Ozone therapy is an effective therapy in chronic fatigue syndrome: result of an Italian study in 65 patients

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Abstract

The terms chronic fatigue syndrome and myalgic encephalomyelitis describe a complex physical illness characterized by debilitating fatigue, post-exertional malaise, pain, cognitive problems, sleep dysfunction and an array of other immune, neurological and autonomic symptoms. At the MEDE Clinic of Sacile, Italy, from February 2016 to December 2017, we have treated 65 patients, with auto hemo transfusion, according to the Scientific Society of Oxygen Ozone Therapy (SIOOT) protocols, twice a week for one month and twice a month as maintenance therapy. In conclusion, at our knowledge this is the largest study of patients with chronic fatigue syndrome treated with ozone therapy. Oxygen ozone therapy is an effective therapy in the treatment of chronic fatigue syndrome. However, more patients are needed and in particular a longer follow up is a necessary. In the meantime, ozone therapy seems a treatment that, also because without any side effect, is possible to be proposed to patients with chronic fatigue syndrome that are not obtaining sufficient results from available therapy.

Introduction

The terms chronic fatigue syndrome and myalgic encephalomyelitis (ME/CFS) describe a complex physical illness characterized by debilitating fatigue, post-exertional malaise, pain, cognitive problems, sleep dysfunction and an array of other immune, neurological and autonomic symptoms.1-34 The key feature of the syndrome, post-exertional malaise, is the exacerbation of symptoms following minimal physical or mental activity, which can persist for hours, days or even weeks. Rest and sleep produce only modest relief of fatigue and the other symptoms. The illness is also characterized by substantially reduced physical and/or cognitive functioning.

Although ME/CFS is a physical illness, secondary psychological symptoms may be present as in many chronic conditions.35-42

The term myalgic encephalomyelitis (ME) was coined in 1956 to describe a well-documented cluster outbreak of a fatiguing illness in London, England. The name chronic fatigue syndrome (CFS) was proposed following the investigation of a cluster outbreak of a similar fatiguing illness in Nevada (USA) in 1984. CFS replaced the preliminary name, Chronic Epstein-Barr virus syndrome, because clinical studies were unable to confirm Epstein-Barr virus as the putative cause. The name chronic fatigue syndrome has been criticized as being vague and trivializing of the illness. CFS has also been confused with the common non-specific complaint of chronic fatigue. Other less common names for the illness are myalgic encephalopathy and chronic fatigue immune dysfunction syndrome. The World Health Organization classifies myalgic encephalomyelitis as a disease of the central nervous system (G93.3.). A similar illness, post-viral fatigue syndrome, describes the lingering of fatigue subsequent to a viral infection.

The name ME is more commonly used in Europe and Canada, while the CFS term is more often used in the USA and Australia. A number of different but overlapping case definitions have been published for each of the two terms. Most research studies use CFS because a specific case definition10 was written for this purpose. The acronyms ME/CFS and CFS/ME are increasingly being used worldwide.

