

EFFECT OF VINPOCETIN ON MITOCHONDRIAL COMPLEXES DURING ISCHEMIA/REPERFUSION

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Cerebral ischemia inducing mitochondrial dysfunction plays a key role in generation of reactive oxygen species (ROS). Antioxidants have been shown to be useful to protect neurons from damage induced by ROS during ischemia/reperfusion (I/R) period. Vinpocetine, an effective scavenger of ROS, has been shown to exert antioxidative effect in brain tissue and protects it against I/R insult. In the light of these findings, we investigated whether vinpocetine has any protective effect on mitochondrial enzymes in hippocampus region of rat brain in I/R. Male Wistar rats were randomly divided into 5 groups ($n = 6$ each group). Group1: The sham operated rats received all surgical procedures but without occlusion of bilateral common carotid artery. Group 2: Ischemia was induced by occlusion of bilateral common carotid artery for 30 minutes. After 30 minutes occlusion, reperfusion was allowed. Group 3: Before ischemia was induced, vinpocetine (5mg/kg) was injected for 15 days. Group 4: After ischemia was induced, vinpocetine (5mg/kg) was injected for 15 days. Group 5: 15 days before and after ischemia, vinpocetine (5mg/kg) was injected. The activities of citrate synthase, complex I and complex-IV enzymes were measured spectrophotometrically in homogenate (10%) of hippocampus region. As compared with sham group, citrate synthase activity was decreased in ischemic group but not significant. Citrate synthase activity in group 3, treated with vinpocetine before ischemia induced, was highly increased when compared with all groups. (Group 3 vs. group 1, group 3 vs. group 2, group3 vs. group 4, group 3 vs. group 5, $p < 0.01$, $p < 0.001$, $p < 0.001$, $p < 0.001$, respectively). Although complex I activity was reduced in ischemic group as compared sham group, the difference was not statistically significant. Interestingly, complex I activity in group 3 was significantly reduced as compared with sham group ($p < 0.05$). Comparing with sham group, complex IV activity was reduced in all groups except group 4, treated with vinpocetin after ischemia induced (group 1 vs. group 2, group 1 vs. group 3, group 1 vs. group 5, $p < 0.05$, $p < 0.01$, $p < 0.05$, respectively). Our findings support the hypothesis that I/R might inhibit the activities of the mitochondrial enzymes, especially, complex IV.

Keywords:

mitochondrial complex, hippocampus, ischemia, vinpocetin