Extended Abstract

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The neuroprotective effect of glutamate receptors group II agonists in an animal model of birth asphyxia is connected with inhibition of caspase independent apoptosis

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Problem statement: Hypoxic-ischemic encephalopathy is one of the leading causes of neonatal death and permanent neurological dysfunction worldwide. It has recently been shown that activation of mGluR2/3 before or after ischemic injury leads to neuroprotection, but the exact mechanism of this effect is unclear. Purpose: The purpose of this study is to investigate whether activation of group II glutamate receptor agonists (mGluR2/3) after hypoxia-ischemia can reduce brain damage and inhibit cell apoptosis. Method: We used an animal model of hypoxic-ischemia (HI) in 7-day-old pups. The animals received unilateral carotid artery ligation combined with hypoxia for 75 minutes under 7.4% oxygen. Model the control pups (anaesthetize and dissect the left common carotid artery, but not ligate). 1 hour or 6 hours after HI (5 mg/kg body weight), the animals were injected intraperitoneally with mGluR2 (LY 379268) and mGluR3 (NAAG) agonists. We examined the ischemic cerebral hemisphere weight defects and the expression of non-caspase-dependent apoptotic factors (AIF, HTR/OMI, and Endonuclease G). The expression of nutritional factors GDNF, BDNF and TGFbeta was also measured. Results: Our results showed that regardless of the application time (from 40% of HI to 1520% of treatment), the application of each agonist reduced the weight loss of the ischemic hemisphere brain tissue. Compared with untreated HI, both mGluR2/3 agonists applied 1 hour or 6 hours after HI reduced the expression of AIF, HTR/OMI and endonuclease G protein. Compared with HI, the application of mGluR2/3 agonists reduced the expression of TGFbeta in the ischemic hemisphere and increased BDNF and GDNF. Conclusion: This study proved the neuroprotective effect of mGluR 2/3 agonist on neonatal hypoxic-ischemic brain injury. The data provided indicate that this effect is related