15th European Congress of Internal Medicine
2 - 3 September 2016
Connecting with the patient

VEGF 936C/T POLYMORPHISM AND METABOLIC SYNDROME COMPONENTS IN OBESE GREEK INDIVIDUALS

Dimitrios Papazoglou, Zerrin Halil Ibram, Stamatia Pouliliou, Georgios Drosatos, Stefanos Roumeliotis, Konstantinos Zagkas, Nikolaos Papanas, Periklis Panagopoulos, Leonidas Papazoglou, Efstratios Maltezos
University General Hospital of Alexandroupolis, Medical School of Democritus University of Thrace, Greece

Endocrine and metabolic disorders

P.0330

Introduction

Vascular endothelial growth factor (VEGF), a potent angiogenic factor, is a main regulatory protein of endothelial cell proliferation. Increased levels of VEGF have been reported in patients with metabolic syndrome (MetS) and plasma VEGF levels were significantly associated with the components of MetS, such as body mass index, WC, blood pressure and inflammation. A Korean study showed that the −634G>C polymorphism and haplotypes of the *VEGF* gene may be risk factors for MetS susceptibility.

Objectives

The role of VEGF polymorphisms in MetS components susceptibility has not been studied in Caucasian populations. The aim of our study was to assess the potential impact of the 936C/T polymorphism of the *VEGF* gene on the features of the metabolic syndrome in obese subjects.

Methods

One hundred and twenty obese patients (age: 42.3±14.2 yr, BMI: 38.2.2±7.3 kg/m2) with various other features of the MetS and 72 age-adjusted normoweight subjects with none trait of MetS, served as a control group, participated in the study. All obese subjects were consecutively recruited from the Outpatient Clinic of Obesity, Diabetes and Metabolism in the Second Department of Internal Medicine at Democritus University of Thrace.

Genotyping of the 936C/T polymorphism of the VEGF gene was performed by polymerase chain reaction-restriction fragment length polymorphism techniques. The following primers gave a product of 208 bps: forward 5´-AAG GAA GAG GAG ACT CTG CGC AGA GC-3´, reverse 5´-TAA ATG TAT GTA TGT GGG TGG GTG TGT CTA CAG G-3´. The VEGF 936C/T polymorphism was analyzed by digestion of the PCR product with restriction endonuclease NlaIII (New England Biolabs). The 936C allele remained uncut (208 bps), while the 936T was cut into two fragments of 122 and 86 bps.

Fisher exact probability test was used was used to evaluate differences between the studied groups. A stratification analysis was used to assess the MetS risk components.

Results

The results of the genotypic determination were as follows:

Group	CC	CT	TT
MetS (n=120)	87	27	6
Controls (n=72)	54	12	4

The distribution of the 936C/T *VEGF* gene polymorphism of the in both groups was in Hardy-Weinberg equilibrium. There was no significant difference in the genotype (p=0.72) or allelic (p=0.66) frequencies between the metabolic syndrome and control groups.

Furthermore the stratification analysis for the risk components showed no association of the different genotypes of this polymorphism with any of the component traits of MetS in the patient's group.

Conclusions

No significant association was found between the polymorphism studied and the components of the metabolic syndrome that characterise the obese phenotype. These results, suggest that the 936C/T polymorphism of the VEGF gene may not associated with the metabolic consequences of obesity in the Caucasians. Further functional and population studies are required to clarify for a possible association between the *VEGF* polymorphisms and MetS.

References

1. Plasma total antioxidant activity in comparison with plasma NO and VEGF levels in patients with metabolic syndrome. Angiology.2009;60:87–92
2. Association between the polymorphisms of the vascular endothelial growth factor gene and metabolic syndrome, Biomed Rep. 2015; 3(3): 319–326
3. Angiogenesis and biomarkers of cardiovascular risk in adults with metabolic syndrome. J Intern Med. 2010;268:338–347

The study is partly funded under the CARRE EC-FP7 project (grant no 611140) on risk factors for cardiorenal disease and comorbidities.