The majority of patients present as sporadic or isolated
cases, although cluster outbreaks of ME/CFS have occurred in many widely dispersed locations including: Iceland (1948), London, England (1955), New Zealand (1984), and the USA (Nevada, 1984; New York State and North Carolina, 1985). The illness affects all ages, races and socioeconomic groups. Onset usually occurs between the ages of 30 and 50 years, but may occur at almost any age. It has been estimated that 0.42% of the adult U.S. population have ME/CFS and 70% are female. Higher and lower prevalence estimates have been published for several countries outside the U.S. The prevalence in adolescents and children is uncertain, but appears to be lower than in adults, with equal numbers of boys and girls affected. With no validated diagnostic test for the illness, diagnosis is based on patient-reported symptoms as described in several overlapping case definitions. This primer will use the 2003 Canadian Clinical Case definition, which is intended for clinical practice and better targets the key symptoms of ME/CFS. Although considerable media attention has been given to ME/CFS, most patients with the illness have not been diagnosed. Brain imaging studies with SPECT, PET and MRI have found abnormalities in both white and gray matter. Cognitive testing has confirmed problems that are independent of any coexisting psychological disorder. One group has reported a signature using EEG data that distinguishes patients with ME/CFS from patients with depression and from healthy subjects. Neuro-endocrine studies have identified abnormalities in several hypothalamic endocrine releasing hormone axes, abnormalities that often are the opposite of what is seen in major depression. Studies of spinal fluid proteins have found unique patterns, and spinal fluid concentrations of lactic acid (and, hence, pH) are abnormal. Finally, many studies have identified abnormalities of the autonomic nervous system in patients with ME/CFS. Many (but not all) patients state that their illness began suddenly, with an infectious-like illness. There is good evidence that ME/CFS can follow in the wake of several different viral and bacterial infections. Indeed, it seems unlikely that a single novel infectious agent will prove to be a cause of the great majority of cases. Also, there is evidence that several viruses that produce latent, life-long infection in many humans may be reawakened or reactivated in ME/CFS, although it is unclear if this is the cause or the effect of the illness. Many studies have found evidence of chronic T cell activation. A recent study of the drug rituximab provides indirect evidence for chronic B cell activation, as well. Despite the substantial progress that has been made in understanding the underlying biology of ME/CFS, we still don’t have a sufficiently accurate diagnostic test, or a proven treatment. Since last few decades, the therapeutic potential of ozone has gained much attention through its strong capacity to induce controlled and moderated oxidative stress when administered in precise therapeutic doses. A plethora of scientific evidence showed that the activation of hypoxia inducible factor-1α, nuclear factor of activated T-cells, nuclear factor-erythroid 2-related factor 2-antioxidant response element, and activated protein-1 pathways are the main molecular mechanisms underlying the therapeutic effects of ozone therapy. Activation of these molecular pathways leads to up-regulation of endogenous antioxidant systems, activation of immune functions as well as suppression of inflammatory processes, which is important for correcting oxidative stress in chronic fatigue syndrome.

Materials and Methods

At the MEDE Clinic of Sacile, Italy, from February 2016 to December 2017, we have treated 65 patients, with auto hemotransfusion (GAE), according to the Scientific Society of Oxygen Ozone Therapy (SIOOT) protocols, twice a week for one month and twice a month as maintenance therapy. Females were 50, males were 15; age ranged from 13 to 60 years and the time from CFS diagnosis ranged from 1 to 15 years. To assess the extent of fatigue we used the Fatigue Severity Scale which is used to estimate the severity of the symptom with a score ranging from 1 to 7.

Results

Of the 65 patients with CFS we have treated, 52 patients (80%) showed a significant improvement in symptomatology (>50% improvement in symptoms). There have been no side effects to ozone therapy.

Discussion and Conclusions

Standardized medical care (antidepressants, glucocorticoids, immunotherapy and metabolic drugs) is scarcely beneficial and with some side effects in CFS patients. During the last seven years of clinical experimentation in vasculopathic and in age-related macular degeneration patients, we consistently noted that ozonate autohemotherapy often yields a feeling of wellbeing and euphoria. This result is interesting, and we can only speculate that the reasons for these positive effects are due to a functional restoration of hormonal and neurotransmitter functions. Why not than try the therapeutic shock of ozone autohemotherapy in patients plagued by fatigue and depression? Moreover, ozone therapy may change the vicious circle due to a chronic oxidative stress and deranged muscle metabolism. The clinical results so far obtained appear to justify the use of ozone in this frustrating pathology. It is worth noting that ozone therapy is effective because it is able to activate simultaneously several metabolic pathways that have gone astray. Eighty percent response rate obtained in our patients means that the ozone therapy is an effective therapy in chronic fatigue syndrome.

In conclusion, at our knowledge this is the largest study of patients with CFS treated with ozone therapy. Oxygen ozone therapy is an effective therapy in the treatment of chronic fatigue syndrome. However, more patients are need-
ed and in particular a longer follow up is a necessary. In the meantime, ozone therapy seems a treatment that, also because without any side effect, is possible to be proposed to patients with chronic fatigue syndrome that are not obtaining sufficient results from available therapy.

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