A107.E997
JACC March 9, 2010
Volume 55, issue 10A

MYOCARDIAL ISCHEMIA AND INFARCTION

THE IMPACT OF GENETIC VARIABILITY OF FIBRINOGEN A-CHAIN GENE DEFINES FIBRINOGEN LEVELS BETWEEN HEALTHY INDIVIDUALS AND PATIENTS WITH DOCUMENTED ATHEROSCLEROSIS: EFFECTS ON PROTHROMBOTIC PROFILE

ACC Poster Contributions
Georgia World Congress Center, Hall B5
Sunday, March 14, 2010, 3:30 p.m.-4:30 p.m.

Session Title: Novel Biomarkers for Diagnosis and Risk Stratification in ACS
Abstract Category: Stable Ischemic Syndrome
Presentation Number: 1098-263

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Background: The G58A polymorphism on fibrinogen a-chain gene has been associated with fibrinogen levels in healthy individuals, but its effect on patients with coronary artery disease (CAD) is still unknown. In the present study we examined the impact of this polymorphism on fibrinogen levels and prothrombotic profile.

Methods: The study population consisted of 230 subjects, 179 of which angiographically documented for CAD. The G58A polymorphism was detected by Polymerase Chain Reaction (PCR) and appropriate restriction enzymes. Fibrinogen levels were measured by immunonephelometry, while plasma levels of D-dimers, factors V, X, plasminogen were measured by standard coagulometry techniques.

Results: The genotype distribution was GG: 39.6%, AG: 40.2%, AA: 20.2% and GG: 37.3%, AG: 49.0%, AA: 13.7% for CAD patients and healthy individuals respectively. Among the three genotypes there was no significant difference in fibrinogen levels of CAD patients (128.6±32.4 vs 115.8±29.5 vs 127.8±33.4 mg/dl, p=NS for all). Patients with CAD had significantly higher levels of fibrinogen than healthy individuals regarding to the G58A polymorphism (456.3±131.2 vs 385.3±102.0 mg/dl, p<0.001). In addition, there were significant differences fibrinogen levels between the same genotypes of the two populations (CAD vs healthy, AA: 477.5±123.1 vs 386.8±62.7, GG: 452.4±146.3 vs 374.8±114.0, AG: 449.1±119.3 vs 393.6±104.0 mg/dl p<0.05 for all). Similarly, d-dimers levels were significantly higher in the CAD than healthy subjects regarding to the G58A polymorphism (555.8±628.7 vs 360.3±336.7 mg/L). On the contrary, levels of thrombotic markers did not differ significantly between CAD and healthy individuals: fV (124.0±31.8 vs 115.0±25.1 %), fX (94.2±23.2 vs 90.3±18.7%), plasminogen (109.5±19.0 vs 107.7±14.7 u/ml) p=NS for all.

Conclusions: Genetic polymorphism G58A on fibrinogen a-chain gene fails to affect the prothrombotic profile as healthy subjects and CAD patients presented with no differences on specific markers. On the contrary, it turns to be effective on fibrinogen levels implying a potential mechanism which promotes atherosclerosis.