칼슘과 파킨슨병

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Calcium and Parkinson's disease

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Despite lots of studies, the exact cause of Parkinson's disease (PD) is poorly understood. Calcium is crucial to normal cell physiology and function of excitable cell. The role of calcium regulation in PD has been much interested as one of the many common pathway of neurodegeneration. Many reports support that mitochondria dysfunction and oxidative stress attributed by environment and genetic mutation are major processes in PD pathology. Maintaining cytoplasmic energy level is impaired and calcium homeostasis is also deteriorated with disease progress in PD. Furthermore, the use of calcium channel blocker reduced in occurrence of PD in retrospective epidemiologic study. The selective vulnerability of dopaminergic neuron in substantia nigra pars compacta, which is autonomous pacemaker by oscillation in calcium concentration, may be associated with Cav1 L-type calcium channel. Accumulation of Alpha-synuclein, which is component of Lewy bodies, also impair cellular and mitochondrial the calcium homeostasis. Collected evidences strongly indicated that impairment of calcium signaling in dopaminergic cell is earliest and major event in pathogenesis in PD. In the future, pharmacological modulation of L-type calcium channel or mitochondrial calcium capacity can be a therapeutic target for treatment of PD.

Key Words: Other primary headache, The International Classification of Headache Disorders 3-beta, Indomethacin

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