Study on the levels of calcitonin gene-related peptide, somatostatin and inflammatory factors in patients with coronary artery disease complicated with type 2 diabetes mellitus

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Abstract

Background

To evaluate the level and correlation of serum neuropeptide cakiNnin gene-elated peptide (CORP), somatostatin (SS) and inflammatory factors (CRP, TNF-o, MCP-1 and sICAM-1) in patients with coronary atherosclerotic heart disease (CAD) complicated with type 2 diabetes mellitus (DM), to explore the mechanism of diabetic patients prone to complicated CAD.

Methods

Patients were divided into three groups according to corcmary angiography results and whether there was a history of type 2 diabetes: control group (no CAD or DM; n 58), CAD group (stable CAD without DM; n 68) and DM+CAD group (stable CAD+DM; n =66). The age, sex ratio and body mass index (BMI) of the three groups were balanced, and the indexes of serum CORP, SS and inflammatory factors (CRP, TNE-a, IL-113, MCP-1 and sICAM-1) were measured by ELASA method. The relationship between serum CORP, SS and inflammatory factors (CRP, TNE-ç,11,-1), MCP-1 and sICAM-1) were analyzed by Spearman correlation analysis, and the risk factors f CAD were analysed by binary logistic regression model.

Results

There were significant differences between neuropeptides (CORP, SS) and inflammatory factors (CRP, TNT', IL-l, MCP-1 and sICAM-1) in the th©© groups. Compared with the control group and the CAD group, CGRP and SS were decreased (P < 0.05), and inflammatory factors were significantly increased (P < 0.05) in the DMTCAD group. CGRP and SS were negatively correlated with inflammatory factors. Logistic regression model showed that CORP, SS, 11-10 and MCP-1 were independent risk factors for CAD (P <0.05).

Conclusion

Compared with the control group and the CAD group, patients in the DMTCAD group had less CGRP and SS but more inflammatory factors. Moreover, the inflammabry factors were negatively correlated with neuropeptides, and neuropeptides and some inflammatory factors are independent risk factors for CAD. This suggests that the TRPV1 injury in the sensory nerve endings and the reduction of neuropeptides release in type 2 diabetic patients may increase the risk of CAD. The mechanism may include that these neuropeptides may inhibit the inflammatory response to some extend

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