Investigation of Relation of FOS Expression and GABA Expression and Their Changes Induced by Focal Cerebral Ischemia In Monkeys

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Research proved that, after cerebral ischemia, the increase of synthesis and release of GABA had directly antagonized and reduced the effect of the accumulation of excitotoxin (Glutamate and aspartate) after ischemia which produced secondary injury to the neurons. To explore the relation between the FOS expression induced by ischemia and the changes of GABA after ischemia, we used in this study a model of focal cerebral infarct induced in Chinese macaque monkeys. Adjacent sections were taken and reacted with direct anti-FOS and GABA immunohistochemical reactions, to see the expressions and changes of FOS and GABA and their relationship. The results were as follows: 2 hours after focal ischemia, the center area of the infarct had a large amount of strong expression FOS immunopositive (FOS-LI) nuclei. At the marginal area of the penumbral zone only a little and scattered medium strong FOS expression was seen. In the same position of the contralateral non-ischemic cortex, no FOS expression was seen. At this time in the central area the immunostaining of the axon terminals of GABAergic neurons were obviously increased. 6 hours after ischemia FOS expression in the central area mostly disappeared, while in the penumbra FOS-LI nuclei were markedly increased. Expressions also increased in intensity, along the central area they form a Band-like distribution. At this time in the central area, dense bead-like GABA positive terminals interwoven with dense GABA fiber network, and parallel GABA terminals intermixed to form string like distribution. In the penumbra, GABAergic neurons had markedly increased in staining. Large amount of positive GABAergic neurons were distributed along the margin of the central foci in the penumbral area, forming a GABA cellular band. It coincided well with the FOS expression band formed by the FOS-LI neurons. In the regions between the ischemic foci and contralateral cortex there were only a few GABA positive neurons distributed scatterly. No GABA positive terminals was seen. After 24 hours ischemia, FOS expression area was seen to slightly expanded towards the non-ischemic area. Adjacent to the ischemic area, the FOS expression diminished. At this time, GABA terminal distribution area also expanded towards outside, and the GABA positive cellular band moved slightly outward, but the amount of cells and the intensity of immunostaining were both weaker than that of 6 hours group. Result of this experiment indicated that: 2-24 hours after focal ischemia in monkey cerebral cortex, the induced FOS expression gradually expanded from ischemic center area to the periphery. FOS expression simultaneously accompanied with increase GABA positive terminals in the central area. In the penumbra, the distribution of GABA positive neurons and the distribution of FOS expressions were very similar, suggesting that FOS expression after ischemia might induce the increase in GABA gene expression, so as to increase the synthesis and release of GABA, and play an protective effect against ischemic neuron injury.