INTRODUCTION

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Homocysteine is a sulfur containing amino acid formed during the metabolism of the essential amino acid methionine. The homocysteine formed is either converted into cysteine via transsulphuration pathway mediated by cystathionine beta synthase (CBS), or it is re-methylated back to methionine by a folate and vitamin B12-dependent enzyme, methionine synthase (MS). MS catalyses the remethylation of homocysteine to methionine by concurrent demethylation of 5-methyltetrahydrofolate to tetrahydrofalte. The production of 5-methyl tetrahydrofolate is mediated by the enzyme 5,10-methylenetetrahydrofolate reductase (MTHFR) ***(Wang et al., 1999).***

Defective activity caused by mutations in these enzymes results in rare inborn errors of metabolism associated with homocysteinuria in which level of homocysteine was greatly increased ***(Mudd et al., 1995).***

In 1969, ***McCully*** reported that in children with hereditary homocysteinuria, a high plasma level of homocysteine gives rise to an appreciable atherosclerotic process. A high level of plasma homocysteine is injurious to the vascular tree by accelerating atherosclerosis and forming a procoagulative state ***(Tawakol et al., 1997).***

In 1976, ***Wilcken and Wilcken*** were first to show the correlation between the level of plasma homocysteine and coronary heart disease.

However several studies around the relationship between high plasma level of homocysteine and the other conventional risk factors of coronary artery disease (CAD) had been carried out. The results of these

studies were controversial. ***Brattstrom et al. (1992)*** showed no relation between Hcy and serum cholestrol level. ***Wu et al. (1994)*** reported that there was a correlation between Hcy levels and LDL-C while no correlation was found between hyperhomocysteinemia and diabetes.

In ***1999 Sharabi et al.*** had found an indication that in hypertensive patients, hyperhomocysteinemia has no additive or synergistic effects on the occurrence of atherothrombotic events.

The relation between smoking and Hcy level in patients with vascular disease has been studied. While ***Glueck et al. (1995)*** showed no correlation, ***Christensen et al. (1999)*** reported that the risk of myocardial infarction (MI) in smokers may at least partly be attributed to hyperhomocysteinemia or low folate.

AIM OF THE WORK

This thesis aims to study the level of plasma homocysteine as a risk factor in coronary artery disease in absence of other conventional risk factors as hyperlipidemia, smoking, diabetes mellitus and hypertension, and its correlation with plasma folic acid level.