Relationship of polymorphism of adhesion molecules *VCAM-1* and *ICAM-1* with preeclampsia.

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Abstract

Objective: To investigate relationship of polymorphism of adhesion molecules *VCAM-1* and *ICAM-1* with preeclampsia.

Method: The present study included 252 patients with PE in our hospital during 2014 February to 2016 December and 200 healthy pregnant women were used as control. Allelic discrimination of the rs5498 polymorphisms of the *ICAM-1* gene and rs3181092 of the *VCAM-1* gene was assessed using the TaqMan assay. Data was analysed using SPSS 18.0.

Results: In PE patients, both ratios of AA and AA+AG genotypes of VCAM-1 were significantly higher than those in the control group, P<0.05. Comparison of genotypes between PE patients of early-onset and late-onset showed that in late-onset PE patients, ratio of AA genotype in *JCAM-1* was significantly higher than that in the early-onset group, P<0.05. Similarly, ratio of genotype AA in severe PE patients was significantly higher than that in mild PE patients, P<0.05. However, in all comparisons, distribution of rs5498 polymorphism of *ICAM-1* showed no significant difference moniferent groups. Conclusion: Rs3181092 polymorphism of *VCAM-1* was associated with occurrence of PE, especially for

Conclusion: R55181092 polymorphism of VCAM-1 was associated with occurrence of PE, especially for late-onset and severe PE patients. However whether r55498 polymorphism of *ICAM-1* was associated with PE needed more investigation.

Keywords: Polymorphism, VCAM-1, ICAM-1, Preeclamps

Introduction

Preeclampsia (PE), a severe complication of pregnancy and one of the leading causes for mortality and merbidity in mothers and fetuses, is characterized by new onset of proteinuria and hypertension after 20 was gestation and affects about 2%~10% pregnant women all over the world [1-3].

It is thought that PE has been associated with insufficient trophoblast invasion of maternal spiral arteries, placental ischemia, impaired placental perfusion, activation of thrombosis, and widespread endothelial cell dysfunction [4-6]. However, the causes for these pathophysiological mechanisms are still unclear. It is considered that genetic factors play an important role in etiology of PE with an estimated heritability of 54% for PE [7].

Studies show that Intercellular Adhesion Molecule-1 (*ICAM-1*), a transmembrane glycoprotein which is expressed in endothelial cells and leukocytes in the immune system, is associated with disorders of female reproductive system such as endometriosis, ovarian stimulation syndrome, and preeclampsia [8,9]. The Vascular Cell Adhesion Molecule-1 (*VCAM-1*), an immunoglobulin like transmembrane protein, is also involved in many inflammatory related diseases [10,11]. It has been proven that polymorphisms in the *VCAM-1* promoter can affect the expression of *VCAM-1* [12]. Studies also showed

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that polymorphisms of *ICAM-1* and *VCAM-1* genes have been implicated as genetic risk factor in several autoimmune diseases [13,14]. However, to our best knowledge, few studies focused on the relationship of polymorphisms of *ICAM-1* and PE, and no study has been reported on polymorphisms of *VCAM-1* in PE patients.

In the present study, we aimed to investigate polymorphism of adhesion molecules *VCAM-1* and *ICAM-1* in preeclampsia and demonstrate relationship of polymorphism of *VCAM-1* and *ICAM-1* with PE. The present study would firstly demonstrate relationship of polymorphism of adhesion molecules *VCAM-1* with preeclampsia, and might provide a new target and better understanding for study and treatment of PE.

Method and Materials

Patients

The present study included 252 patients with PE in our hospital during 2014 February to 2016 December. The diagnosis of PE was confirmed according to clinical findings including increased blood pressure (\geq 140 mmHg systolic or \geq 90 mmHg diastolic on 2 or more measurements at least 6 h apart) and proteinuria \geq 0.3 g/24 h or \geq +1 on a urine dipstick after 20 w of gestation [7]. Patients were further divided into early-onset