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论著

心肌梗死患者血管紧张素转换酶基因多态性与ACE、PAI-1活性的相关性

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摘要 目的:研究心肌梗死(MI)患者血管紧张素转换酶(ACE)基因插入/缺失(I/D)多态性与ACE、PAI-1活性的关系。 方法: 应用PCR方法扩增93例MI患者及87例健康体检者ACE基因特异性片段，同时应用比色法测定血清ACE活性，发色底物法测定PAI-1活性，并对结果进行相关性分析。 结果:①MI组ACE DD基因型频率(32.3%)和D等位基因频率(54.3%)显著高于对照组(12.6%和37.4%)(均P<0.01)。②MI组血清ACE(216.00±58.26)U/L及血浆PAI-1活性(0.85±0.19)AU/mL均显著高于对照组(170.19±48.99)U/L, (0.66±0.20)AU/mL(均P<0.01)；MI组与对照组ACE与PAI-1活性均呈显著正相关(r分别为0.7108，0.7829,均P<0.01)；③MI组DD基因型血清ACE(251.64±57.76)U/L、血浆PAI-1活性(0.96±0.16)AU/mL显著高于ID基因型(211.47±51.87)U/L，(0.82±0.18)AU/mL及Ⅱ基因型(179.84±52.65)U/L，(0.71±0.17)AU/mL(均P<0.01)；ID基因型血清ACE、血浆PAI-1活性亦显著高于Ⅱ型(P<0.05)。对照组DD基因型血清ACE(195.53±54.76)U/L、血浆PAI-1活性(0.78±0.20)AU/mL，显著高于II基因型(154.98±52.74)U/L，(0.59±0.17)AU/mL(均P<0.05)。 结论:由ACE基因所决定的ACE活性，可能参与血浆PAI-1水平的调节；ACE基因I/D多态性与ACE、PAI-1水平相关，ACE基因种类影响纤溶平衡，这可能是其促使MI发病的重要机制之一。

关键词 心肌梗死 血管紧张素转换酶 基因 纤维蛋白溶解

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Relationship between polymorphism of angiotensin I converting enzyme gene insertion/deletion and ACE, PAI-1 activity in patients with myocardial infarction

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Abstract

<FONT face=Verdana>AIM:To explore the relationship between polymorphism of angiotensin I converting enzyme gene insertion/deletion (I/D) and ACE, PAI-1 activity in patients with myocardial infarction (MI).
METHODS:Ninety-three patients with MI and eighty-seven healthy controls were tested. ACE genomic DNA was amplified using the polymerase chain reaction (PCR). Serum ACE activity was measured by colorimetry, plasma level of PAI-1 activity was determined by spectrophotometric assay.
RESULTS:① The frequency of ACE DD genotype and D alleles (32.3% and 54.3%) in MI group was significantly higher than those in control group (12.6% and 37.4%, P<0.01, respectively). ② The ACE activity in serum (216.00±58.26)U/L and plasma PAI-1 activity (0.85±0.19)AU/mL in MI group were significantly higher than those in control group (170.19±48.99)U/L, (0.66±0.20)AU/mL, P<0.01, respectively. The serum ACE activity was positively correlated with plasma PAI-1 activity both in MI group and control group (r=0.7108 and r=0.7829;P<0.01, respectively). ③ In MI group, the serum ACE activity and plasma PAI-1 activity showed a significantly higher level in subjects with DD genotype (251.64±57.76)U/L, (0.96±0.16)AU/mL than those with ID (211.47±51.87)U/L, (0.82±0.18) AU/mL and Ⅱ genotypes (179.84±52.65)U/L, (0.71±0.17)AU/mL. The serum ACE activity and plasma PAI-1 activity were significantly higher in subjects

with ID genotype than those with II genotype (P<0.05). In control group, the serum ACE activity and plasma PAI-1 activity showed a significantly higher level in subjects with DD genotype (195.53±54.76)U/L, (0.78±0.20)AU/mL than the subjects with Ⅱ genotype (154.98±52.74)U/L, (0.59±0.17)AU/mL (P<0.05).
CONCLUSION:The increased ACE activity caused by DD polymorphism may play an important role in elevating the level of plasma PAI-1. The DD genotype of ACE is associated with high PAI-1 level. The genetic variation of ACE contributes to the balance of fibrinolytic pathway, indicating the pathogenesis mechanisms linking to the ACE I/D genotype and MI.</FONT>

Key words Myocardial infarction Angiotensin converting enzyme Genes Fibrinolysis

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通讯作者