中国药理学与毒理学杂志 2005 19 (2): 87-92 ISSN: 1000-3002 CN: 11-1115/R

论著

一氧化氮合酶抑制剂氨基胍对脑缺血大鼠脑组织氨基酸含量的影响

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收稿日期 2004-9-6 修回日期 网络版发布日期 2008-6-13 接受日期 2005-1-10

摘要 目的 探讨氨基胍对大鼠脑缺血组织的保护作用及其作用机制。方法 采用线栓法复制大鼠中脑动脉梗死模型，缺血后给予氨基胍治疗。相应时间断头取脑，然后测定脑梗死体积、脑组织中氨基酸的含量。结果 脑梗死体积氨基胍组较缺血组明显缩小；缺血组比假手术组纹状体、海马、皮质中天门冬氨酸、谷氨酸、甘氨酸、GABA含量显著增加，给予氨基胍治疗后，天门冬氨酸、谷氨酸的含量明显降低，甘氨酸、GABA含量明显升高。结论 氨基胍降低脑组织中兴奋性氨基酸的含量，升高抑制性氨基酸的含量可能是保护脑缺血的重要机制。

关键词 脑缺血 一氧化氮 一氮化氮合酶 氨基胍 氨基酸

分类号 R972

Effect of nitric oxide synthase inhibitor aminoguanidine on amino acid contents of ischemic brain in rat

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Abstract

AIM To investigate the beneficial effect of aminoguanide (AG) on cerebral ischemic injury and the possible mechanism. **METHODS** The model of focal cerebral ischemia in rat was prepared. Rats were divided into sham-operated group, ischemic group and AG group. Each group was further divided into 3 subgroups (n=6 for each): drugs were administrated at 2, 6 and 12 h after the middle cerebral artery occlusion (MCAO), respectively. AG (100 mg·kg^{-1}, ip) was administrated, 2 times a day, for 3 consecutive days. The changes in infarcted volume and the contents of amino acids were assayed. **RESULTS** The infarcted volume (15.1±3.4, 18.4±5.1, 25.7±3.5) was much decreased compared with that of ischemic group (23.2±2.9, 28.0±3.9,37.2±2.9) when AG was administrated at 2, 6 and 12 h after MCAO respectively (%, P<0.05, n=6). The contents of aspartate, glutamate, glycine and GABA in striatum, hippocampus and cortex in ischemic group were significantly increased compared with sham-operated group(P<0.05 or P<0.01, n=6). The contents of glutamate in striatum, hippocampus and cortex were markedly decreased when AG was given at 2, 6 and 12 h after ischemia respectively(P<0.05 or P<0.01, n=6). The contents of aspartate in striatum, hippocampus and cortex were markedly decreased when AG was given at 2 and 6 h, and the contents of aspartate in hippocampus and cortex were decreased when AG was given at 12 h after ischemia (P<0.05 or P<0.01, n=6). The contents of GABA in hippocampus and cortex were increased when AG was given at 2 and 6 h, and the contents of GABA in striatum and cortex were increased when AG was given at 12 h after ischemia(P<0.05 or P<0.01, n=6). The contents of glycine were increased in striatum, hippocampus and cortex when AG was given at 2 h, the contents of glycine were increased in cortex when AG was given at 6 h, and the contents of glycine in hippocampus and cortex when AG was given at 12 h after ischemia respectively(P<0.05 or P<0.01, n=6). **CONCLUSION** AG has beneficial effect on ischemic cerebral injury. The possible mechanism is that AG can decrease the contents of aspartate and glutamate, increase the contents of glycine and GABA.

Key words ischemia nitric oxide nitric oxide synthase aminoguanidine amino acid

DOI:

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