Abstract

Although the electroconvulsive shock (ECS) is an effective treatment modality in various psychiatric illnesses including depression, mania and schizophrenia, the mechanism of action is unknown. But, one of the obvious facts about clinical effect of ECS is that repeated administrations are necessary in the initiation and maintenance of therapeutic effect in majority of the cases. Among the diverse influences of ECS on post-receptor, intracellular signal transduction system, the most evident one is the expression of immediate early genes (IEGs). So, in order to search the therapeutic mechanism of repeated ECS, authors investigated the expressions of protein molecules involved in the N-methyl-D-aspartate (NMDA) receptor-mediated Ca$^{2+}$ signal transduction, which is one of the mechanisms of IEGs expression.

Rats in the experimental groups were treated once a day with ECT for 1 day, 5 days, 10 days respectively. After rat hippocampi were separated from the control and the experimental groups, the expression of the protein molecules involved in the NMDA receptor-mediated Ca$^{2+}$ signal transduction were analyzed by Western blot method.

The results showed that NMDA receptor subunit 1 and 2A, calmodulin,
calcineurin, CaMK II, CaM IV, CREB increased significantly but phosphorylated CaMK II decreased significantly in the repeated ECS-treated groups.

These results suggest that repeated ECS activates the NMDA receptor-mediated Ca\textsuperscript{2+} signal transduction by increasing the expression of the protein molecules involved in the Ca\textsuperscript{2+} signal transduction system. As the calmodulin and calcineurin were also known to have protective function against overstimulation of NMDA receptor, the repeated ECS is considered not only to facilitate the NMDA receptor-mediated Ca\textsuperscript{2+} signal transduction, but also to inhibit the neurotoxicity from hyperactivity of NMDA receptor

Keywords: Repeated ECS, NMDA receptor, Calmodulin, Calcineurin, Ca\textsuperscript{2+}/calmodulin dependent protein kinase II/IV, cyclic AMP response element

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