Myasthenia Gravis: Challenges and Therapeutics Solution

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Myasthenia gravis (MG) is an autoimmune disease leading to fluctuating muscle weakness and fatigability and patients have autoantibodies against the acetylcholine receptor. MG is a post-synaptic disorder caused by the antibody-mediated destruction of acetylcholine receptors (AChRs) and blockage of the binding of acetylcholine to acetylcholine receptor. Lambert-Eaton myasthenic syndrome (LEMS) is distinct autoimmune disorders that affect neuromuscular transmission. A study by Jee-Ae Kim et al. showing same clinical features and electrophysiological finding in patients of MG and LEMS [1]. MG includes worsening with repetitive activities and improving with rest. Weakness is worsened by exposure to heat, infection and stress [2]. A wide range of weakness showing in patients in ocular, bulbar, proximal extremities and neck region with 26% mild, 36% moderate and severe in 39% patients [3]. The method like miRNA expression profiles have been studied in peripheral blood mononuclear cells (PBMC) of MG patients and let-7c and miR320 have been down regulated and contribute to MG induction/progression by regulating the expression of some cytokines. miR146a is up regulated in patients and it regulates genes as CD40, CD80, TLR4 and NFkB. Due to miRNA stability in fresh or cryopreserved samples and their easy obtain from blood, they can be easily studied, monitored and used as biomarkers for diagnosis, prognosis or treatment study by Henry Kaminski et al., [4]. miRNA levels in MG patients remained low, regardless of the treatment. Interestingly, most of the patients belonged to the untreated or steroid treated group and steroids did not modify the levels of the studied miRNAs. miRNA-15b and miR20b are two of the miRNAs found at lower levels in MG patients. The genomic region that codes miR15b is commonly affected in more than 50% of the patients with chronic lymphocytic leukemia. This miRNA functions as a tumor suppressor by inhibiting the expression of B-cell lymphoma-2 (BCL2) and plays an important role in controlling B cell homeostasis. The circulating miRNAs provide insights into the pathogenesis of MG since some of these miRNAs have also been found lower in PBMC and have targets with important roles in B cell survival and antibody production. Further studies are needed in larger cohorts of patients will be needed to determine whether these miRNAs could be useful biomarkers in clinical prognosis or response to therapy.

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