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Diabetes Medicare Expo 2017: VASPIN – A NOVEL PREDICTOR OF CORONARY ANGIOGRAPHY RESULT IN SCAD (STABLE CORONARY ARTERY DISEASE) PATIENTS

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Vaspin recently discovered adipocytokine. For the first time, it was isolated from the visceral adipose tissue of rats. The hypothesis that vaspin is an insulin sensitizing adipocytokine was proposed in 2005. The researchers later described an association of vaspin with obesity and insulin resistance. From a biochemical point of view, vaspin is a member of the serpin superfamily. It acts both paracrine - inside the visceral adipose tissue and in an endocrine way - mainly affecting the central nervous system and the liver. In the liver, vaspin binds to the GRP 78 chaperone, which has already been incorporated into the cytoplasmic membrane in response to stress from the endoplasmic reticle. Following this translocation of GRP 78, vaspin binds to GRP 78 and exerts beneficial effects on the metabolic dysfunctions induced by stress of the endoplasmic reticle. After intrathecal application, vaspin exerted appetite-reducing effects that lasted 24 hours and subsequently improved insulin sensitivity. This experimental result revealed that vaspin has an effect on the central regulation of appetite in the brain. In addition, there are indications that mutations in the vaspin gene may play a role in a proportion of genetically determined forms of obesity. The role of vaspin has therefore become an important research subject in the cardiometabolic field. In a model of aortic intima lesion in the context of diabetes mellitus, vaspin, by its binding to GRP 78 - a complex of anionic channels dependent on blood pressure, showed an inhibitory potential on calcium-mediated apoptosis and facilitated endothelial repair. Jung et al. have shown a positive impact of vaspin on the endothelial activity of nitric oxide synthase in the endothelium. Based on these experimental results was born the idea to study a connection of vaspin with the degree of coronary artery disease (CAD) lesions in patients suffering from ischemic heart disease. In the study by Kobat et al., Vaspin was significantly lower in patients with CAD, compared to controls. A similarly designed study examining the role of vaspin in patients with unstable angina showed significantly lower plasma concentrations of vaspin in unstable angina. A recent meta-analysis of six studies studying the link between vaspin and obesity confirmed higher concentrations of vaspin in obese and diabetic patients. Based on the results of these studies, it can be assumed that superior vaspin is the associated phenomenon of the protective and compensatory mechanisms triggered in the situation of development of a cardiometabolic disorder. On the other hand, Choi et al. published results, which have demonstrated the existence of an inverse association between vaspin and stenotic damage to the coronary artery represented by the Agatston calcium score. However, this association was found only in the subgroup of women, but not in men. Kadlogou et al. with the cohort of 108 patients with stable coronary artery disease (SCAD) illustrated, that vaspin is an independent determinant of the severity of CAD. Hao et al. recently published article with the results of their own research on the role of vaspin in diabetes mellitus and coronary artery disease. In this study, the levels of vaspin were significantly increased in diabetics compared to healthy individuals and increased further in patients with diabetes and CAD. Based on the results of these studies, we cannot conclude whether the upregulation of vaspin exerts protective effects on the coronary arteries before the development of CAD or whether the action of vaspin is only compensatory. With regard to the role of vaspin in calcium-mediated apoptosis and endothelial repair, it seems reasonable to assume that measuring the plasma concentration of vaspin could be of great benefit when estimating the probability of hemodynamically significant stenosis of the coronary artery (HSCS) in a patient with SCAD. However, no clinically usable criteria on the use of vaspin as a diagnostic marker for HSCS exists today. Additional research is needed to clarify the role of measuring the concentration of vaspin in SCAD.

Study Objectives:

First objective was to verify the existence of independent relation between vaspin and the diagnosis of SCAD. Second objective was to test the relation between vaspin and the pre–test probability (PTP). Third objective was to address the hypothesis that vaspin has the potential to be used as a marker of HSCS in SCAD patients.

Summary:

The roles of vaspin in the pathogenesis of stable coronary artery disease (SCAD) have been repeatedly addressed in clinical studies. In our study, data from 106 SCAD patients who received coronary angiography (CA) and 85 healthy controls were analyzed. Patients were divided into subgroups based on their likelihood of pretest (PTP) and the outcome of the CA. Fasting vaspin was compared between subgroups of SCAD patients and between the target group and controls. The effect of age and smoking on the result of coronary angiography was compared to the effect of vaspin using binomial regression. We did not find any significant difference in the level of vaspin between the target group and the controls. Unless PTP was taken into account, we did not find a difference in vaspin in the target group, when dividing patients based on the presence of significant coronary stenosis. In the subgroup of patients with PTP 15% - 65%, those with strictures had a higher vaspin (0.579 ± 0.898 ng / ml) than patients without significant strictures (0.379 ± 0.732

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ng / ml) t = -2.595 ; p = 0.012; d = 0.658; 1-β = 0.850. Age, smoking and vaspin contributed to the prediction of coronary stenosis in the binomial low PTP regression model (OR: 1.1, 4.9 and 8.7 respectively). According to our results, vaspin cannot be used as a marker independent of the presence of SCAD. Measuring vaspin may be clinically useful in patients with PTP less than 66%. This study was supported by the SRDA grant APVV - 14 - 0153 and by the grant VEGA 1/0160/16.

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