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人参皂苷Rg1对大鼠急性缺血心肌血管再生的促进作

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Title: Ginsenoside-Rg1 promotes angiogenesis in rats with acute myocardial ischemia

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关键词: 心肌缺血; 人参皂甙类; 血管生成; 血管内皮生长因子; 一氧化氮

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摘要: 目的 研究人参皂苷Rg1对急性心肌缺血大鼠心肌血管再生的分子机制。 方法 建立大鼠急性心肌缺血模型，48只大鼠随机抽签法分为急性心肌缺血模型组，阳性药组(美托洛尔4.5 mg/kg)，人参皂苷Rg1 5、10、15 mg/kg组，假手术组，每组各8只。连续腹腔注射14 d后取材。TTC法测定心肌梗死面积，免疫组化染色法测定心肌梗死边缘区微血管密度，心肌梗死边缘区VEGF、VEGFR1、VEGFR2和p-Akt表达，Griess法测定大鼠心肌组织NO水平。 结果 与模型组比较，人参皂苷Rg1 10、15 mg/kg组心肌梗死面积明显减小($P<0.01$)，微血管密度显著增加($P<0.01$)。人参皂苷Rg1 10 mg/kg组VEGF、VEGFR1、VEGFR2及p-Akt表达均为次强阳性，Rg1 15 mg/kg组VEGF、VEGFR1、VEGFR2及p-Akt表达则均为强阳性，结果与阳性药物作用相当，其中以Rg1 15 mg/kg剂量效果最佳。此外，与模型组比较，人参皂苷Rg1 10、15 mg/kg组心肌组织NO水平明显升高 ($P<0.05$, $P<0.01$)。 结论 人参皂苷Rg1可促进大鼠急性缺血心肌血管再生，该作用与人参皂苷Rg1增加心肌组织VEGF、VEGFR、p-Akt以及NO的表达有关。

Abstract: Objective To investigate the role of ginsenoside-Rg1 in the angiogenesis in

rats with acute myocardial ischemia (AMI). Methods The acute ischemia model was established by ligation of the left front descending branch of the coronary artery. The rats were divided randomly into the model group, metoprolol treatment (MET, 4.5 mg/kg) group, ginsenoside-Rg1 treatment groups (5, 10, and 15 mg/kg), 8 rats in each group. Another 8 rats receiving sham operation served as sham operation group. The drugs were administered for 14 d. The myocardial infarction area was measured after 2, 3, 5-triphenyltetrazoliumchloride (TTC) staining. The microvascular density (MVD) was observed by immunohistochemistry staining. The expression of myocardial VEGF, VEGFR1, VEGFR2 and p-Akt was examined by immunohistochemical staining. The myocardial NO levels were examined by Griess assay. Results Compared with the model group, the infarct area was reduced obviously in ginsenoside-Rg1 10 mg/kg group and 15 mg/kg group in AMI rats (P<0.01). And also, ginsenoside-Rg1 of 10 and 15 mg/kg resulted in an obvious increase in MVD of ischemic myocardium (P<0.01). Ginsenoside-Rg1 of 10 mg/kg led to moderate expression of myocardial VEGF, VEGFR1, VEGFR2 and p-Akt. And that of 15 mg/kg led to strong expression of above proteins. These effects were similar to the positive control group. The best effect was found in the ginsenoside-Rg1 15mg/kg group. In addition, ginsenoside-Rg1 of 10 and 15 mg/kg resulted in an obvious increase in the myocardial NO levels compared with the model group (P<0.05, P<0.01). Conclusion Ginsenoside-Rg1 induces angiogenesis in rats after myocardial ischemia. The role of ginsenoside-Rg1 on angiogenesis is correlated with the increased expression of VEGF, VEGFR, p-Akt and NO in the myocardium.

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