INTRODUCTION

Atherosclerosis has been an important cause of cardiovascular morbidity and mortality in recent years ***(Narin et al., 2002).***

Since many patients experience cardiovascular events despite the absence of such classical risk factors as hypertension, high blood lipid levels, diabetes or smoking, the search for underlying causes of vascular disease is continuing ***(Rafflenbeul, 2005).***

Over the last twenty years, homocysteine has taken on increasing importance as an independent risk factor for various forms of vascular diseases. This association has been ascertained in many retrospective and prospective studies, but the strength of risk has not yet firmly established ***(Fowler, 2005).***

Homocysteine, is a metabolic product of methyl group donation by the indispensable amino acid methionine. It has several potentially deleterious vascular actions including increased oxidant stress, impaired endothelial function, stimulation of mitogenesis and induction of thrombosis ***(Haynes, 2002).***

Homocysteine is metabolized either by remethylation which regenerates methionine, this requires folate and vitamin B12 or transsulfurated to cystathionine and cysteine, this requires vitamin B6 ***(Selhub, 1999).***

Folic acid deficiency is the most common B-vitamin deficiency as animal foods with the exception of liver are poor sources of folic acid, and plant sources rich in folic acid are not frequently obtained in adequate amount in the diet ***(Alpert et al., 2000).***

While folic acid reduces homocysteine levels, it still unclear whether folic acid supplementation will reduce the risk of cardiovascular diseases, morbidity and mortality. It is also unclear whether any benefit of folic acid is attributable to lowering homocysteine levels ***(Splaver et al., 2004).***

AIM OF THE WORK

Is to investigate the role of folic acid in the protection against isoprenaline induced myocardial injury and its relation to hyperhomocysteinemia.