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低氧预适应对脑缺血-再灌注大鼠神经元凋亡及P53蛋白表达的影响

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摘要：

目的：探讨低氧预适应对局部缺血-再灌注大鼠脑的保护作用及其分子机制。方法：24只大鼠随机分为假手术组、缺血再灌注（I/R）组、低氧预适应（HP＋I／R）组。用线穿法建立大鼠局灶脑缺血-再灌注模型。脑缺血前12h将HP＋I／R组大鼠放在8%低氧舱中完成低氧预适应。分别用免疫组化、原位末端标记法检测P53的表达和神经元凋亡，并进行神经体征观察。结果：缺血3h再灌注24h后HP＋I／R组神经行为缺陷计分明显低于I／R组（P〈0. 05）；HP＋I／R组凋亡细胞数明显少于I／R组（P〈0. 05）；HP＋I/R组P53蛋白阳性细胞数明显低于I／R组（P〈0. 05）。结论：低氧预适应可降低大鼠脑缺血再灌注后的神经功能缺陷和神经元凋亡。下调神经元中P53蛋白表达可能是低氧预适应脑保护作用的分子机制之一。

关键词：脑缺血-再灌注损伤 低氧预适应 凋亡 P53 神经功能缺陷

The effect of hypoxic preconditioning on neuronal apoptosis and expression of P53 protein in rats with focal cerebral ischemia reperfusion injury

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Abstract:

Objective:To explore molecular mechanism of cerebral protection by hypoxic preconditioning in rats model of focal ischemia-reperfusion.Method:Twenty-four SD rats were randomly divided into sham operation group,ischemia/reperfusion(I/R) group and hypoxic preconditioning(HP+I/R) group. The model of focal cerebral ischemia-reperfusion was made in rats by reversible inserting a nylon thread into the middle cerebral artery and hypoxic preconditioning was performed 12 hours before ischemia by placing rats in the hypoxic chamber of 8% oxygen. The neuronal apoptosis,expression of P53 and neurological deficit was respectively evaluated by the immunohistochemistry staining and TUNEL reaction and Zea-Longa method at different time(I1h,I2h,I3h,R1h,R4h,R8h,R24h) after I/R.Result: 24h after reperfusion,the neurological score in the HP+I/R group was markedly lower than that in the I/R group(P<0.05); The number of apoptosis cells in HP+I/R group was obviously fewer than that in the I/R group(P<0.05); The positive cell numbers of P53 immunostaining was significantly decreased as compared with that in the I/R group(P<0.05).Conclusion:Hypoxic preconditioning could decrease the neurological deficit and the neuronal apoptosis of rats with ischemia-reperfusion. Down-regulating the expression of P53 protein of neurons would be the one of the molecular mechanism on cerebral protection of hypoxic preconditioning.

Keywords:ischemia-reperfusion injury hypoxic preconditioning apoptosis P53 neurological deficit

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