INTRODUCTION
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An increase in serum total cholesterol is clinically related to development of atherosclerotic patch. Certain factor like hypertension, diabetes, obesity, smoking, stress, sedentary life style and family history of ischemic heart disease also aggravate the risk of atherosclerosis.

There is now conclusive proof that low density lipoprotein (LDL) is a factor etiologically related to atherosclerotic vascular disease and in particular to coronary heart disease (Ross et al., 1986). A protective effect against coronary heart disease of elevated serum high density lipoprotein (HDL) has also been observed in several epidemiological and clinical studies (Miller and Miller, 1975). A low serum level of HDL appears to be an important risk factor, particularly in population where serum level of total cholesterol is high.

Many workers say that postprandial response of an individual to high cholesterol fat load may more appropriately be related to risk of developing atherosclerosis in future. Little has been done in establishing a correlation between postprandial response of an individual and risk of atherosclerosis. Even more, postprandial responses after cholesterol fat load on different lipid lipoprotein have not been studied. Proper definition of these responses and correct interpretation may perhaps be the first
major step towards formulation of cholesterol tolerance test.

Previous efforts in this direction by several workers (Albrink and Man et al, 1956; Pomeranze, 1954) did not reveal much primarily because each of them calculated STC and other lipid subfraction 2-6 hours after test feeding, thinking that cholesterol is a slowly absorbed substance and cannot alter blood cholesterol level before two hours. The disappearance of intravenous radioactive HDL cholesterol within 20 minutes of injection from the vascular compartment, reflect the high dynamic state. Perhaps this dynamic equilibrium was achieved by activated LDL receptor activity (Goldstein). There is possibility that some unidentified hormonal or neurogenic reflexes affecting these receptors.

There is enormous individual variability in response after cholesterol fat feeding. Results of previous studies in our department have further strengthen this view. Feeding high cholesterol breakfast for seven days in young and old subjects resulted in increase level of serum total cholesterol with prominent rise of HDL in youngers and that of LDL in older subjects (Arora et al, 1984 and 1985). This study also brought out the fact, that in some subjects, prolonged cholesterol fat feeding resulted in fall in STC level instead of rising. Present knowledge offers no satisfactory explanation for the above phenomenon. Since prolonged feeding is not practicable
in mass scale for screening purpose. So we decided to study postprandial changes in fasting lipoprotein profile after ingestion of single high cholesterol load in normal subjects and persons predisposed to premature atherosclerosis. A pilot study was also done which showed acute changes in STC, LDL and STG after single high cholesterol (egg cholesterol) test load. On the basis of this pilot study, a study was carried out by Arora et al (1989) which demonstrated that acute changes in STC, HDL, LDL and STG after high cholesterol test feeding in healthy subjects majority of the healthy population showed a fall in STC and LDL level at the one hour while in diabetics, first degree relatives of IHD and minority of healthy population display a rise in STC and LDL levels at one hour.

The above particular behaviour prompted us to study changes in lipid lipoprotein profile after single high cholesterol (egg cholesterol) test feeding in diseased subjects, so that the concept of cholesterol tolerance test can be further assessed.

**AIMS OF THE STUDY**

1. To assess the changes in basal lipoprotein profile after giving single high cholesterol test load in diseased subjects.

2. To correlate these changes for quantitative and qualitative risk of an individual for atherogenesis.