/L)	19	23	34		22	19	
Goiter presence	+	50% of initial	+	-	-	-	-

Adrenal Protocol Initiated January 2012

Patient Noncompliance Leads to Goiter

Abbreviations: l = low level; LDL = low-density lipoprotein; h = high level; HDL = high-density lipoprotein; TSH = thyroid stimulating hormone; T₄ = thyroxine; T₃ = triiodothyronine; TAB = thyroglobulin antibody; TPO = thyroid peroxidase; AST = aspartate aminotransferase; ALT = alanine aminotransferase.

^aThis table includes all pertinent results. Unless otherwise stated, assume normal lab results.

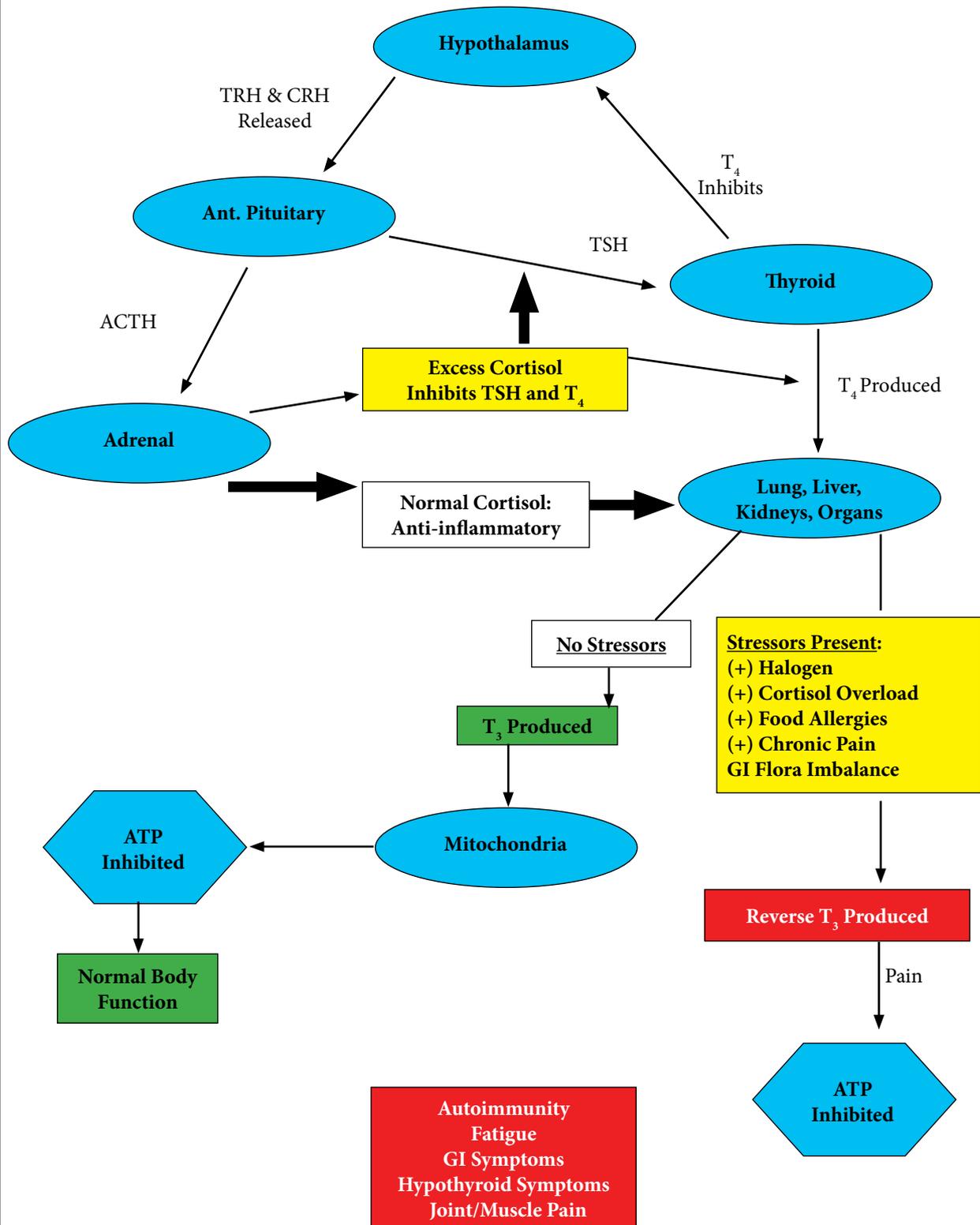
^bInitial amylase was normal at 68 U/L.

^cMedication only.

^dGoiter was absent in December 2010.

^eGoiter is 2-3 × size of initial presentation.

Figure 1. HPA Axis Feedback Mechanism



Abbreviations: HPA = hypothalamus-pituitary-adrenal; TRH = thyrotropin-releasing hormone; CRH = corticotropin-releasing hormone; Ant = anterior; TSH = thyroid stimulating hormone; T₄ = thyroxine; ACTH = adrenocorticotropic hormone; T₃ = triiodothyronine; GI = gastrointestinal; ATP = adenosine triphosphate.

