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高压氧预适应对大鼠大脑中动脉闭塞后骨桥蛋白表

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Title: Effect of hyperbaric oxygen preconditioning on osteopontin expression in MCAO rats

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摘要: 目的 观察高压氧预适应(hyperbaric oxygen preconditioning, HBOP)对大鼠大脑中动脉闭塞(middle cerebral artery occlusion, MCAO)后的神经保护作用并初步探讨其对骨桥蛋白(osteopontin, OPN)表达的影响。 方法 雄性SD大鼠72只,分为假手术组, HBOP+假手术组, 单纯MCAO组、HBOP+MCAO组, 每组18只。采用大鼠中动脉线栓法建立大鼠MCAO模型。于术后12 h取材, 观察大鼠神经功能和脑梗死变化情况; HE染色光镜下观察缺血区脑组织的病理形态学改变。Western blot检测缺血区OPN的表达变化。 结果 神经功能缺损评分: 术后12 h假手术组及HBOP+假手术组评分均为0分, 单纯MCAO组(3.80±0.79)分, HBOP+MCAO组(2.50±0.97)分; 平衡能力评分: 术后12 h假手术组及HBOP+假手术组大鼠平衡能力评分均为5分, 单纯MCAO组(1.50±1.08)分, HBOP+MCAO组(2.70±2.95)分; 脑梗死率: 术后12 h假手术组及HBOP+假手术组大鼠梗死率均为0, 单纯MCAO组(49.22±7.07)%, HBOP+MCAO组(41.96±3.15)%。可见与单纯MCAO组比较, HBOP+MCAO组在术后12 h神经功能缺损评分明显降低($P<0.05$), 平衡能力评分显著提高($P<0.05$); 脑梗死率明显小于单纯MCAO组($P<0.05$); HE染色见缺血区脑组织病理损伤明显减轻; Western blot检测提示OPN的表达显著增强。 结论 高压氧预适应可以诱导大鼠MCAO后的神经保护作用, 其机制可能与HBOP引起的OPN表达增高有关。

Abstract: Objective To observe the neuroprotective effect of hyperbaric oxygen

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[本期目录/Table of Contents](#)

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preconditioning (HBOP) in rats after middle cerebral artery occlusion (MCAO), and to investigate the effect on osteopontin (OPN) expression. **Methods** Seventy-two Sprague-Dawley rats were randomly and equally divided into 4 groups including a sham group, a HBOP+sham group, a simple MCAO group and a HBOP+MCAO group ($n=18$). The neurological deficits and cerebral infarction volume were detected in the rats at 12 h after permanent MCAO. The pathological changes of ischemic regions were observed under a light microscope by HE staining, and the expression of OPN in ischemic regions was determined by Western blotting. **Results** The scores of neurological deficits at 12 h after MCAO were 0 in the sham and HBOP+sham groups, 3.80 ± 0.79 in the simple MCAO group, and 2.50 ± 0.97 in the HBOP+MCAO group. The scores of balance ability at 12 hours after MCAO were 5 in the sham and HBOP+sham groups, 1.50 ± 1.08 in the simple MCAO group, and 2.70 ± 2.95 in the HBOP+MCAO group. The proportions of cerebral infarction volume at 12 hours after MCAO were 0 in the sham and HBOP+sham groups, 49.22 ± 7.07 in the simple MCAO group, and 41.96 ± 3.15 in the HBOP+MCAO group. Compared with the simple MCAO group, the score of neurological deficits reduced significantly ($P<0.05$), the score of balance ability increased significantly ($P<0.05$), and the proportion of cerebral infarction volume was significantly lower in the HBOP+MCAO group at 12 h after MCAO ($P<0.05$). The pathological injury of brain tissues in ischemic region was significantly relieved, and the expression of OPN was upregulated in the HBOP+MCAO group. **Conclusion** HBOP plays a neuroprotective role in rats after MCAO, and the mechanism may be associated with the upregulation of OPN.

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