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Prevention

ALTERED PROTHROMBOTIC STATE MEDIATED BY A SINGLE NUCLEOTIDE POLYMORPHISM ON FIBRINOGEN ALPHA CHAIN INDEPENDENTLY OF RISK FACTORS FOR ATHEROSCLEROSIS

ACC Moderated Poster Contributions
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Background: The G58A polymorphism on fibrinogen alpha (a)-chain gene has been associated with increased fibrinogen levels in healthy individuals, but its effect on patients with coronary artery disease (CAD) regarding to the effects on thrombosis remains unknown. In the present study we examined the impact of this polymorphism on fibrinogen and D-dimers levels as well as its interaction with established risk factors in the development of coronary artery disease (CAD).

Methods: The study population consisted of 339 patients with CAD and 260 healthy controls. The G58A polymorphism was detected by polymerase chain reaction and the ACil restriction enzyme. Fibrinogen levels and D-dimers were measured by standard techniques. Risk factors for atherosclerosis were determined according to the biochemical measurements (routinely performed), clinical examination and medical history.

Results: In patients with CAD fibrinogen levels (mg/dl) were not significant higher for 58AA homozygotes vs 58G allele carriers (p=NS). Similar difference occurred in controls (AA: 468.9±17.7 vs GG+GA: 445.4±10.7, p=NS). Importantly, there was a significant difference in D-dimers levels (μg/l) for 58G carriers vs 58AA homozygotes for CAD patients ($p<0.05$), but not for controls (AA: 443.9±52.8 vs GG+GA: 345.8±24.2, p=NS). Although, 58AA homozygotes developed CAD earlier (years) than 58G carriers, this difference was not significant (p=NS). In addition, between 58G carriers and 58AA homozygotes developing CAD, there was no significant difference regarding to hypertension (p=NS), hypercholesterolemia (OR: 1.161, 95%CI (0.497-2.709), p=NS) and DM2 (OR: 1.304, 95%CI (0.599-2.837), p=NS). Similarly, in CAD patients smoking as well as gender (p=NS) did not differ significantly.

Conclusions: Our results showed that the G58A polymorphism on fibrinogen a-chain gene affects D-dimers levels in patients with coronary artery disease. No significant relationship was found between this polymorphism and the risk factors for atherosclerosis. These findings provide a possible mechanism by which this polymorphism may affect thrombotic process/coagulation independently of risk factors for atherosclerosis.