

Clinical features, pathogenesis, and treatment of myasthenia gravis: a supplement to the Guidelines of the German Neurological Society

Nico Melzer¹ · Tobias Ruck¹ · Peter Fuhr² · Ralf Gold³ · Reinhard Hohlfeld⁴ · Alexander Mars⁵ · Arthur Melms⁶ · Björn Tackenberg⁷ · Berthold Schälke⁸ · Christiane Schneider-Gold⁹ · Fritz Zimprich⁹ · Sven G. Meuth¹ · Heinz Wiendl¹

Received: 12 December 2015 / Revised: 20 January 2016 / Accepted: 21 January 2016 / Published online: 17 February 2016
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Abstract Myasthenia gravis (MG) is an autoimmune antibody-mediated disorder of neuromuscular synaptic transmission. The clinical hallmark of MG consists of fluctuating fatigability and weakness affecting ocular, bulbar and (proximal) limb skeletal muscle groups. MG may either occur as an autoimmune disease with distinct immunogenetic characteristics or as a paraneoplastic syndrome associated with tumors of the thymus. Impairment of central thymic and peripheral self-tolerance mechanisms in both cases is thought to favor an autoimmune CD4⁺ T cell-mediated B cell activation and synthesis of pathogenic high-affinity autoantibodies of either the IgG1 and 3 or IgG4 subclass. These autoantibodies bind to the nicotinic acetylcholine receptor (AChR) itself, or muscle-specific tyrosine-kinase (MuSK), lipoprotein receptor-related protein 4 (LRP4) and agrin involved in clustering of AChRs

within the postsynaptic membrane and structural maintenance of the neuromuscular synapse. This results in disturbance of neuromuscular transmission and thus clinical manifestation of the disease. Emphasizing evidence from clinical trials, we provide an updated overview on immunopathogenesis, and derived current and future treatment strategies for MG divided into: (a) symptomatic treatments facilitating neuromuscular transmission, (b) antibody-depleting treatments, and (c) immunotherapeutic treatment strategies.

Keywords Myasthenia gravis · Pathogenesis · Treatment guidelines

Introduction

Myasthenia gravis (MG) is regarded an autoimmune antibody-mediated disorder of neuromuscular synaptic transmission as (a) auto-antibody depositions are detectable at the neuromuscular junction (NMJ) [175, 176]; (b) autoantibodies from MG patients cause MG symptoms when

This manuscript has partially been adopted from the clinical guidelines “Diagnostik und Therapie der Myasthenia gravis und des Lambert-Eaton-Syndroms” of the German Neurological Society (DGN) published by some of the authors [44]. Publication has been permitted by the German Neurological Society.

✉ Nico Melzer
nico.melzer@ukmuenster.de

Heinz Wiendl
heinz.wiendl@ukmuenster.de

¹ Department of Neurology, University of Münster, Albert-Schweitzer-Campus 1, 48149 Münster, Germany

² Department of Neurology, University of Basel, Basel, Switzerland

³ Department of Neurology, University of Bochum, Bochum, Germany

⁴ Institute of Clinical Neuroimmunology, Ludwig-Maximilians-University Munich, Munich, Germany

⁵ Institute of Pathology, University Medical Centre Mannheim, University of Heidelberg, Mannheim, Germany

⁶ Department of Neurology, University of Erlangen, Erlangen, Germany

⁷ Department of Neurology, University of Marburg, Marburg, Germany

⁸ Department of Neurology, University of Regensburg, Regensburg, Germany

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