INTRODUCTION
1.1 Motivation of this work

Atherosclerosis is a serious condition; its constant increasing numbers suggest that it will be the most dangerous reason of health related deaths. According to WHO Indians are more susceptible to it. The atherogenic process initiates early in life, this includes endothelial damage, lipid infiltration, followed by intimal thickening, platelet adherence, smooth muscle cell proliferation and plaque formation.

This pattern provides a window of opportunity for the high risk subjects, and the application of appropriate preventing strategies.

Asymmetric dimethylarginine (ADMA) is a naturally occurring amino acid that circulates in plasma, is excreted in urine, and is found in tissues and cells. It has aroused interest because it inhibits nitric oxide synthases (NOSs) and therefore has the potential to produce considerable biological effects, particularly in the cardiovascular system. Recently, several studies have suggested that the plasma concentrations of ADMA provide a marker of risk for endothelial dysfunction and cardiovascular disease. ADMA is synthesized when arginine residues in proteins are methylated by the action of protein arginine methyltransferases (PRMTs). The asymmetrically methylated arginine (ADMA) is inhibitor of NOS, it inhibits all three isoforms of NOS, in addition to blocking NO formation, it may uncouple NOS and leads to generation of superoxide. It elevates blood pressure, causes vasoconstriction, impairs endothelium-dependent relaxation, and increases endothelial cell adhesiveness. Like ADMA, homocysteine, mylperoxidase and lipoprotein(a) have role in vascular pathology mediated through NO.

hs-CRP, fibrinogen, albumin excretion rate were known inflammatory markers. These markers increases oxidative stress of body leading to decreased NO because of its antioxidant properties. Diabetes and glycation of protein associated with oxidative stress, which leads to
change in vascular physiology, provides a platform for atherosclerosis initiation and progression.

Aim of the present study is to find out a newer potential marker for early and better diagnosis of CAD or its progression. During this research estimation of each individual risk factor was performed in diseased group (DM, CAD and DM with CAD) and normal healthy control subjects and it was attempted to find out which one is most reliable.

1.2 Thesis Outline
The remaining of this thesis is organized as follows. In chapter 2 describes previous work and background of individual risk factor. Chapter 3 describes experimental data preparation, experimental design. Chapter 4 presents experimental results. Chapter 5 discusses results of study in light of previous one. In Chapter 6 concludes present work and it also presents the avenues for future work. Chapter 7 discusses summary of the work done. Chapter 8 presents tables while Chapter 9 presents graphs and ligands. Chapter 10 contains list of citations and Chapter 11 presents published papers related to present work.