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论著

sCR1-SCR15-18蛋白减轻补体介导的大鼠脑缺血/再灌注损伤

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摘要 目的: 探讨补体在大鼠大脑缺血/再灌注(ischemia-reperfusion，I/R)损伤中的作用及重组人可溶性补体受体Ⅰ型SCR15-18蛋白(sCR1-SCR15-18)的保护作用。方法: 75只雄性SD大鼠，随机分为假手术组、I/R组和sCR1 -SCR15-18保护组。采用线栓法建立大鼠大脑中动脉闭塞模型(middle cerebral artery occlusion MCAO)，缺血2 h，再灌注24 h后，进行神经功能学评分，测定脑梗死体积、大脑皮质髓过氧化物酶(myeloperoxidase,MPO)活性，观察大脑皮质区补体C3b沉积和病理改变。结果: 缺血/再灌注24 h后，sCR1-SCR15-18保护组神经功能学评分，脑梗死体积及脑皮质MPO活性明显低于I/R组(P<0.05);sCR1 -SCR15-18保护组缺血脑组织补体C3b沉积明显减少，病理损伤减轻。结论: 补体在脑I/R损伤中起一定作用，sCR1-SCR15-18蛋白对大鼠I/R损伤脑具有保护作用。

关键词 补体 可溶性补体受体1型SCR15-18 脑缺血 再灌注损伤 炎症

分类号 R392

Protective effect of sCR1-SCR15-18 on cerebral ischemia/reperfusion injury in rat via inhibition of complement

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Abstract

<FONT face=Verdana>AIM: To explore the effect of complement on the cerebral ischemia/reperfusion injury in rat and the protection by sCR1-SCR15-18. METHODS: 75 male SD rats were randomly divided into three groups: sham operation group (SO, n=15), middle cerebral artery occlusion and reperfusion (MCAO) without treatment group (I/R, n=30); MCAO treated with sCR1-SCR15-18 group (sCR1-SCR15-18, n=30). After the MCAO for 2 h, then reperfusion for 24 h, the scores of neural behavioral functional deficits were determined. Infarction area was measured by TTC staining. Activity of MPO in cerebral cortex was detected. C3b deposition and pathological change were observed by immunohistochemial staining and HE staining, respectively. RESULTS: After reperfusion for 24 h, the neurological deficits score, infarction area and activity of MPO in sCR1-SCR15-18 group were decreased compared to I/R group. In sCR1-SCR15-18 group, C3b deposition in ischemic area was decreased and pathological injury was improved compared to I/R group. CONCLUSION: Complement plays a role in cerebral ischemia-reperfusion injury and sCR1-SCR15-18 exerts a protective effect by inhibiting the excessive activation of complement.</FONT>

Key words Complement sCR1-SCR15-18 Brain ischemia Reperfusion injury Inflammatory

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