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Role of C677T MTHFR gene polymorphism in coronary artery disease

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Background: Coronary Artery Disease (CAD) is the leading cause of deaths worldwide. Both genetic and environment factors are responsible for CAD. However, these genetic and nongentic factors and the interaction between them need to be further clarified. Methylene tetrahydrofolate reductase (MTHFR) enzyme converts 5, 10-methylene tetrahydrofolate to 5-methyltetrahydrofolate and finally homocysteine to methionine. Individuals having C677T substitution have a reduced enzyme activity and hence higher homocysteine level but lower folate levels than the individuals without this substitution.

Aim: To evaluate the role of C 677T *MTHFR* gene polymorphism in coronary artery disease patients.

Methodology: 50 CAD confirmed patients and normal healthy controls were included in this study. Patients were diagnosed clinically first and then DNA from both CAD patients and healthy controls was extracted using gene aid DNA extraction kit. We employed PCR-RFLP technique to characterize C677T polymorphism in *MTHFR* gene.

Results: Out of 50 CAD patients 31 had CC genotype, 11 had TT genotype and 9 had C/T genotype. No significant association of C677T polymorphism (p=0.33) was found with CAD. Also, there was no statistically significant correlation of C677T polymorphism in MTHFR gene with other clinicopathological parameters like alcoholism, diabetes mellitus, hypertension, smoking, diet, age and gender.

Conclusion: No significant role of C677T polymorphism in *MTHFR* gene was found in the pathogenesis of CAD which may be due to small sample size. We suggest that additional study needs to be done on a larger sample size in order to fully investigate the role of this polymorphism in pathogenesis of CAD.

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