Chiropractic Management of a Patient With Chronic Fatigue: A Case Report

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Abstract

Objective: The purpose of this case report was to describe the examination and management of a patient with chronic fatigue.
Clinical Features: A 34-year-old woman presented to a chiropractic clinic with complaints of fatigue and inability to lose weight for 2 years. When tested, she was found to have high serum thyroglobulin antibodies, low serum vitamin D_3, low saliva dehydroepiandrosterone-sulfate, and low saliva total and diurnal cortisol.
Intervention and Outcome: The patient was placed on an anti-inflammatory ancestral diet and given recommendations to decrease the aerobic intensity of her exercise routine. On the basis of the result of conventional and functional laboratory tests, she was prescribed a treatment plan of targeted supplementation. After 12 weeks of application of dietary, lifestyle, and supplementation recommendations, the patient reported experiencing increased energy and weight loss of 15 pounds. Her thyroglobulin antibodies returned within reference range, salivary cortisol increased and closely followed the proper circadian rhythm, and dehydroepiandrosterone-sulfate increased.
Conclusions: This report describes improvement in a patient with chronic fatigue with the use of nonpharmaceutical polytherapy involving dietary changes, lifestyle modification, and supplementation. (J Chiropr Med 2016;15:314-320)
Key Indexing Terms: *Adrenal Insufficiency; Hypothalamic Dysfunction Syndromes; Hashimoto Thyroiditis*

Introduction

Fatigue is a common symptom seen in clinical practice; however, it is difficult to define, measure, and specifically relate to the chief complaint within the clinical encounter. Because of its subjective nature, clinicians often ignore fatigue as a symptom and rely on objective findings to steer the diagnosis. Evaluation and management of patients who experience fatigue as their major or only complaint could then be difficult for the clinician.[1]

Fatigue frequently is a major part of the complex pathophysiology of the presenting patient. Fatigue can be described broadly as being either acute and self-limiting or chronic and debilitating.[2] Fatigue is also categorized as being peripheral or central in origin. Peripheral fatigue is caused by peripheral neurotransmitter imbalance and causes impairment in the peripheral nerves and muscular contraction. Central fatigue relates to abnormalities of neurotransmitter balance within the central nervous system and is often present with psychological complaints, such as anxiety and depression.[3] Without proper and ample focus on fatigue as a symptom, the underlying problem may not be identified, and multiple medications, including antidepressants, antipsychotics, and benzodiazepines, could be prescribed. Long-term use of these and other medications could prolong fatigue and affect the patient to the point that chronic fatigue syndrome (CFS) and other chronic conditions could develop.[4]

Chronic fatigue syndrome is characterized by persistent fatigue that may be associated with many other debilitative conditions.[5] Chronic fatigue syndrome is not necessarily caused by exertion and not usually relieved by rest.[6] Common symptoms of CFS include sudden onset of an infectious-type illness, subsequent chronic and debilitating fatigue, pharyngitis, and postexertional malaise.[7] As the cause of CFS is still not known, and its multifaceted mechanism is not understood, effective treatment is difficult.[8] Treatment of CFS conventionally has been restricted to cognitive behavioral therapy and medication.[9] The effectiveness of medications, including antidepressants and immunomodulatory agents, has not been confirmed.[10] There is a growing body of research that supports acquired abnormalities of the hypothalamic-pituitary-adrenal (HPA) axis, including decreased levels of cortisol, enhanced cortisol negative feedback, and blunted HPA axis response in patients with CFS.[11] Reduced activity of the HPA axis and, thus, the hyposecretion of cortisol has been associated with fatigue, although a temporal association has not yet been established.[12]

Chronic fatigue syndrome also has a distinct inflammatory component that can aggravate many of its symptoms and associate it with other endocrine and immunologic disorders that

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are often seen in clinical practice. Chronic fatigue syndrome has been associated with cytokine abnormalities,[12,13] which are very likely indicative of immune activation and pathogenic inflammation. Furthermore, objective fatigue symptom scores in CFS have been significantly correlated with levels of proinflammatory cytokines.[14]

Hypothyroidism is characterized by decreased levels of the thyroid hormone (thyroxine [T_4] and triiodothronine [T_3]) or normal levels of the thyroid hormone but with elevated, and sometimes compensatory, thyroid-stimulating hormone (TSH). Symptoms may include dry skin, poor memory, slow thinking, muscular weakness, muscle cramps, cold intolerance, puffy eyes, constipation, hoarseness, and fatigue. The overall prevalence of hypothyroidism is between 4% and 10% of the general population.[15]

