The Association of the Anti-GAD Antibodies to the Neurological Conditions

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Abstract

Glutamic acid decarboxylase (GAD) is an enzyme which converts the glutamic acid to the neurotransmitter gamma-aminobutyric acid (GABA). GABA is an inhibitory neurotransmitter that inhibits or weakens the neuronal stimulations. Presynaptic GABAergic neurons in the central neurons system (CNS) and the cells in the islets of Langerhans in the pancreas generate GAD. There are two isoforms of GAD namely GAD-65 and GAD-67. Antibodies against GAD (anti-GAD-Ab) are related to some neurological conditions that these antibodies usually attack the GAD-65 isoform. Neurological conditions such as stiff-person syndrome, epilepsy, limbic encephalitis and cerebellar ataxia are associated to the antibodies against GAD. Furthermore, those antibodies are synthesized in the IDDM (Insulin-Dependent Diabetes Mellitus). There are various mechanisms for the function of these antibodies in different conditions but they mainly function by inhibiting the postsynaptic transmission in Purkinje cells, creating the neuronal dysfunction and reducing the GABA by GAD destruction that these mechanisms demonstrate the relationship between the neurological conditions and the antibodies against GAD.

Keywords: Neurological, Glutamic Acid, Antibodies

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