Mitochondrial Support in the Treatment of Chronic Fatigue Syndrome: A Case Report

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Introduction

Chronic Fatigue Syndrome (CFS) is an illness with a primary complaint of persistent or relapsing fatigue along with multiple, non-specific symptoms. These symptoms can include headaches, recurrent sore throats, muscle and joint pains and cognitive complaints. The fatigue can come on suddenly or gradually, but must last for at least six months and is not alleviated by rest. The fatigue results in substantial reduction in previous levels of work, school or personal activities. In 1994 the diagnostic criteria of CFS was established by an international panel for the U.S. Centers for Disease Control. This criteria is based on time and severity of debilitating fatigue and related symptoms.

The International Case Definition of CFS 1994¹

Medically unexplained, persistent, or relapsing chronic fatigue that is of new or definite onset:
• Not due to exertion, not relieved by rest.
• Results in substantial reduction in previous levels of occupational, educational, social, or personal activities.

Concurrent presence of four or more of the following symptoms which must have persisted or recurred for six or more consecutive months of illness and must not have pre-dated the fatigue:
• Impairment in short-term memory or concentration severe enough to cause substantial reduction in previous levels of activity.
• Painful lymph nodes, cervical or axillary.
• Sore throat.
• Muscle pain.
• Pain in more than one joint without redness or swelling.

• Headaches of a new type.
• Unrefreshing sleep.
• Postexertional malaise lasting more than twenty-four hours.

Differential Diagnosis

It is estimated that 25% of all patients visiting a GP complain of prolonged fatigue, but CFS is a very specific diagnosis and only a small percentage of patients meet the criteria.² The diagnosis of CFS should be made by exclusion. As mentioned, many other illnesses bring about chronic fatigue so clinicians must be able to differentially diagnose and therefore consider the following conditions:²

Hypothyroidism
Sleep apnea
Narcolepsy
Chronic Hepatitis
Alcohol/Drug Abuse
Severe Obesity
Iatrogenic
Lupus
Multiple Sclerosis
Cancer
Dementia
Lyme Disease
AIDS
Severe anemia
Pre-existing depression
Psychiatric Illness

Although patients may look healthy, and routine blood tests are usually normal, CFS is a very real illness and many research articles have documented the physiological differences between CFS patients and normal controls. Of relevance to this case is the research which has shown CFS patients to have mitochondrial abnormalities and decreased serum carnitine.³,⁴,⁵

Case

Mr. S., age 31, presented with symptoms of CFS. He reported that the illness

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began with sudden onset at the time of a viral infection six years ago. Since then, the patient has reported that his health and vitality seem to be muddled with mild flu like symptomology such as low grade fever, fatigue, swollen lymph nodes and gastrointestinal upset. The patient clinical profile fits the criteria of the CDC definition of CFS and the most troubling symptoms are those of muscle pain, unrefreshing sleep and postexertional fatigue. Patient reported some previous improvement after self-prescribed essential fatty acids (Evening Primrose Oil, 2 g daily). No other supplements were taken. No outstanding results were found on physical exam and routine blood tests were normal.

After reviewing the medical literature the treatment protocol chosen was based on the fact that CFS patients possessed mitochondrial abnormalities and therefore the treatment mode used addressed this phenomenon. Researchers have observed mitochondrial abnormalities which include degeneration, swelling, vacuolation, myelin figures and secondary lysosomes. Also observed was mild to moderate lipid excess and atrophy of Type II mitochondrial fibers. Researchers have also found that CFS patients have a deficiency of serum acylcarnitine. This is significant because carnitine is important in the transport of fatty acids into the mitochondria for energy by modulating the intramitochondrial CoA:acyl CoA ratio. Researchers further found that acylcarnitine serum concentration increases in CFS patients with recovery from general fatigue.

Follow up research has found that not only are acylcarnitine levels low in CFS patients, but there is also lower serum total carnitine and free carnitine. Higher levels of serum carnitine are related to better overall functioning. Finally, related to the mitochondria, there is recent evidence that the reduced form of nicotinamide adenine dinucleotide (NADH) can be beneficial in the treatment of CFS. The NADH is said to replenish the depleted stores of cellular ATP to improve fatigue and cognitive dysfunction.

Based on this research it was decided to initiate a therapeutic protocol using L-carnitine, NADH and ubiquinone (CoQ10). The addition of CoQ10 is based on its role as an electron carrier in the mitochondrial electron transfer process of respiration and coupled phosphorylation. Anecdotal evidence supports its use in CFS. The mitochondrial supplementation is:

- L-carnitine 500 mg bid with meals
- NADH 2.5 mg tid before meals
- CoQ10 100 mg bid

Results

After six weeks on this protocol, Mr. S. was evaluated regarding the previously mentioned symptoms. Patient reported a marked reduction of symptoms, particularly those related to muscle aches and postexertional fatigue. Mental concentration was also reported to be improved. There were no complaints of adverse reactions to the supplementation and the patient plans to continue with the protocol because it was well tolerated.

Discussion

Chronic Fatigue Syndrome is an illness which is very complex and despite volumes of research, it is still medically unexplained and difficult to manage. There appears to be more controversies over CFS etiology and treatment than there are accepted facts.

The treatment plan was initiated to address one area of research where nutritional support may be of benefit. The results in this case were positive and demonstrate the need for further research into supplementation which can improve mitochondrial activity. Finally, the research of the literature and actual treatment protocol relating to CFS has not been well defined. This particular report of a case can only serve as a reminder to all practitioners that the mosaic symptomatology of CFS must be addressed by a very individual and holistic approach.
References