Hashimoto thyroiditis (HT), an autoimmune condition that attacks the thyroid gland by an immune process mediated by cellular T-helper 1 (Th1), commonly results in hypothyroidism. Hashimoto thyroiditis occurs in 0.3-1.5 per 1000 individuals worldwide and is more predominant in females, with gender prevalence ratios of 5–20:1.[16] Symptoms of HT include weight gain, depression, anxiety, sensitivity to cold, and chronic fatigue.[17] Diagnosis for HT is made by testing TSH, free T_4, free T_3, thyroid peroxidase antibodies (anti-TPO), and thyroglobulin antibodies (anti-TG). Diagnosis may be assisted by performing ultrasonography of the thyroid gland, where a mild goiter is sometimes present.[18] Conventional treatment for HT includes life-long replacement of hormone levels, by levothyroxine, triiodothyronine, or desiccated thyroid extract.[19] With regard to nonpharmacologic intervention, a systematic review and a meta-analysis showed an association with routine supplementation of selenium and not only a reduction of anti-TPO but also an improvement in well-being and mood.[20] Immune-endocrine interaction via the HPA axis has also been proven vital for HT, even though its onset and course are insidious. Human studies have demonstrated that physiologic stressors induce various immunologic changes, either directly or indirectly, through the nervous and endocrine systems. These changes in immune function may contribute to autoimmune inflammation in patients who have been already diagnosed or are genetically predisposed to HT. Physiologic stressors, in various forms, can be one of the environmental factors for thyroid autoimmunity, including HT.[21]

There are few cases in the literature describing chiropractic management of chronic fatigue with the use of a polytherapeutic approach. Therefore, the purpose of this case report was to describe the examination and management of a patient with chronic fatigue.

Case Report

A 34-year-old female presented with complaints of chronic fatigue, inability to lose weight, and mood swings. The patient reported to the chiropractic physician that she had been having these symptoms for 2 years. She also complained of bilateral breast tenderness around the time of her menstruation for the last 4 months. The patient described experiencing increased anxiety when driving on bridges over the past 2 years. Her fatigue had worsened over the past 3 weeks, and since then, she had stopped her 6-days-a-week running regimen and her caffeine supplement.

Table 1. *Patient Information Pre- and Post-Treatment*

Patient Information	Initial Visit	12 Weeks Post-Treatment	Reference Range
Weight (lb)	164	149	
BMI	28.1	25.6	18.5–24.9
Vitamin D_3 (pg/mL)	16.8	89.8	18–78 pg/mL
Anti-TG (IU/mL)	225.0	51.0	<116 IU/mL
TSH (mU/mL)	2.645	1.706	0.3–5 mU/L
T_4 free (ng/L)	1.2	1.5	0.8–1.8 ng/dL
T_3 free (pg/mL)	2.9	3.4	2.3–4.2 pg/mL
DHEA-S (ng/mL)	1.92	8.78	2–10 ng/mL
Cortisol – morning (nM/L)	8.8	14.9	13–24 nM/L
Cortisol – noon (nM/L)	4.3	6.2	5–8 nM/L
Cortisol – afternoon (nM/L)	3.1	3.7	4–7 nM/L
Cortisol – nighttime (nM/L)	2.4	2.6	1–3 nM/L
Cortisol sum (nM/L)	18.6	27.4	23–42 nM/L
Cortisol-to-DHEA-S ratio	9.69	3.12	5–6

Anti-TG, thyroglobulin antibodies; *BMI,* body mass index; *DHEA-S,* dehydroepiandrosterone sulfate; *T_3,* triiodothronine; *T_4,* thyroxine; *TSH,* thyroid-stimulating hormone.

A review of her systems included past medical history of trigonocephaly at 8 months of age as well as 3 pregnancies and vaginal deliveries with no miscarriages or abortions. She had had no abnormal Pap test results. The patient had a family history of diabetes, thyroid problems, and seizures. She reported current history of nasal congestion, earaches, voice hoarseness and coughing, snoring, and excessive thirst. She denied being depressed or feeling down. The patient denied taking medications, nutritional supplements, and herbal supplements.

Upon examination, her blood pressure was 106/64 mm Hg, pulse 61 beats per minute, temperature 96.9°F (36.1°C), weight 164 lb (74.4 kg), and height 5 feet 4 inches (162.6 cm). She initially had a body mass index of 28.1. Her general physical examination was unremarkable, with the exception of a left-sided nontender prominence on the thyroid, which was consistent with thyroid nodules and goiter.

Thyroid ultrasonography was ordered on the basis of the physical examination findings, along with a complete blood count, comprehensive metabolic panel, TSH, free T_4, free T_3, reverse T_3, anti-TG, anti-TPO, lipid panel, C-reactive protein, antinuclear antibodies, and serum vitamin D_3 and B_{12}. A saliva adrenal function panel, which included samples of cortisol measured 4 times daily, and dehydroepiandrosterone-sulfate (DHEA-S) measured twice daily, was also ordered because of her complaint of fatigue.

The patient's thyroid ultrasound image was consistent with a multinodular goiter with no dominant cystic or solid