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巨刺法对大鼠脑缺血再灌注损伤的神经保护机制 点此下载全文

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摘要：

摘要目的：探寻巨刺法对大鼠脑缺血再灌注损伤的神经保护机制。方法：将80只健康成年的Sprague Dawley大鼠（SD鼠）驯养3d后随机分成假手术组、模型组、非巨刺组及巨刺组，每组各20只，使用线栓法对模型组、巨刺组及非巨刺组大鼠进行脑缺血模型制作，缺血2h后进行再灌注，再灌注3d后将大鼠断头取脑，进行TTC染色及图像软件分析观察脑梗死体积，蛋白印迹法（Western blotting）、荧光PCR检测环磷腺苷反应元件结合蛋白（CREB）的蛋白及基因表达情况，使用酶联吸附试验法（ELISA）检测腺苷酸环化酶（AC）、环磷酸腺苷（cAMP）、蛋白激酶（PKA）的活性。结果：①TTC及其图像软件分析：针刺干预组（包括非巨刺组以及巨刺组）的SD大鼠脑梗死体积明显小于模型组（P＜0.01）；巨刺组脑梗死体积小于非巨刺组（P＜0.05）。②Western blotting：针刺干预组的SD大鼠CREB蛋白水平明显高于模型组（P＜0.01）；巨刺组SD大鼠CREB蛋白水平高于非巨刺组（P＜0.05）。③荧光PCR：针刺干预组的SD大鼠CREB基因水平明显高于模型组（P＜0.01）；巨刺组SD大鼠CREB基因水平高于非巨刺组（P＜0.05）。④ELISA：针刺干预组的SD大鼠AC、环磷酸腺苷（cAMP）、蛋白激酶A（PKA）的活性显著高于模型组（P＜0.01）。结论：巨刺法改善脑缺血再灌注损伤可以通过调节环磷酸腺苷-蛋白激酶A-环磷腺苷反应元件结合蛋白（cAMP-PKA-CREB）信号传导通路实现。

关键词：脑缺血 再灌注损伤 SD大鼠 巨刺法 环磷酸腺苷-蛋白激酶A-环磷腺苷反应元件结合蛋白信号通路

Exploration of neuroprotective mechanism of opposing needling on the ischemia-reperfusion injured rats Download Fulltext

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Fund Project:

Abstract:

Abstract Objective: To explore the neuroprotective mechanism of opposing needling on the ischemia-reperfusion rats. Method: Eighty healthy adult Sprague Dawley (SD) rats (domesticated for 3d) were randomly divided into the shame group, the model group, the non-opposing needling group and the opposing needling group, 20 rats each group. The thread embolism method was used to make MCAO-cerebral ischemia models for the model group, the non-opposing needling and the opposing needling group. The reperfusion took place at 2h after ischemia. Rats were beheaded and the brains were taken off at 3d after reperfusion. TTC staining and image analysis were proceed to observe the cerebral infarction volume, the protein and gene expressions of CREB were detected by Western blotting, real-time PCR and enzyme linked adsorption method (ELISA) were used to test the activities of acethyl choline(AC), cyclic adenosine monophosphate(cAMP), protein kinase A(PKA). Result: ① TTC and the image analysis: The cerebral infarction volume of acupuncture intervention groups (including the non-opposing needling group and the opposing needling group) were significantly smaller than that of the model group (P<0.01); the cerebral infarction volume of the opposing needling group were smaller than that of the non-opposing needling group (P<0.05). ② Western blotting: the protein level of cAMP-response element binding protein（CREB） in the acupuncture intervention groups were obviously higher than that in the model group (P<0.01); The protein level of CREB in the opposing needling group were higher than that in the non-opposing needling group (P<0.05). ③ Real-time PCR: The gene level of CREB in the acupuncture intervention groups were obviously higher than that in the model group (P<0.01); the gene level of CREB in the opposing needling group were higher than that in the non-opposing needling group (P<0.05). ④ ELISA: Compared with the model group, the activities of AC, cAMP, PKA were significantly different in the acupuncture intervention groups (P<0.01), the activities in the acupuncture intervention groups were much higher than that in the model group. Conclusion: The opposing needling improved cerebral ischemia reperfusion injury, it's mechanism might realized by regulating the cAMP - PKA - CREB signaling pathway.

Keywords:stroke reperfusion injury rat opposing needling cAMP - PKA - CREB signaling pathway

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