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Myasthenia gravis: Neurochemical and neuroimmunological bases of new therapeutic approach

Eduardo Jahn

Universidad Central de Venezuela, Venezuela

Abstract

It is an autoimmune neuromuscular disorder that produces weakness and tendency to skeletal muscle fatigue; it consists of autoantibodies directed against acetylcholine receptors (AChR) in neuromuscular junctions. Weakness of bulbar innervation muscle groups can occur generating palpebral ptosis, diplopia, dysphagia, dyslalia, dyspnea, among other symptoms that accompany different degrees of generalized weakness. The usual treatment consists of the use of acetylcholinesterase inhibitors (Pyridostigmine), steroids and immunosuppressants, plasmapheresis, gamma globulins and timectomy, all of which can generate multiple complications.

Neurochemical findings, and immunological show low ratio NA / Ad, very high values of plasma free serotonin (5Ht-l) and, an TH2 immunological profile, plus levels of anticolinoceptors that do not have a linear relationship with the intensity of the clinic.

We found that neurochemical findings yield with medication that increases neural but not adrenal norepinephrine by reversing not only the symptoms but also the TH2 profile. However, bulbar symptoms may return or not give very completely.

The nucleus A5 mainly gives the neural sympathetic activity (Na). A5 (NA) works in intense association with the Middle Rafe (MR), which in turn stimulates and regulates the cranial nerve nuclei. Thus, any pharmacological manipulation that favors the activation of MR improves the bulbar symptoms in MG.

In addition, the impact on the general condition of the patient due to pharmacological manipulations that decrease 5Ht-l are dramatic. Not only does 5Ht-l generate TH1 immunosuppression, it interferes with neuromuscular transmission but it also increases bronchial secretions, smooth muscle contraction and vagal activity, facilitating complications.

Through this therapeutic approach aimed at correcting neurochemical and neuroautonomic findings coupled with the experience of recent years, allow us to attend both generalized weakness and bulbar symptoms with a differential and complementary approach and adjusted to the individual therapeutic response. We thus avoid multiple complications given by the usual medication.

Biography

Contrary to the specialized view of medical practice, I consider rather the integration of internal medicine with basic sciences in the area of neuropharmacology and neuroimmunology, which can be pointed out as relevant in my professional practice.

This integration allows a more complex and coherent vision of the way in which the different systems of the body are organized around a biological director little studied in medical schools: The Activating Reticular System (ARS)

The integration of knowledge allows a therapeutic approach aimed at addressing both the consequences of the disease and its causes or aggravating factors within the Autonomic Nervous System and the ARS.

I have participated in the development of new therapeutic designs for various conditions and diseases for two decades. I have contributed to research in international publications in the area of neuropharmacology and neuroimmunology, integrated to the multidisciplinary group in the Institute of Experimental Medicine the UCV.

Publication

• "Pathophysiological and neuropharmacological mechanisms underlying the therapeutical effects of Tianeptine". Lechin F, van der Dijs B. Open Medicine Journal 4: 3-6; 2017.
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