

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

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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<sup>+</sup> 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.

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web jun 8 2019 figure 1 motor control module note do not ground the mcm this can result in false codes being logged the motor control module mcm incorporates a heat exchanger which uses fuel to cool the mcm the top plate of the heat exchanger incorporates the inlet and outlet fitting for fuel supply entering and exiting the heat

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web the motor control module mcm monitors the rail pressure and when rail pressure deviation is greater than 200 bar for eight seconds the mcm sets the code this fault can occur due to the conditions listed below external fuel leakage between the high pressure pump and fuel injectors pressure limiting valve leakage internal

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