Psychogenic Myasthenia Gravis

To the Editor: Myasthenia gravis (MG) is an autoimmune neuromuscular disorder mediated by the production of antibodies targeting different epitopes of the postsynaptic part of the neuromuscular junction, leading to use-dependent muscular weakness.[1] We report a case of functional neurological symptoms mimicking a relapse of MG in a patient who had achieved clinical remission. It has been long proposed that psychogenic neurologic disorders may be due in part to the habituation of idiosyncratic learned, conditioned past response patterns (e.g., psychogenic seizures in an epileptic patient), but this is, to our knowledge, the first report of such behavior mimicking the active phase of the MG illness.

"Mrs. A," a 33-year-old, right-handed, single mother of three, presented with a short history of fatigable ptosis and mild facial and neck weakness. She also suffered with free-floating anxiety symptoms, had an external locus of control, and documented past history of recurrent depression against the background of childhood sexual abuse. A diagnosis of myasthenia gravis was made on the basis of her clinical picture, positive anti-acetylcholine receptor antibodies, and a thymoma detected on CT of the chest, which was revealed in a subsequent thymectomy to be a microinvasive type B3 thymoma (WHO classification). After the operation, she recovered steadily on a combination of prednisolone and pyridostigmine. Clinical remission was achieved, and she returned to work. Shortly afterward, Mrs. A became pregnant again and developed recurrent paroxysmal episodes of hemifacial "weakness/paralysis." The habitual attack consisted of left pseudo-ptosis with associated contraction of orbicularis oculis and oris. Here, the abnormal facial movements appeared to follow a slowly progressive "crescendo–decrescendo" spread to the remaining left hemiface. At its peak, the left lid would completely occlude. Weakness in her neck and left arm with additional dysphonia were also reported during more severe attacks. The attacks lasted between 5 and 40 minutes, and were typically precipitated by low mood, stress, and tiredness. Also, photophobia was described, and, on examination, induction of symptoms was achieved by shining of the light in her left eye, but not in the right eye. The apparent "hemifacial paralysis"' was the result of excessive contraction of orbicularis oris, oculis, and platysma, rather than weakness, and was distractible. Psychological input was initiated (psychodynamic psychotherapy and psychoeducation), and her affective symptomatology successfully treated with the addition of citalopram 40 mg each morning. Although she continued to experience attacks, these considerably decreased in frequency (from several a day to once weekly) and severity, allowing for the gradual discontinuation of steroids without reemergence of other clinical symptoms.

Incidence of trait anxiety, depression, and cognitive impairment have all been found to be increased in MG patients, hypothesized to be related to a central cholinergic deficit.[1] We suggest that clinicians should also be aware of psychogenic/functional symptoms mimicking a relapse or worsening of MG, as another disorder on the spectrum of mental syndromes associated with MG. Interestingly, the abnormal regulation of inhibitory interneuronal mechanisms, where cholinergic modulation is thought to play an important part, has been proposed as a potential pathophysiological mechanism behind certain psychogenic disorders.[2] Hence, one can speculate that this, along with other overt psychological factors, may have led to increased susceptibility to conversion disorder in our patient.

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