

Coronary Heart Diseases

Tuesday, October 29, 2013, 08:30 AM–09:45 AM

Hall: SARAJEVO

Abstract nos: 158-163

OP-158

The Beta-Fibrinogen 455 G/A Gene Polymorphism Associated with Coronary Artery Disease

Atilla İçli¹, Ahmet Altınbaş², Yasin Türker³, Recep Sütçü⁴, Salaheddin Akçay⁵, Habil Yücel⁶, Özkan Görgülü⁷, Hasan Aydın Baş², Fatih Aksoy², Bayram Ali Uysal²

¹Department of Cardiology, Ahi Evran University Education and Research Hospital, Kırşehir, ²Department of Cardiology, Suleyman Demirel University, Isparta, ³Department of Cardiology, Düzce University, Düzce, ⁴Department of Biochemistry, Katip Celebi University, İzmir, ⁵Department of Cardiology, Celal Bayar University, Manisa, ⁶Department of Cardiology, Isparta State Hospital, Isparta, ⁷Department of Biostatistics and Medikal Informatics, Ahi Evran University Education and Research Hospital, Kırşehir

Background: The genetic model underlying a multifactorial disease such as coronary artery disease (CAD) is complex as it embodies a potpourri of genes and environmental factors. Accordingly, studies have averred that the polymorphisms in the components of renin-angiotensin-aldosterone system are important in the development and progression of CAD. We investigated relationship between β -fibrinogen 455 G/A gene polymorphism and CAD.

Methods: Sixty five patients with CAD (mean age 55 ± 7 years) and 65 patients with normal coronary angiograms (mean age 51 ± 7 years) were included in the study. The types of β -fibrinogen 455 G/A gene polymorphisms were analysed by polymerase chain reaction and restriction fragment length polymorphism. For each polymorphic position, one of three possible patterns may be obtained: Normal (GG) genotype, heterozygous (GA), or homozygous (AA) mutant genotype. Demographic characteristics and major risk factors for atherosclerosis were evaluated in the study groups.

Results: There was no significant difference with respect to age and gender between groups. The frequency of the GA heterozygous genotype was significantly higher in CAD group than controls (37 (%56.9) vs 25 (%38.5), $p=0.035$). The frequency of the AA homozygous mutant genotype was significantly higher in CAD group than controls (8 (%12.3) vs 1 (%1.5), $p=0.016$). Between the two groups were compared according to the dominant genetic model (GA+AA vs. GG). The number of patients carrying at least one A mutant allele (GA+AA) were significantly higher in CAD than controls (45 (%69.2) vs 26 (%40), $p=0.001$). With respect to allelic distribution (G vs A, additive model), the frequency of the A mutant allele was significantly higher in CAD patients. (53 (%40.7) vs 27 (%20.7), $p=0.001$).

Conclusions: In this study, we found that the frequency of β -fibrinogen 455 G/A gene polymorphism was higher in patients with CAD compared to control subject. However, further large-sized studies are required for determining relationship between β -fibrinogen 455 G/A gene polymorphisms and CAD.

General

OP-159

Association between Vitamin D Levels and Cardiac Syndrome X

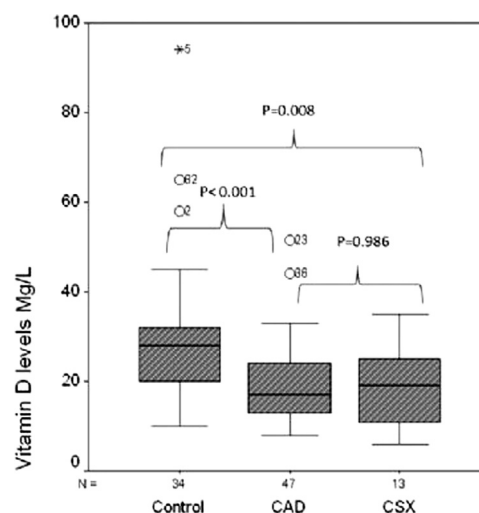
Serpil Eroğlu, Elif Sade, Emre Özçaltık, Gökhan Özyıldız, Uğur Abbas Bal, Kadirhan Akyol, Burcu Ersoy, Haldun Müderrisoğlu
Baskent University, Faculty of Medicine, Department of Cardiology, Ankara

Aim: The effect of Vitamin D on cardiovascular health is controversial. Cardiac syndrome X which is angina-like chest pain, a positive result from a stress test and normal coronary arteries is encountered frequently in cardiology practice. Cardiac syndrome X patients are frequently admitted to hospital. We investigated vitamin D levels in cardiac syndrome X patients.