At that point in time, the treatment program was altered to include (1) the addition of 400 µg of selenium to help stabilize her thyroid, (2) an increase in the iodine to 26 mg per day, (3) discontinuation of the oregano oil (ADP), and (4) reduction of Intenzyme Forte to 2 tablets between meals, 2 times per day.

At her 7-week follow-up visit, the patient continued to “feel great” with an increased energy level. Her weight decreased again to 69.85 kg for a total loss of 8.62 kg. The authors’ physical examination revealed normal thyroid size and tenderness bilaterally. The anterior lymph nodes were inflamed, without tenderness, because she had a slight cold. The laboratory values showed a normalization of total cholesterol and a reduction of the LDL compared with her initial results on presentation (Table 1). Her thyroid profile showed a stable fT_4 at 1.3 ng/dL, whereas her TSH was reduced to a low of 0.14 from 0.74 µIU/mL prior to initiation of treatment. Because her TSH had decreased while her fT_4 had remained stable, due to the improvement of the HPA axis, the authors believe that the patient may have been overmedicated when using Synthroid. It was recommended that the woman visit the original prescribing practitioner for a medication assessment.

Table 1 shows the woman’s laboratory results in the 3 years since initiation of treatment.

Adrenal Treatment

As illustrated in Figure 1, cortisol hypersecretion can cause symptoms of a hypothyroid state, due to the feedback mechanisms involved, by reducing peripheral conversion of T_4 to T_3 and by inhibiting TSH stimulation of the thyroid. The problem arises when the feedback mechanism continues to act as if the patient is in a hypothyroid state due to this issue of peripheral conversion, while symptoms of a hypothyroid state continue even with a normalized TSH. In addition to hypothyroid symptomatology, the patient may experience bouts of female irregularity, lowered testosterone levels, and multiple muscle pains due to the conversion of pregnenolone into dehydroepiandrosterone (DHEA). Because DHEA is reduced into estrogens and testosterone, it has wide-ranging effects that could mask male- or female-specific conditions.

After 1 year of treatment with the nutritional supplements, most of the patient’s symptoms had subsided, including multijoint pain, alopecia, GI upset, and cholesterol issues. However, she continued to suffer from mild fatigue and, therefore, an Adrenal Stress Index test from DiagnosTechs (Kent, WA, USA) was performed to determine her salivary cortisol and the effects of potential pregnenolone steal. Pregnenolone steal occurs when cortisol levels are high for prolonged periods of time, with pregnenolone converting immediately to cortisol instead of the intermediary progesterone. See Table 2 for a comprehensive listing of the adrenal stress index test.

Table 2. Comparative Salivary Cortisol Levels^a

	January 2012	January 2013
Cortisol 6:00 AM-8:00 AM (13-24 nM)	26	10.9
Cortisol 11:00 AM-2:00 PM (5-10 nM)	6	3.5
Cortisol 4:00 PM-6:00 PM (3-8 nM)	9	2.7
Cortisol 10:00 PM-midnight (1-4 nM)	8	1.2
Cortisol load (23-42 nM)	49	18.3
DHEA (3-10 ng/mL)	4	1.62
Progesterone (22-100 pg/mL)	78	41.3

Abbreviations: DHEA = dehydroepiandrosterone.

^aCortisol load was reduced significantly in January 2013.

The treatment protocol for the diagnosed adrenal insufficiency included the addition of Drenatrophin PMG (Standard Process Inc), Drenamin (Standard Process Inc) and eleuthero (Standard Process Inc). After 1 year of treatment, the patient’s total cortisol load had reduced from 49 nM to 18.3 nM, indicating a significant reduction in her overall stress load and physiological improvements in immunity, GI health, and adrenal sufficiency due to the reduced cortisol load. After symptomatic relief was obtained and success was noted within the thyroid testing itself, a second adrenal test was performed that showed that the woman had reduced her cortisol levels from hyper to hypo, with an apparent improved regulation of her subsequent thyroid hormones, as indicated in Table 2

As of March 2013, the patient had a complete resolution of her initial presenting complaints, including fatigue, alopecia, goiter, menstruation irregularities, multijoint pain, abdominal bloating, edema in her hands, hypercholesterolemia, and acid reflux. Her dietary regimen has stayed relatively similar throughout the past few years. She has paid attention to avoiding gluten-containing foods, but she does eat dairy occasionally and sugar during celebrations. She maintains her current thyroid status with 2 tablets of GTA Forte, 400 µg of selenium, and 6.75 mg of iodine. Her current adrenal treatment includes 2.4 mg of DHEA 3 times per day, 14 mg of pregnenolone 3 times per day, and 1 Adrenotone capsule from Designs For Health (Suffield, CT, USA) 2 times per day.

