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A Regression of Miller Fisher Syndrome using Photic Feedback: Possibility of a New Complementary Therapy

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We present a case of The Miller Fisher Syndrome (MFS), showing a remission during a recently developed noninvasive therapy. Two weeks after an appearance of cough and fever, a 35 years old Japanese male developed diplopia, ataxia and numbness of his fingers and toes. He was diagnosed as MFS, and a fixed dose of prednisolone acetate (60mg/day) was administered for 3 months, but little improvement was observed. In addition to this administration, we tried 20 minutes of Photic Feedback (PFB) treatment daily for 40 days. The PFB system detects brain waves from the subject's forehead, and extracts alpha waves by the band-pass filter with a center frequency set at 10.0Hz. It also simultaneously modulates the augmentation of a red light-emitting diode, corresponding with the amplitudes of the extracted alpha waves. In this treatment, this adjusted photic stimulation was given to the subject's closed eyes, resulting in the effective alpha enhancement by photic driving response. The numbness increased during each of PFB treatment, but the symptoms started to improve gradually after 10 days. Other symptoms disappeared after 40 days. CD20 levels increased with this treatment. This case suggests that the PFB treatment may speed the natural remission of MFS. This treatment may be worth considering in patients who suffer polyneuropathy.

Key words: Photic Feedback, Miller Fisher syndrome, remission, CD20, humoral immunity, polyneuropathy

INTRODUCTION

Miller Fisher syndrome, first reported by Fisher [1], is known to be a variant of Guillain-Barre syndrome, and is reportedly observed in approximately 5% of patients with Guillain-Barre syndrome. These patients frequently demonstrate high serum anti-GQ1b antibody,

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implicating humoral immunity in the etiology of this disorder. We previously examined changes in alpha rhythm and the levels of natural killer cell activity and lymphocyte subsets during the operation of an electroencephalograph feedback system using photic driving (Photic Feedback). Changes in natural killer cell activity seems to correspond with the standard deviations in alpha wave frequency detected in the right frontal region of the head (Fp_2) [2]. Thus, Photic Feedback may have some effect upon the immune system. Here, we report single case of the use of Photic Feedback as an adjunct therapy for a patient with Miller Fisher syndrome. Pre- and post-treatment natural killer cell activity and lymphocyte subsets are reported.

CASE REPORT

Two weeks after experienced a cough and fever, a 35-year-old Japanese male visited the Department of Neurology in Shimane Prefectural Central Hospital, presenting with diplopia, ataxia and bilateral numbness of his fingers and toes. He was subsequently diagnosed with Miller-Fisher syndrome, and was administered a fixed dose of prednisolone acetate (60mg/day) for 3 months. However, little improvement was observed. Consequently, a 20-minute-per-day Photic Feedback treatment program was introduced for 40 days as an adjunct therapy. Informed consent was obtained from the patient prior to treatment.

The Photic Feedback system [3] detects alpha waves every per second from an electrode on the subject's right frontal region (Fp_2) via a band-pass filter with a center frequency set at 10.0 Hz. It simultaneously modulates the intensity of a red light-emitting diode, corresponding with the amplitudes of the alpha waves. During the treatment in this case, adjusted photic stimulation was presented from approximately 10 cm in front of the subject's closed eyes (5 to 15 lx in illumination), resulting in effective alpha enhancement using the photic driving response.

Initially, the patient reported increased numbness during each 20minute Photic Feedback treatment; however, his symptoms started to improve gradually after 10 days. By 40 days, diplopia, ataxia and numbness had disappeared. CD20 (B1), a lymphocyte subset, levels increased 1.2 times as a result of this treatment. However, no significant changes in CD3, CD4, CD8 and natural killer cell activity (effector-to-target cell ratio, 20:1) were observed. In the seven years following this treatment program, no recurrence of symptoms has been observed in the patient.

DISCUSSION

Miller-Fisher syndrome is characterized by opthalmoplegia, ataxia, and areflexa. A number of studies have reported that sera from patients with Miller-Fisher syndrome demonstrate IgG anti-GQ1b antibodies during the acute phase of illness [4-6]. Some patients develop Miller-Fisher syndrome following infection with C. jejuni. According to Yuki [6], subsequent to the development of C. jejuni enteritis, B cells produce IgG anti-GQ1b antibodies in response to the lipopolysaccharides of C.jejuni, which bear the GQ1b epitope. IgG anti-GQ1b antibodies bind to the oculomotor (III), trochlear (IV), and abducens (VI) nerves and to deep cerebellar nuclei, causing external opthalmoplegia and cerebellar ataxia. CD20 (B1) is known to have an important function in B cell activation, proliferation, and differentation. Furthermore, CD20 reportedly plays a direct role in regulating the transmembrane conductive Ca^{2+} flux of B cells, and Ca^{2+} influx is required for B cell activation [7]. An increase of 1.2 times in CD20 (B1) levels in only a single case study can not be claimed as a significant increase, but this increase in CD20 may possibly be attributable to the Photic Feedback treatment. Natural killer cells play an important role in immune surveillance and/or the prevention of infectious disease. Though ten subjects in a prior study showed significant photic driving responses using Photic Feedback, with a 49% mean increase in alpha amplitude [3], photic stimulation itself is considered to be a stressor for the subject [8]. Thus,

the increase of natural killer cell activity as a result of using Photic Feedback [2] was possibly offset by photic stimulation, resulting in no significant change in this case. Photic Feedback treatment appears to act as a kind of phototherapy [9] and as a method for inducing deep mental relaxation [3]. These effects could be causing some type of immune response [9,10]. Though the details are unclear, a possible hypothesis we considered in this case is that the red light-emitting diode stimulates some immunological regulatory center in the brain, such neural impulses passing along a route including the suprachiasmatic nucleus of the hypothalamus, the pituitary and the pineal gland, probably resulting in the production of neurotransmitters and neuropeptide hormones [11]. Some researchers have demonstrated a favorable clinical response to plasma exchange or human immunogloblin administration in cases of Miller-Fisher syndrome [12,13]. However, these treatments involve the potentially serious risks of anaphylactic reaction or transfer of viral disease. Recently, immunoadosorption therapy, which involves fewer risks than plasma exchange or human immunogloblin administration, has been applied with some success to Miller-Fisher syndrome patients [14]. On the other hand, Yamawaki, et al. [15] reported that immunoadosorption plasmapheresis could be the initial treatment of choice for many neuroimmunological disorders, but subsequent therapies should be carefully considered in prolonged cases. The present case, though highly tentative and speculative in nature, demonstrates that Photic Feedback treatment, which is non-invasive and promotes mental relaxation, may be effective as an adjunct therapy for patients who present with polyneuropathies. A further full-scale clinical study will be necessary to fully determine the benefits of such treatment. Other methods of biofeedback and relaxation therapies should also be considered.

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