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Oral Presentation

Cellular Injury and Various Receptor Expression in the Epileptic Human Amygdala

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Abstract

Mesial temporal lobe epilepsy (MTLE) is the most ordinary type of partial onset epilepsy. Despite several types of treatment, going therapies are insufficient about 20% to 30% of patients. For some, other therapeutic options are need. To achieve this goal, it is essential to develop more precisely the molecular and cellular mechanisms of the disease. This study was planned according to the role of excitatory and inhibitory roles of some important receptors in the amygdala complex as a major part of temporal lobe and effects of these changes on amygdala damage and function. We evaluated amygdala damage by tunnel staining correlate with alteration in GABAA (R α 1, R β 3, and R γ 2), GABACR ρ 2, GAD and GABAB (R1, and R2), also Glutamate receptors NMDA (NR2B, NR1, and mGluR1 α) and AMPA (GluR1, GluR2) immunoreactivity to measure the expression and distribution of these receptors. The present data revealed an increased rate of Dark cells as a hallmark of cell damage as well as apoptotic cells as a marker for cell death, and decreased expression levels of several GABAergic receptor subunits and GAD65 in the amygdala obtained during epilepsy surgery compared to autopsy specimens. Furthermore, the increased occurrence of apoptotic cells in the amygdala was negatively correlated with the reduced expression of the studied GABAergic receptor subunits and GAD65. The present data indicate the importance of GABAergic neurotransmission in seizure-induced cell injury in the amygdala and suggest several GABA receptor subunits as potential candidates for preventive and therapeutic management to control epilepsy and its comorbid disorders, such as anxiety.

Keywords: Human Epileptic Tissue, Amygdala, Temporal Lobe Epilepsy, Excitatory Receptors, Inhibitory Receptors.

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