Discussion

Because chiropractic physicians and functional medicine specialists tend to focus more on the function of an organ system rather than on apparent pathology, the testing and treatment protocols differ from the traditional medical physician's protocols. For instance, functional practitioners tend to test the fT_4 instead of the total T_4 , because it is the active form of thyroxine and is thought to be a more accurate reflection of the actual thyroid function.⁷ And although the TSH is a valuable tool for initial thyroid screening, many practitioners tend to think of it more as a marker for the HPA axis to determine the health of the feedback loop, which is an important determinant for subclinical hypothyroidism and hyperthyroidism.^{1,2}

Comprehensive testing that includes free levels of thyroid hormones, fT_4 , and fT_3 to evaluate problems with peripheral conversion (T_4 into T_3) should be performed on patients who are suspected of having peripheral or subclinical hypothyroidism. When a thyroid condition is verified, either an overt or a subclinical one, then antithyroperoxidase (TPO) antibodies should be measured to rule in or out an autoimmune thyroid disease (ie, Graves or Hashimoto's disease).^{1,2} Although the value of this test is limited, because much of the treatment is similar, it shows the overall health of the person outside of the immediate autoimmune condition.

When a thyroid condition is suspected, the adrenal function should be measured and appropriately managed to obtain maximum therapeutic effect. The effects of long-term cortisol stimulation on the HPA axis have become apparent, and this stimulation affects widespread organ systems, namely gut motility and health, as well as causes immune-system dysfunction. Although it may not be necessary, and sometimes may be impossible, to normalize the cortisol levels completely, it is important to note the inherent importance of treating the adrenals and thyroid in a combined effort. The reasoning is simple: When the adrenals have been weakened by chronic stress, emotional stress, trauma, or chemical stressors—such as food, the environment, and medications—this stress causes stimulation of cortisol and, over time, reduces TSH and T_4 to T_3 conversion and increases conversion to reverse T_3 . This effect causes the patient to feel even more fatigue and exhaustion.⁸ Treatment may be as simple as reducing the stress factors to reduce the disease progression. Figure 1 shows the feedback mechanisms and potential thought process when treating these types of patients.

Treating functional limitations within the body can be a complex process. Not only do feedback mechanisms come into play, but common stressors such as swimming in a pool (chemical exposures), food allergies, chronic pain, or even medications, such as propylthiouracil that reduces 5 α -deiodinase, which the patient in the current case study was taking, create altered physiology that must be taken into account. The use of natural medicines to help many of the functional processes affected in this way has

been controversial, even with today's knowledge of physiology. For instance, supplementation with nonradioactive inorganic iodine, such as Lugol's Solution, for thyroid conditions has been well-documented and verified since the early 1900s. Controversy arose in 1948 with the Wolff-Chaikoff study, which showed that iodine supplementation may cause a hyperthyroid state in rats. This study theorized that increased iodine ingestion of more than 2 mg per day can cause severe inhibition of oxidation of the iodine in the thyroid gland, leading to a severe hypothyroid state.⁹ Interestingly, this condition is treated with iodine and can be relieved within a 6-week period. Controversy over iodine supplementation occurred because epidemiological studies performed on the Japanese have repeatedly shown that they regularly consume, by some accounts, from 5 to 13.8 mg of elemental iodine on a daily basis.¹⁰ Yet, Japanese citizens suffer few thyroid conditions and have consistently shown a low cancer rate and low fibrocystic breast disease.¹¹ The apparent risk from iodine supplementation appears to come with patients previously treated with radioactive iodine or with those who have undergone thyroid treatment and suffer from autoimmune hyper- and hypothyroidism. The complication arises due to the lack of down-regulation of the sodium-iodine symporter, which is turned off for some individuals who have had prior treatment.¹²

Among the many causes of goiter, iodine deficiency is one of the most common and, therefore, supplementation with iodine should be considered as primary therapy in iodine-deficiency goiter, partly due to iodine's ability to reduce halogen overload.^{13,14} This halogen overload may cause the conversion of T_4 into reverse T_3 instead of T_3 , which then reduces the body's ability to create ATP, thereby creating many symptoms such as fatigue, muscle pain, and a lowered immune-response system (Figure 1).

Conclusion

To treat thyroid patients without the use of medications, a practitioner should look at the simplest forms of treatment and start with the foundation of the current condition being treated, such as the GI system. A normally functioning GI tract usually equates to reduced stress and increased immune response, which is of utmost importance in treating autoimmune patients of any variety. Although normalization of the HPA axis may never be achieved, a greater regulatory response via feedback mechanisms can have a widespread and normalizing effect. Furthermore, as this case study shows, it is possible to treat a person suffering with a multisymptom pathology effectively and safely without the use of prescription medications. In addition, as the case study shows, the treatment of thyroid disorders with medication alone can have less favorable outcomes than with thyroid nutritional supplementation, which in turn can produce less favorable outcomes than combining support for both the thyroid

and adrenal systems. Due to the favorable response in this case study, with complete resolution of the patient's mixed hyperlipidemia, further studies should be performed to determine if treating the HPA axis is a viable treatment for mixed hyperlipidemia and hypercholesterolemia.

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