Hypothesis

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Hypothesis on the Presence of Smooth Muscle Lysis Factor as a Marker In the Diagnosis of Neuroleptic Malignant Syndrome

Hoghoughi Nazanin[1*], Maroufi Azad[2], Hoghoughi Negin[3]

[*1]Department of Psychiatry, Kurdistan University of Medical Sciences, Sanandaj, Iran

[2]Department of Psychiatry, Kurdistan University of Medical Sciences,

Sanandaj, Iran

[3]Department of Cellular and Molecular Biology, Faculty of Biology, Ashrafi Isfahani Univerituy, Isfahan,

Iran

[*]**Corresponding author:** Hoghoughi Nazanin, Department of Economics, Tsinghua Universit, Beijing, China, Tel: 8615651713611; Email: 20165308007@nuist.edu.cn

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Abstract

Dopamine as a neurotransmitter acts to convey messages from one cell to another and it's secretion decrease leads to numerous diseases of motor impairment such as Parkinson disease. In the same way, the use of particular drugs that block the dopamine receptors in the brain cells results in muscle rigidity increase. One of the side effects of these drugs can be Neuroleptic Malignant Syndrome (NMS). NMS is a relatively rare but potentially fatal complication of the use of neuroleptic drugs. Measuring blood CPK (Creatine Phosphokinase) enzyme – the result of the tension and lysis in the body of stripped muscles– can be used as one of the experimental and documentary factors to diagnose and also confirm this disease. The symptoms of NMS -considered as atypical symptoms in scientific articles- can be explained by the changes on the level of Acetylcholine. Acetylcholine functions as a neurotransmitter in the autonomic nervous system, acting in both preganglionic and postganglionic neurons as well as being the final released product of parasympathetic postganglionic neurons. Acetylcholine release from cholinergic interneurons activates muscarinic receptors and has multiple effects that oppose dopamine release and signaling (the dopamine whose function is related to motor actions). This led to the formulation of the hypothesis that the imbalance between dopamine and acetylcholine in the striatum area is critical for maintaining normal motor function. Due to the effect of acetylcholine on the smooth muscles of the body internal organs, this hypothesis focuses on the idea shows that the tension and lysis in the body smooth muscles might produce a factor similar to CPK in the blood.

Key Words:

Dopamine; Neuroleptic Malignant Syndrome; Acetylcholine; smooth muscles; Creatine Phosphokinase

Introduction

Dopamine function as a neurotransmitter in the brain is associated with reward- motivated behaviors, motor control , executive function , motivation, arousal and reinforcement through signaling cascades that are exerted via binding to dopaminergic receptors at the projections found in the substantia nigra, ventral tegmental area, and arcuate nucleus of the hypothalamus (1). This neurotransmitter acts to convey messages from one cell to another and it's secretion decrease leads to numerous diseases of motor impairment such as Parkinson disease. In the same way, the use of particular drugs that block the dopamine receptors in the brain cells results in muscle rigidity increase (2).

One of the side effects of these drugs can be Neuroleptic Malignant Syndrome (NMS). NMS is a relatively rare but potentially fatal complication of the use of neuroleptic drugs (3). Two major theories to explain NMS are a neuroleptic-induced alteration of central neuroregulatory mechanisms and an abnormal reaction of predisposed skeletal muscle (4). Measuring blood CPK (Creatine Phosphokinase) enzyme – the result of the tension and lysis in the body of stripped muscles– can be used as one of the experimental and documentary factors to diagnose and also confirm this disease. The symptoms of NMS like tone and amplitude increase of muscle contractions, the secretory activity of the stomach and intestine through vagus nerve stimulation in the gastrointestinal system, sphincters release and the gallbladder detrusor muscle contraction increase in the urinary system -considered as atypical symptoms in scientific articles- can be explained by the changes on the level of Acetylcholine (5).

Acetylcholine is a chemical neurotransmitter with a wide variety of functions in the brain and other body organs specifically acts as a chemical messenger that is released by neurons and allows them to communicate with one another and other specialized cells such as myocytes and cells found in glandular tissues. While acetylcholine operates as a

neurotransmitter in many parts of the body, it's operation in the neuromuscular junction is much more common. Infact the neuromuscular junction is where motor neurons located in the ventral spinal cord establish a synapse with the body muscles to be activated. Acetylcholine also functions as a neurotransmitter in the autonomic nervous system, acting in both preganglionic and postganglionic neurons as well as being the final released product of parasympathetic postganglionic neurons (6). Acetylcholine release from cholinergic interneurons activates muscarinic receptors and has multiple effects that oppose dopamine release and signaling (the dopamine whose function is related to motor actions) (7). This led to the formulation of the hypothesis that the imbalance between dopamine and acetylcholine in the striatum area is critical for maintaining normal motor function (8). In most of the vascular smooth muscles, acetylcholine depolarizes the membrane and produces a contraction (9).

Due to the effect of acetylcholine on the smooth muscles of the body internal organs, this hypothesis focuses on the idea shows that the tension and lysis in the body smooth muscles might produce a factor similar to CPK in the blood. Determining the validity of this hypothesis could be a valuable clinical sign to distinguish NMS earlier in the process of the disease.

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