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论著

血管紧张素Ⅱ刺激心衰患者外周血单个核细胞分泌TNF-α和NO

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摘要 目的：检测充血性心力衰竭（CHF）患者血清肿瘤坏死因子-α（TNF-α）、一氧化氮（NO）的变化，以及血管紧张素Ⅱ（AngⅡ）、缬沙坦对培养的正常人和CHF患者单核细胞（PBMC）分泌TNF-α、NO的影响,探讨CHF时肾素血管紧张素系统与细胞因子的联系以及缬沙坦治疗CHF的细胞因子机制。方法： 取16例Ⅲ-Ⅳ级的CHF患者静脉血，检测血清TNF-α、NO含量；分离PBMC,置24孔培养板中，分别加入AngⅡ，使其终浓度分别为0、0.01、0.1、1 μmol/L，另外一孔加入0.1 μmol/L的AngⅡ和0.1 μmol/L的缬沙坦，经24 h孵化后，检测培养上清中TNF-α、NO。结果： CHF患者血清TNF-α、NO水平显着高于对照组（P<0.01），心功能Ⅳ级组显著高于心功能Ⅲ级（P<0.01）。不同病因CHF患者之间TNF-α、NO含量无显著差异（P>0.05）。 AngⅡ对正常人和CHF患者PBMC分泌TNF-α、NO均有促进作用，缬沙坦抑制AngⅡ诱导的PBMC分泌TNF-α、NO。结论: AngⅡ促进PBMC产生TNF-α、NO，提示肾素-血管紧张素系统与TNF-α、NO存在一定的联系，缬沙坦可能通过抑制TNF-α、NO的产生在CHF的治疗中发挥作用。

关键词 心力衰竭,充血性； 肿瘤坏死因子； 血管紧张素Ⅱ； 缬沙坦

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Angiotensin Ⅱ stimulates TNF-α and NO production in peripheral blood mononuclear cells in heart failure patients

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Abstract

<FONT face=Verdana>AIM: To examine the change of serum tumor necrosis factor-α (TNF-α), nitric oxide (NO) in patient with congestive heart failure (CHF) and the effect of angiotensin Ⅱ (AngⅡ), valsartan on TNF-α and NO production in culture peripheral blood mononuclear cells (PBMC), to assess the relationship between the renin-angiotensin system and cytokines. METHODS: Venous blood of both healthy volunteers (n=12) and patients with CHF (n=16) were collected. Serum TNF-α and NO were examined. Peripheral blood mononuclear cells (PBMC) were obtained from both the control and the patients groups and cultured with AngⅡ at concentrations of 0, 0.01, 0.1, 1 μmol/L, respectively. AngⅡ at concentration of 0.1 μmol/L combined with 0.1 μmol/L of valsartan was also used. After 24 h incubation, the contents of TNF-α and NO in the culture supernatants were measured. RESULTS: Serum TNF-α and NO production in CHF group were significantly higher than that in control group (P<0.01). The higher the heart failure degree, the higher the levels of TNF-α and NO (P<0.01), and no significant among different etiologies of CHF (P> 0.05) were observed. AngⅡ stimulated TNF-α and NO release from PBMC of patients with CHF and normal person, which was inhibited by valsartan. CONCLUSIONS: AngⅡ obviously increases TNF-α and NO production from PBMC, which indicates there is relationship between the renin-angiotensin system and TNF-α, NO. The fact that valsartan inhibits TNF-α production may be one of the mechanisms in treating CHF.</FONT>

Key words Heart failure congestive Tumor necrosis factor Angiotensin Ⅱ Valsartan

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