The 2nd International Neuroinflammation Congress and 2nd Student Festival of Neurosience

Shefa Neuroscience Research Center, Tehran, Iran, 17-19 April, 2018

The Neuroscience Journal of Shefaye Khatam

Volume 6, No. 2, Suppl 1

Poster Presentation

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

Milad Ashrafizadeh[1*], Zahra Ahmadi[2]

[1]Department of Basic Science, Faculty of Veterinary Medicine, Tabriz University, Tabriz, Iran

[2]Department of Basic Science, Faculty of Veterinary Medicine, Shushtar University, Shushtar, Iran

Published: *17 April, 2018*

Abstract

Glutamic acid decarboxylase (GAD) is an enzyme which converts the glutamic acid to the neurotransmitter gamma-amino butyric 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

Corresponding Author: Milad Ashrafizadeh

Email: *Dvm.milad73@yahoo.com*

[Downloaded from shefayekhatam.ir on 2026-11-28]