Dysfunctional Chemosensation in Myasthenia Gravis: A Systematic Review

Fidias E. Leon-Sarmiento, MD, PhD,†‡ Daniel S. Leon-Ariza, MD,‡¶ and Richard L. Doty, PhD*†

Abstract

Objectives: Myasthenia gravis has traditionally been viewed as a disorder that solely affects the neuromuscular junction within the peripheral nervous system. However, there is now evidence that the cholinergic dysfunction of this disorder may be more widespread than previously believed. This article provides a systematic review of the studies that examined smell and taste function in myasthenia gravis.

Methods: We analyzed studies that reported chemosensory function alterations in patients with myasthenia gravis. Published, MEDLINE, Web of Science, EMBASE, and Scielo, searched to identify articles published from January 1950 through December 2012, were supplemented by relevant articles. The following information was identified from each article: the number of patients, number of controls (if any), clinical stage of patients, neurological involvement, serological state, taste or smell involvement, chemosensory test used, and country of publication.

Results: Ten studies reporting smell and taste function and dysfunction in patients with myasthenia gravis were identified, most of which were case reports commenting on apparent abnormalities in the taste system. The sole empirical study that investigated taste function, however, was negative, suggesting that some reports of taste loss may reflect olfactory loss. One study clearly documented olfactory dysfunction in patients with myasthenia gravis, dysfunction most likely attributable to altered central nervous system cholinergic function.

Conclusions: Chemosensory dysfunction has been reported in a number of patients with myasthenia gravis. Given the close association between complaints of taste dysfunction and loss of flavor sensations secondary to olfactory system damage, quantitative testing should be used to accurately assess the nature and degree of the dysfunction present in this debilitating disorder.

Key Words: myasthenia gravis, smell, taste, Parkinson disease, Alzheimer disease

(j Clin Neurosci 2013;15:1–6)

INTRODUCTION

Myasthenia gravis (MG) was described in 1672 by Thomas Willis.1 MG is commonly viewed by most practitioners as a disorder of the peripheral motor system caused by the dysfunction of cholinergic neural transmission at the neuromuscular junction.2,3 This disorder is characterized by fluctuating weakness that is occasionally difficult to treat. On rare occasions, this disorder can be fatal.2,3 Ocular muscles are frequently involved, indeed, palpebral ptosis is one of the hallmarks of the disease.1 However, the weakness can also involve limb, bulbar, and respiratory muscles.5 The involvement of the skeletal muscles in MG and associated features have allowed for its classification into subtypes based on disease distribution (eg, ocular vs. generalized), age at onset, pregnancy, thymus abnormalities, and autoantibody profiles.5–11 About 80% of patients with MG are positive for the anti-acetylcholine receptor (AChR) antibody; approximately 50% of the anti-AChR-negative antibody group is positive for muscle specific kinase antibodies.5,12,13 Interestingly enough, antibodies against other subunits of the acetylcholine receptor may also trigger myasthenic disorders. Although much is known about the immunological abnormalities present in MG, the breadth of involvement of nonmuscle elements of the disease is still largely unknown.3,4

However, a good number of studies have reported MG-related changes in central