Methods: In all 95 patients (13 patients with cardiac syndrome X, 47 patients with coronary artery disease and 35 control subjects) were enrolled. 25(OH) D3 levels were measured in all participants in winter season. We compared vitamin D levels between the groups.

Results: Vitamin D levels were different between the 3 groups ($p<0.001$). Vitamin D levels were lower in patients with cardiac syndrome X than in controls (18.8 ± 8.6 $\mu\text{g/L}$ vs. 30.1 ± 16 $\mu\text{g/L}$; $p<0.001$) but vitamin D levels were similar in patients with cardiac syndrome X and coronary artery disease (18.8 ± 8.6 $\mu\text{g/L}$ vs. 19.3 ± 8.9 $\mu\text{g/L}$; P NS) (Figure 1).

Conclusion: Vitamin D levels were reduced in cardiac syndrome X patients similarly to with coronary artery disease. This suggests that vitamin D deficiency could influence coronary flow. Whether correction of Vitamin D deficiency can help to reduce hospital admissions in patients with cardiac syndrome X and it needs to be studied further.



Coronary Heart Diseases

OP-160

The Assessment of Metformin Treatment on Coronary Collateral Development in Patients with Type 2 Diabetes Mellitus

Türkan Seda Tan Kürekli, Aydan Ongun, Hacı Ali Kürekli, Çiğdem Koca, Ali Timuçin Altın, Çağdaş Özdoğru, Çetin Erol
Ankara University School of Medicine, Cardiology Department, Ankara

Background: Metformin is a biguanid class, oral antidiabetic drug used in patients with type 2 Diabetes Mellitus (DM). By increasing insulin sensitivity in tissues, metformin mitigates insulin resistance which is the most important factor causing endothelial dysfunction. It is known that metformin limits the infarct size after myocardial infarction, reduces aneurysm development and remodeling in the ventricle even in the prediabetic period. The current study was undertaken to assess the effects of metformin therapy on coroner collateral development in type 2 diabetes patients.

Material-Methods: Study population consisted of 152 consecutive patients who have undergone coronary angiography and who had at least one major coronary artery stenosis of $\geq 95\%$. Coronary collateral circulations were graded from 0 to 3 according to the Cohen-Rentrop method. And, collateral grading was classified as poor collateral development when the collateral grade was 0 to 1, and as good collateral development when the grade was 2 to 3. The effect of metformin therapy on the coronary collateral growth was analyzed. Furthermore, patients taking metformin were divided into two subgroups as high-dose (>1000 mg) and low-dose (≤ 1000 mg) patients. So, the effect of the dosage of the drug on coronary collateral development was also assessed.

Results: The mean age of the study population was 65.24 ± 9.71 years. 89 of the cases (58,55%) were males, and 63 of them (41,45%) were females. No statistical difference was determined between the groups' demographic and laboratory characteristics. Metformin therapy had no effect on coronary collateral development according to the Rentrop classification ($p=0,657$). Correspondingly, good and poor coronary collateral formations of the groups were similar ($p=0,837$). 31 patients (63,30%) in the high-dose metformin group were identified as having good collaterals, whereas only 7 cases (31,80%) of the low-dose group had good collaterals. In conclusion, patients who have used high-dose metformin had significantly better coronary collaterals ($p=0,014$).

Conclusions: In our study, we have found that metformin therapy had no effect on the development of coronary collaterals in the patients with type 2 DM. In contrast, patients who were on high-dose metformin therapy had significantly better coronary collateral development. To the best of our knowledge, this is the first study evaluating the effects of metformin on coronary collateral formation. Finally, larger, prospective studies are necessary to find out the effects of metformin treatment on coronary collateral development.