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基础医学

HMGB1介导急性高血糖对脑缺血大鼠血-脑脊液屏障的损伤

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摘要：

目的 探讨急性高血糖对脑缺血大鼠血-脑脊液屏障（BBB）损伤的作用及机制。方法 实验大鼠随机分为假手术组、正常血糖（NG）组、加甘草酸（GL）（NG+GL）组、高血糖（HG）组和加GL（HG+GL）组。于脑缺血再灌注不同时间段检测脑脊液高迁移率族蛋白B1（HMGB1）含量、BBB通透性、脑水肿和脑梗死体积，评估神经缺失。结果 与NG组比较, HG组大鼠的脑脊液HMGB1含量显著提高(P＜0. 01)；同时，伊文思蓝（EB）外渗率，脑梗死体积及脑水肿显著加重(P＜0. 01)， 神经功能缺陷加重（P＜0. 05）；进行GL干预后，上述指标显著改善(P＜0. 01)。结论 高血糖可促进缺血脑组织HMGB1的释放，加重BBB损伤。抑制HMGB1的活性，对高血糖大鼠脑缺血后BBB的损伤具有保护作用。

关键词： 高迁移率族蛋白B1; 血-脑脊液屏障; 甘草酸; 脑缺血; 大鼠

Acute hyperglycemia worsens ischemic stroke-induced blood-brain barrier impairment via HMGB1 in rats

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Abstract:

Objective To investigate the effects of acute hyperglycemia on ischemia-induced blood-brain barrier (BBB) impairment and the mechanism involved. Methods Wistar rats were randomly divided into sham operation group, normoglycemia (NG) group, hyperglycemia (HG) group and glycyrrhizic acid (GL) intervention (NG+GL, HG+GL)groups. Ischemia was induced by 90 minutes middle cerebral artery occlusion. Western blotting was used to detect the release of high mobility group box 1 (HMGB1) in cerebrospinal fluid, and BBB permeability was evaluated by Evan′s blue (EB) leakage. Brain edema, infarction volume, neurological deficit scores were evaluated after operation. Results Hyperglycemia significantly enhanced the release of HMGB1 in cerebrospinal fluid, compared with the NG group (P<0.01). Meanwhile, Hyperglycemia significantly increased the brain edema and the infarction volume (P<0.01) and worsened the neurological deficit (P<0.05). Inhibition of HMGB1 with GL significantly reduced EB leakage, brain edema, infarction volume, and neurological deficit (P<0.01). Conclusion In transient middle cerebral artery occlusion, the increased release of HMGB1 may contribute to the hyperglycemia-exacerbated BBB damage. Inhibiting HMGB1 may protect the BBB from the hyperglycemia-exacerbated damage.

Keywords: High mobility group box 1; Blood-brain barrier; Glycyrrhizic acid; Cerebral ischemia; Rats

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